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# NÖRO PSİKIYATRİ ARSİVİ

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**Bildiri Özетleri**

**10<sup>th</sup> MENA Meeting**  
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**Abstracts**



# NÖRO PSİKIYATRİ ARŞİVİ

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- **YAYIN ÜCRETLERİ**

### YAYIN SÜRECİ

Derginin editöryel ve yayın süreçleri International Committee of Medical Journal Editors (ICMJE), World Association of Medical Editors (WAME), Council of Science Editors (CSE), Committee on Publication Ethics (COPE), European Association of Science Editors (EASE) ve National Information Standards Organization (NISO) kılavuzlarına uygun olarak biçimlendirilmiştir. Ayrıca bu süreçler, Principles of Transparency and Best Practice in Scholarly Publishing ([doaj.org/bestpractice](http://doaj.org/bestpractice)) ilkelerine uygun olarak yürütülmektedir.

Gönderilen yazıların daha önce başka bir elektronik ya da basılı dergide, kitapta ya da farklı bir ortamda yayınlanmamış olması ya da yayınlanmak için değerlendirme aşamasında olmaması gereklidir. Daha önce başka bir dergiye gönderilen ancak yayına kabul edilmeyen yazılar için bilgi verilmesi ve önceki hakem raporlarının Nöropsikiyatri Arşivi Yayın Kurulu'na gönderilmesi değerlendirme sürecinin hızlanması sağlayacaktır. Önceden toplantınlarda sunulan çalışmalar için, toplantıının tam adı, tarihi, şehri ve ülkesi belirtilmelidir.

Nöropsikiyatri Arşivi'ne gönderilen makaleler, bilimsel nitelik ve özgünlük açısından incelenir. Makalelerin özgünlükleri, yöntemleri, konunun önemi ve dergi kapsamında olup olmadığı öncelikle Yayın Kurulu tarafından değerlendirilir. Nöropsikiyatri Arşivi'nin bilimsel yayın standartlarını karşılayan makaleler, tarafsız değerlendirme sürecini sağlamak için alanlarında uzman en az iki dış-bağımsız hakeme gönderilir. Makalelerin karar sürecinde bağımsız, tarafsız, çift-köy hakem değerlendirme raporları temel alınmaktadır. Bütün makalelerin karar verme süreçlerinde son karar yetkisi Genel Yayın Yönetmenleri'ne aittir. Dergi Yayın Kurulu üyeleri tarafından gönderilecek makalelerin değerlendirme süreçleri, davet edilecek dış bağımsız yayın yönetmenleri tarafından yönetilecektir.

Klinik ve deneysel çalışmalar, ilaç araştırmaları ve bazı olgu sunumları için Dünya Tabipler Birliği Helsinki Bildirgesi "İnsan Katılımcılar İçin Tibbi Araştırmalar İçin Etik İlkeler" (Ekim 2024), çerçevesinde hazırlanmış Etik Kurul raporu gerekmektedir. Gerekli görülmeli halinde, Etik Kurul raporu veya eş değeri olan resmi bir yazı, yazarlardan talep edilebilir. İnsanlar üzerinde yapılmış deneysel çalışmaların sonuçlarını bildiren yazınlarda, çalışmanın yapıldığı kişilere uygulanan prosedürlerin niteliği tümüyle açıklandıktan sonra, yazılı onaylarının alındığına ilişkin bir açıklama metin içerisinde yer verilmelidir. Hayvanlar üzerinde yapılan çalışmalarla ise ağrı, acı ve rahatsızlık verilmemesi için yapılmış olan işlemler makalede açık olarak belirtilmelidir. Hasta onamları, Etik Kurul raporunun alındığı kurumun adı, onay belgesinin numarası ve

tarihi ana metin dosyasında yer alan "Yöntem" başlığı altında belirtilmelidir. Hastaların kimliklerinin gizliliğini korumak yazarların sorumluluğundadır. Hastaların kimliğini açığa çıkarabilecek fotoğraflar için hastadan ya da yasal temsilcilerinden alınan imzaların de gönderilmesi gereklidir.

İnsanlar üzerinde yapılacak her araştırma, Helsinki Bildirgesi'ne uygun biçimde, ilk katılımcı araştırmaya alınmadan önce kamuya açık bir veri tabanına kaydedilmiş olmalıdır. Tüm klinik çalışmaların ICMJE ölçütlerine uygun bir şekilde Dünya Sağlık Örgütü'nün "Uluslararası Klinik Çalışmalar Kayıt Platformu (International Clinical Trial Registry Platform)" ya da eşdeğeri bir kamuya açık veri tabanınınına kaydedilmesi gerekmektedir. Türkiye'de yapılan klinik çalışmalar için ayrıca Türkiye İlaç ve Tıbbi Cihaz Kurumu'ndan izin alınmış olmalıdır. Klinik çalışmaların kayıt numaraları "Özet" bölümünün sonuna eklenmelidir.

Yazının hazırlanmasında Yapay Zeka (YZ), makine öğrenme ya da benzer bir teknolojiden yararlanılmış ise kullanılan aracın adı, versiyonu ve YZ tarafından oluşturulan içerik yöntem bölümünde açık olarak belirtilmelidir. Ek olarak, yazının sonuna bir "bilgilendirme" bölümü eklenerek, yazının hazırlanmasında YZ araçlarından yararlanıldığı ve YZ tarafından oluşturulan içerik ile ilgili tüm sorumluluğun yazarlara ait olduğu ve hem kullanılan YZ aracının hem de YZ tarafından oluşturulan içeriğin yöntem bölümünde açıklandığı yazılmalıdır. Aynı bilgilendirme makale başvurusu sırasında editörlere gönderilen üst yazda da belirtilmelidir.

Bütün makalelerin benzerlik tespiti denetimi, iThenticate yazılımı aracılığıyla yapılmaktadır.

Kurum Bilgileri: Yazarlar hem çalışmanın yapıldığı kurumun hem de güncel kurumlarının isimlerini belirtmelidir.

Makalelerin değerlendirilmesi sırasında ya da kabul ve yayınlanması sonrasında yazarlık ile ilgili konularda herhangi bir anlaşılmazlık ortaya çıkması durumunda Nöropsikiyatri Arşivi inceleme yapma ya da yargıya varma konumunda olmayacağından emin olmalıdır. Yazarların yazarlıkla ilişkili anlaşmazlıklarını kendi aralarında çözümlemeleri beklenmektedir. Gerekli görülmeli durumunda Nöropsikiyatri Arşivi ilgili kurumlara ulaşma ve makaleleri reddetme ya da geri çekme hakkını saklı tutar.

Yayın Kurulu, dergiye gönderilen çalışmalar hakkındaki intihal, atıf manipülasyonu ve veri sahteciliği iddia ve şüpheleri karşısında COPE kurallarına uygun olarak hareket edecektr. Yayın Kurulu tarafından yazarlara ilgili her türlü itiraz ve yakınma COPE rehberleri kapsamında işleme alınmaktadır. Yazarlar, itiraz ve şikayetleri için doğrudan Editorial Ofis ile temasla gelebilirler. Gerek duyulduğunda Yayın Kurulu kendi içinde çözemediği konular için COPE ile iletişime geçip danışmanlık alabilir. Bu durumda COPE önerisi son karar olarak kabul edilecektir.

Yazar olarak listelenen herkesin ICMJE ([www.icmje.org](http://www.icmje.org)) tarafından önerilen yazarlık kriterlerini karşılaması gerekmektedir. ICMJE, yazarların aşağıdaki dört kriteri karşılamasını önermektedir:

1. Çalışmanın yaratım ve tasarımlına; ya da çalışma için verilerin toplanmasına, analiz edilmesine ve yorumlanmasına önemli katkı sağlamış olmak; VE
2. Çalışmanın yazılmasında yer almak ya da çalışmanın önemli düşünsel içeriğinin eleştirel incelemesini yapmak; VE
3. Yazının yayından önceki son biçiminin gözden geçirmiş ve onaylamış olmak; VE
4. Çalışmanın herhangi bir bölümünün geçerliliği ve doğruluğuna ilişkin soruların uygun biçimde soruşturulduğu ve çözümlendiğinin garantisini vermek amacıyla çalışmanın her yönünden sorumlu olmayı kabul etmek.

Bir yazar, çalışmada katkı sağladığı kısımların sorumluluğunu almasına ek olarak, diğer yazarların çalışmanın hangi bölümlerinden sorumlu olduğunu da saptayabilмелidir. Ayrıca, yazarlar birbirlerinin katkılarının sağlamlığına güven duymalılardır. Yazar olarak belirtilen her kişi, yazarlığın dört ölçütünü de karşılamalıdır ve bu dört ölçütü karşılayan her kişi yazar olarak tanımlanmalıdır. Dört ölçütün hepsini karşılamayan kişilere makalenin başlık sayfasında teşekkür edilmelidir.

Yazarlık haklarına uygun hareket etmek ve hayalet ya da lütfü yazarlığın önemnesini sağlamak amacıyla sorumlu yazarlar makale yükleme sürecinde [www.noropsikiyatriarsivi.com](http://www.noropsikiyatriarsivi.com) adresinden erişilebilen Yazar Katkı Formu'nu imzalamalı ve taranmış versiyonunu yazıyla birlikte göndermelidir. Yayın Kurulu'nun gönderilen bir makalede "lütfü yazarlık" olduğundan şüphelenmesi durumunda söz konusu makale değerlendirme yapılmaksızın reddedilecektir. Makale gönderimi kapsamında; sorumlu yazar makale gönderim ve değerlendirme süreçleri boyunca yazarlık ile ilgili tüm sorumluluğu kabul ettiğini bildiren kısa bir ön yazı göndermelidir.

Çalışma çok sayıda yazarın yer aldığı büyük bir grup tarafından gerçekleştirilmiş ise, grup çalışma başlamadan önce kimlerin yazar olacağına karar vermelii ve makaleyi göndermeden önce yazar isimlerini onaylamış olmalıdır. Grubun tüm üyeleri yazarlık için gereken her dört koşulu karşılamalı ve çıkar çatışması açıklaması formlarını bireysel olarak doldurmalıdır. Bazı çok yazarlı büyük gruplar kendilerine bir grup ismi belirleyerek bu grup ismini yazar adı olarak kullanır. Böyle bir durumda dergi ile yazışmayı yürüten yazar, grup ismini belirtmeli ve çalışmada yazar olarak sorumluluk alacak üye isimlerini açıkça yazmalıdır.

Nöropsikiyatri Arşivi; gönderilen makalelerin değerlendirme sürecine dâhil olan bireylerin ve yazarların, potansiyel çıkar çatışmasına ya da önyargiya yol açabilecek finansal, kurumsal ve diğer ilişkiler dâhil mevcut ya da potansiyel çıkar çatışmalarını beyan etmelerini talep ve teşvik eder. Bir çalışma için bir birey ya da kurumdan alınan her türlü finansal destek ya da diğer destekler Yayın Kurulu'na beyan edilmeli ve potansiyel çıkar çatışmalarını beyan etmek amacıyla [ICMJE Potansiyel Çıkar Çatışmaları Formu](#) katkı sağlayan tüm yazarlar tarafından ayrı ayrı doldurulmalıdır. Yayın Yönetmenleri, yazarlar ve hakemler ile ilgili potansiyel çıkar çatışması vakaları derginin Yayın Kurulu tarafından [COPE](#) ve [ICMJE](#) rehberleri kapsamında çözülmektedir.

Başvuru aşamasında makaleler dergiye Türkçe ya da İngilizce olarak gönderilebilir. Değerlendirme süreci tamamlandıktan sonra yayınlanmak üzere kabul edilen makaleler her iki dilde hem Türkçe hem İngilizce olarak yayınlanmaktadır. Yayına kabul edilen Türkçe makalelerin İngilizce çevirisinin ve İngilizce makalelerin Türkçe çevirisinin yazarlar tarafından yapılması beklenmektedir. Türkiye dışından gönderilmiş ve yazarların hiçbirisinin Türkçe bilmemiş İngilizce makalelerin yayına kabul edilmeleri durumunda Türkçe'ye çeviri dergi tarafından sağlanacaktır.

Nöropsikiyatri Arşivi'ne makale gönderirken yazarlar makalelerinin telif haklarını Türk Nöropsikiyatri Derneği'ne devretmeyi

kabul ederler. Reddedilen makalelerin telif hakları yazarlarına geri verilecektir. Nöropsikiyatri Arşivi her makalenin [www.noropsikiyatriarsivi.com](http://www.noropsikiyatriarsivi.com) adresinden erişebileceğiniz [Yayın Hakkı Devir Formu](#) ile beraber gönderilmesini talep eder. Yazarlar, basılı ya da elektronik formatta yer alan resimler, tablolar ya da diğer her türlü içerik dahil dâhil daha önce yayımlanmış içeriği kullanırken telif hakkı sahibinden izin almalıdır. Bu konudaki mali ve cezai yasal sorumluluk yazarlara aittir.

Dergide yayımlanan makalelerde ifade edilen görüş ve düşünceler, Nöropsikiyatri Arşivi, Genel Yayın Yönetmenleri, Yardımcı Yayın Yönetmenleri, Yayın Kurulu ve Yayıncı'nın değil, yazar(lar)ın bakış açılarını yansıtır. Genel Yayın Yönetmenleri, Yardımcı Yayın Yönetmenleri, Yayın Kurulu ve Yayıncı bu gibi durumlar için hiçbir sorumluluk ya da yükümlülük kabul etmemektedir. Yayınlanan içerik ile ilgili tüm sorumluluk yazarlara aittir.

## MAKALE GÖNDERME

Makaleler, ICMJE-Recommendations for the Conduct, Reporting, Editing and Publication of Scholarly Work in Medical Journals (updated in January 2025 - <http://www.icmje.org/icmje-recommendations.pdf>) ile uyumlu olarak hazırlanmalıdır. Randomize çalışmalar CONSORT, gözlemlsel çalışmalar STROBE, tanışsal değerli çalışmalar STARD, sistematik derleme ve meta-analizler PRISMA, hayvan deneyli çalışmalar ARRIVE, randomize olmayan davranış ve halk sağlığı ilgili çalışmalar TREND ve olgu sunumları CARE kılavuzlarına uyumlu olmalıdır.

Makaleler sadece [www.noropsikiyatriarsivi.com](http://www.noropsikiyatriarsivi.com) adresinde yer alan derginin online makale yükleme ve değerlendirme sistemi üzerinden gönderilebilir. Diğer mecralardan gönderilen makaleler değerlendirilmeye alınmayacaktır.

Gönderilen makalelerin dergi yazım kurallarına uygunluğu ilk olarak Editorial Ofis tarafından kontrol edilecek, dergi yazım kurallarına uygun hazırlanmamış makaleler teknik düzeltme talepleri ile birlikte yazarlarına geri gönderilecektir. Yazarların [Yayın Hakkı Devir Formu](#) [Yazar Katkı Formu](#) ve [ICMJE Potansiyel Çıkar Çatışmaları Formu](#)'nu (bu form, tüm yazarlar tarafından doldurulmalıdır) ilk gönderim sırasında online makale sisteme yüklemeleri gerekmektedir. Bu formlara [www.noropsikiyatriarsivi.com](http://www.noropsikiyatriarsivi.com) adresinden erişilebilmektedir.

Gönderilen tüm makalelerle birlikte Microsoft Word (docx) formatında ayrı bir "başlık sayfası" dosyası gönderilmelidir. Başlık sayfası şu bilgileri içermelidir:

- Makalenin başlığı ve 50 karakteri geçmeyen kısa başlığı,
- Yazarların isimleri, kurum bilgileri (çalışmanın yapıldığı ve güncel kurum) ve eğitim dereceleri,
- Finansal destek bilgisi ve diğer destek kaynakları hakkında ayrıntılı bilgi,
- Sorumlu yazarın ismi, adresi, telefon numarası (cep telefonu dâhil) ve e-posta adresi,
- Makale hazırlama sürecine katkıda bulunan ama yazarlık ölçütlerini karşılamayan bireylerle ilgili bilgiler.

## MAKALEİN HAZIRLANMASI

Makaleler "Times New Roman" karakteri, 12 punto ve 1,5 satır aralığı ile yazılmalı ve Microsoft Word (docx) formatında olmalıdır. Makale içerisinde geçen tüm kısaltmalar, ana metin ve özette ayrı ayrı olmak üzere ilk kez kullanıldıkları yerde tanımlanarak kısaltma tanımın ardından parantez içerisinde verilmelidir.

Ana metin içerisinde cihaz, yazılım, ilaç vb. ürünlerden bahsedildiğinde, ürünün ismi, üreticisi, üretildiği şehir ve ülke

bilgisini içeren ürün bilgisi parantez içinde verilmelidir; "Discovery St PET/CT scanner (General Electric, Milwaukee, WI, USA)".

Tüm kaynaklar, tablolar ve resimlere ana metin içinde uygun olan yerlerde sırayla numara verilerek atıf yapılmalıdır.

İstatistiksel analizler, tıbbi dergilerdeki istatistik verilerini bildirme kurallarına göre yapılmalıdır (Altman DG, Gore SM, Gardner MJ, Pocock SJ. Statistical guidelines for contributors to medical journals. Br Med J 1983; 7: 1489-93). İstatistiksel analiz için kullanılan yazılım kesinlikle tanımlanmalıdır.

Birimler, uluslararası birim sistemi olan International System of Units'e (SI) uygun biçimde sunulmalıdır.

**Başlık:** Makale başlığının kısa ve öz ama aynı zamanda kapsamlı ve açıklayıcı olması beklenir. Soru cümlesi biçimindeki başlıklar yalnızca "editöre mektup" yazıları için kabul edilmektedir.

**Özet:** Editöre Mektup türündeki yazılar dışında kalan tüm makalelerin Türkçe ve İngilizce özetleri olmalıdır. Özgün Araştırma ve derleme makalelerinin özetleri "Giriş ve Amaç", "Yöntem", "Bulgular" ve "Sonuç" alt başlıklarını içerecek biçimde hazırlanmalıdır. Olgu sunumlarının özetleri "Giriş ve Amaç", "Olgı" ve "Sonuç" alt başlıklarını içermelidir. Özetler en az 250 en çok 300 kelimeden oluşmalıdır. Özet oluşturma kuralları için lütfen aşağıdaki Tablo 1'i kontrol edin.

**Anahtar Sözcükler:** Tüm makaleler en az üç, en fazla altı anahtar kelimeyle birlikte gönderilmeli, anahtar sözcükler özetin hemen altına, alfabetik sırada yazılmalıdır. Anahtar sözcükler National Library of Medicine (NLM) tarafından hazırlanan Medical Subject Headings (MeSH) veritabanından seçilmelidir (<https://www.nlm.nih.gov/mesh/MBrowser.html>).

**Öne Çıkan Noktalar:** Bu bölüm makalelerin arama motorları tarafından bulunması ve dijital ortamda tanıtımı için özel önem taşımaktadır. Tüm makaleler için (arastırma, olgu sunumu, gözden geçirme, editöre mektup dahil) en az 3, en fazla 5 adet öne çıkan nokta belirtilmelidir. Öne çıkan noktalar makalenin alana kazandığı yeni bulgular, kullandığı yenilikçi yöntemler ya da makalenin en çarpıcı sonuçları olabilir. Her bir öne çıkan nokta boşluklar dahil 85 karakteri geçmeyen kısa bir cümle biçiminde olmalı ve maddeler halinde yazılmalıdır.

Yayın Kurulu yazarları bilgilendirerek "başlık", "anahtar sözcükler" ve "öne çıkan noktalar" için uygun gördüğü değişiklikleri yapma hakkını saklı tutar.

## Makale Türleri

**Özgün Araştırma:** Ana metin; "Giriş", "Yöntem", "Bulgular" ve "Tartışma" alt başlıklarını içermelidir. "Giriş" bölümünde çalışmanın gerekçelerine yer verilmeli ve çalışmanın amaç ve varsayımları son paragrafta açıkça belirtilmelidir. "Yöntem" bölümü "örneklem", "desen", "araçlar", "istatistik" ve "etik" alt başlıklarını içermelidir. "Bulgular" bölümünde sunulan bulgular, okumayı kolaylaştıracak biçimde tablo ve görsellerle desteklenmelidir.

"Tartışma" bölümünde bulguların literatürdeki ilgili çalışmalarla karşılaştırılarak tartışımasına, çalışmanın kısıtlılıklarına ve güçlü yönlerine ve yazarların varlıklarına yer verilmelidir. Özgün Araştırmalar için sözcük sayısı sınırları Tablo 1'dedir.

**Derleme:** Derginin ilgi alanındaki konuları güncel bilgiler ışığında anlatan, tartışan, değerlendiren ve yapılabilecek yeni çalışmalarla ilişkin öneriler sunan yazılardır. Derleme yazarlarının konu hakkındaki birikimi uluslararası literatüre yayın ve atıf sayısı olarak yansımış uzmanlar olması gereklidir. Bu ölçütlerle uygun yazarlar derleme yazısı yazmaları için Yayın Kurulunca davet edilebilir. Yayın Kurulunun daveti olmaksızın gönderilen derleme yazılarından birinin konuya ilgili üç veya daha çok özgün araştırma yayımlamış olması beklenir. Bu yazılar "Giriş", "Yöntem", "Bulgular" ve "Tartışma" alt başlıklarını içermelidir. Derleme yazarlarının sözcük sayısı sınırları Tablo 1'dedir.

**Olgı Sunumu:** Olgı sunumlarına sınırlı yer ayrılmakta ve sadece ender görülen, tanı ve tedavisi güç olan hastalıklarla ilgili, yeni bir yöntem öneren, kitaplarda yer verilmeyen bilgileri yansitan, ilgi çekici ve öğretici özelliği olan olgular yayına kabul edilmektedir. Bu yazılar, "Giriş", "Olgı Sunumu" ve "Tartışma" alt başlıklarını içermelidir. Olgı Sunumlarının sözcük sayısı sınırları Tablo 1'dedir.

**Kısa Bildirim:** Özgün araştırma bulguları veya derginin ilgi alanındaki konularda kuramsal bilgiye ve uygulama sorunlarına değinen, özgün düşüncelerin bildirildiği ve tartışıldığı yazılar bu formatta sunulabilir. Kısa Bildirim yazıları (başlık sayfası, kaynaklar, tablo/şekil/resim hariç) 2500 kelimeyi geçmemelidir. Kısa Bildirimlerin sözcük sayısı sınırları Tablo 1'dedir.

**Editöre Mektuplar:** Dergide yayımlanmış bir makale hakkında konunun uzmanı olan veya makalenin değerlendirmesini yapmış olan hakemler görüş veya yorumlarını Editöre Mektupla bildirebilirler. Kabul edilen Mektuplar, yayımlanmalarından önce konu alındıkları makalenin yazarına gönderilir ve ek görüş bildirmek, cevap vermek isteyip istemedikleri sorulur. Bu tür yazılar mümkün oldukça ilgili yazının yazarlarının yanıtlarıyla birlikte yayımlanır.

## Özel Bölüm

Internet sitelerinin tanıtımı: Derginin yayın alıyla ilgili konularda bilimsel bir içeriği olan, nitelikli internet sitelerini tanıtıci yazılardır.

Kitap Tanımı: Derginin yayın alıyla ilgili konularda yayımlanmış bulunan kitapları/tezleri tanıtan yazılardır. Özel bölüm için yazılmış yazılar 500 sözcükle sınırlıdır (başlık sayfası, kaynaklar, tablo/şekil/resim hariç). En fazla beş referans ve bir tablo veya bir şeke izin verilir.

## Tablolar

Tablolar ana dosya eklenmeli, kaynak listesi sonrasında sunulmalı, ana metin içerisindeki geçiş sıralarına uygun olarak numaralandırılmamıştır. Tabloların üzerinde tanımlayıcı bir başlık yer almali ve tablo içerisinde geçen kısaltmaların açıklamaları tablo altına tanımlanmalıdır. Tablo altındaki bu açıklamalar alfabetik

**Table 1.** Makale türleri için kısıtlamalar

Makale türü	Sözcük sınırı	Özet sözcük sınırı	Kaynak sınırı	Tablo sınırı	Resim sınırı
Özgün Araştırma	4000	250 (Alt başlıklı)	30	6	7 ya da toplamda 15 resim
Derleme	5000	250	50	6	10 ya da toplamda 20 resim
Olgı Sunumu	1500	150	15	Tablo yok	10 ya da toplamda 20 resim
Kısa Bildirim	2500	250	20	6	10 ya da toplamda 20 resim
Editöre Mektup	500	Uygulanamaz	5	Tablo yok	Resim yok
Özel Bölüm	500	Uygulanamaz	5	1	1

sırada listelenmeli ve aralarına noktalı virgül konmalıdır (DEHB: Dikkat Eksikliği Hiperaktivite Bozukluğu; OKB: Obsesif Kompulsif Bozukluk). Tablolar Microsoft Office Word dosyası içinde "Tablo Ekle" komutu kullanılarak hazırlanmalı ve kolay okunabilir şekilde düzenlenmelidir. Tablolarda sunulan veriler ana metinde sunulan verilerin tekrarı olmamalı; ana metindeki verileri destekleyici nitelikte olmalıdır.

### Resim ve Resim Altyazıları

Resimler, grafikler ve fotoğraflar (TIFF ya da JPEG formatında) ayrı dosyalar halinde sisteme yüklenmelidir. Görseller bir Word dosyası dokümanı ya da ana doküman içerisinde sunulmamalıdır. Alt birimlere ayrılan görseller olduğunda, alt birimler tek bir görsel içerisinde verilmemelidir. Her bir alt birim sisteme ayrı bir dosya olarak yüklenmelidir. Resimler alt birimleri belli etme amacıyla etiketlenmemelidir (a, b, c vb.). Resimlerde altyazıları desteklemek için kalın ve ince oklar, ok başları, yıldızlar, asteriksler ve benzer işaretler kullanılabilir. Makalenin geri kalanında olduğu gibi resimler de körleştirilmiş olmalıdır. Bu sebeple, resimlerde yer alan kişiler tanınmamalı ve kurum bilgileri de silinmelidir. Görsellerin minimum çözünürlüğü 300 DPI olmalıdır. Değerlendirme sürecindeki aksaklıları önlemek için gönderilen bütün görsellerin çözünürlüğü net ve boyutu büyük (minimum boyutlar 100x100 mm) olmalıdır. Resim altyazıları ana metnin sonunda yer almalıdır.

### Kaynaklar

Atıf yapılmırken en son ve en güncel yayınlar tercih edilmelidir. Atıf yapılan erken çevrimiçi makalelerin DOI numaraları mutlaka sağlanmalıdır. Kaynakların doğruluğundan yazarlar sorumludur. Dergi isimleri Index Medicus/Medline/PubMed'de yer alan dergi kısaltmaları ile uyumlu olarak kısaltılmalıdır. Yazar sayısı 6 veya daha az ise tüm yazar isimleri listelenmelidir. Yazar sayısı 6'dan fazla ise, 6. yazardan sonra "et al." şeklinde kısaltma yapılmalıdır. Ana metinde kaynaklara atıf yapılmırken parantez içinde Arabık numaralar kullanılmalıdır.

Kaynak gösterme için Mendeley programını kullanan yazarlar programdaki özel stil URL'si bölümne <https://csl.mendeley.com/styles/26629101/nparsivi> adresini kopyalayarak dergimizin kaynak stiline ulaşabilir.

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Farklı yayın türleri için kaynak stilleri aşağıdaki örneklerde sunulmuştur:

**Dergi makalesi:** Katz MM, Koslow SH, Maas JW, Frazer A, Bowden CL, Casper R et al. The timing, specificity and clinical prediction of tricyclic drug effects in depression. *Psychol Med* 1987;17:297-309. doi: 10.1017/s0033291700024831

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**Tez:** Yılmaz B. Ankara Üniversitesi'ndeki Öğrencilerin Beslenme Durumları, Fiziksel Aktiviteleri ve Beden Kitle İndeksleri Kan Lipidleri Arasındaki İlişkiler [Doctoral Thesis]. H.Ü. Sağlık Bilimleri Enstitüsü, 2007.

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**Erken Çevrimiçi Yayın:** Cai L, Yeh BM, Westphalen AC, Roberts JP, Wang ZJ. Adult living donor liver imaging. *Diagn Interv Radiol* 2016, Feb 24. doi: 10.5152/dir.2016.15323 [Epub ahead of print]

**Elektronik formatta yayınlanan yazı:** Morse SS. Factors in the emergence of infectious diseases. *Emerg Infect Dis* (serial online) 1995 Jan-Mar (cited 1996 June 5): 1(1): (24 screens) <http://www.cdc.gov/ncidod/EID/cid.htm>

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# NÖRO PSİKIYATRİ ARŞİVİ

ARCHIVES OF NEUROPSYCHIATRY

**Nöropsikiyatri Arşivi için 2024 yılında hakemlik yapan uzmanlara değerli katkıları için çok teşekkür ederiz.**

*We would like to thank the experts who served as reviewer for Archives of Neuropsychiatry in 2024 for their valuable contribution.*

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## 10<sup>th</sup> MENA Meeting 6<sup>th</sup> Turkish-African Meeting of Headache and Pain Management

**AXIII** **BİLDİRİ ÖZETLERİ / ABSTRACTS**

**AXIV** **ORGANİZASYON KOMİTESİ / ORGANIZING COMMITTEE**

**AXV** **DAVET MEKTUBU /INVITATION LETTER**

**1** **SÖZEL BİLDİRİLER / ORAL PRESENTATIONS**



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# **10<sup>th</sup> MENA Meeting 6<sup>th</sup> Turkish-African Meeting of Headache and Pain Management**

**17-19 October 2025**

**Mersin, Türkiye**

## **Abstracts**



# 10<sup>th</sup> MENA Meeting

## 6<sup>th</sup> Turkish-African Meeting of Headache and Pain Management

17-19 October 2025  
Mersin, Türkiye

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# 10<sup>th</sup> MENA Meeting 6<sup>th</sup> Turkish-African Meeting of Headache and Pain Management

17-19 October 2025  
Mersin, Türkiye

## Invitation letter

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Dear Colleagues,

For many years, we have been enriching our knowledge with case-based practical approaches by compiling current expertise in headaches with our colleagues in MENA and Central Asia. As we get to know each other and enjoy learning, our interest in the field increases even more. This year we want to focus on 'practical solutions for sustainable and realistic headache science'. We want to meet you with a multidisciplinary team in Mersin, where the unique nature of the Taurus Mountains and the Mediterranean Sea meet. We will bring you together with the pioneers of headaches and offer one-to-one communication. At the same time, we will practice interventional treatments with small groups on models.

Additionally, we will have a pleasant time together in our growing family by getting to know you.

We want to meet at the meeting point of cultures and experiences.

*On behalf of the Organising and Scientific Committee*

Prof. Dr. Aynur Özge (Chair)

Assoc. Prof. Dr Nevra Öksüz Gürlen (Scientific Secretariat)



**OP-1**

## **MIGRAINE AND COPING IN THE WORKPLACE**

**Lala Bouna SECK<sup>1</sup>, Ahmadou Bamba MBODJI<sup>2</sup>, Arame GAYE<sup>2</sup>, Henriette SENGHOR<sup>2</sup>,  
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**BACKGROUND:** Migraine pain leads to coping responses, most of the time defensive ones. We aimed to assess migraine impact and negative coping responses in working condition during migraine fits, and tried to find interrelated factors.

**METHODS:** We conducted a prospective cross sectional study at the neurosciences department of Fann National University Hospital in Dakar. We recruited female subjects working in the health field, suffering from headache (s) with at least one of them meeting the IHS migraine criteria, after free and informed consent. We focused on fits occurring in the workplace. We collected data on civil status, clinical and therapeutic aspects of the migraine, the migraine impact using MIDAS and HIT-6 scales, the negative coping responses using the Canadian-French version of the Pain Catastrophizing Scale (PCS-CF). We tried to find interrelated factors.

**RESULTS:** Twenty females, civil servants in the field of health were interviewed, all meeting the criteria, with aura present in 60% of the cases. Their mean age was 34,7 +/- 8 years. A non-migraine headache was associated for 35% of them. The migraine duration was 1 to 40 years with EVA score 35 to 100. Half of them reported improvement of the disease. In working condition, the MIDAS scale showed a moderate to significant functional disability in 60%, and HIT-6 score, a significant to major impact on work in 80%. According to PCS-CF scale, 45% of patients had significant score for migraine attacks occurring on the workplace. There was no statistical link between MIDAS and HIT-6 scores on one hand and on the other hand, disease duration, headache intensity, the existence or not of aura. PCS-CF score was not correlated with attacks severity or MIDAS and HIT-6 scores or the migraine evolution.

**CONCLUSION:** Despite the debilitating effect of their migraine in the workplace for most of the patients, the majority do not adopt catastrophizing behaviours to cope with pain. Other factors may probably come into play to determine the type of coping in migraine in African people, like cultural factor.

**Key words:** migraine, coping, workplace

**OP-2**

## **POSTERIOR CEREBRAL CIRCULATION VARIANTS IN MIGRAINE: THE POSSIBLE ROLE OF VERTEBRAL ARTERY HYPOPLASIA**

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**BACKGROUND:** Vertebral artery hypoplasia (VAH) is a common vascular variant in the general population and has been implicated in altered posterior circulation hemodynamics. While its association with posterior circulation ischemic stroke is well recognized, the relationship between VAH and migraine phenotypes, particularly chronic migraine, remains controversial. Previous studies have reported inconsistent results with aura, headache laterality, and medication-overuse headache (MOH). We aimed to investigate the frequency and laterality of VAH in migraine patients and its relationship with migraine aura, chronicity, headache laterality, pain intensity, and MOH.

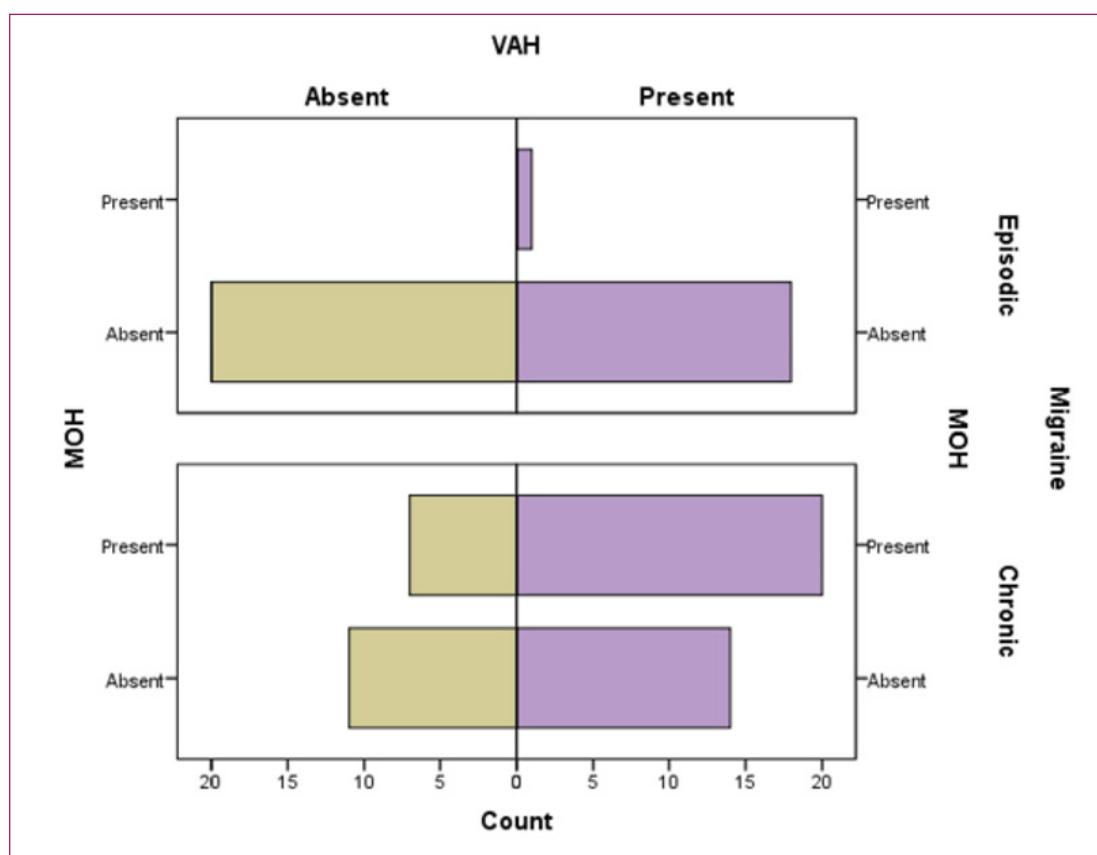
**METHODS:** This single-center, cross-sectional study included 91 patients diagnosed with migraine according to ICHD-3 criteria. Vertebral artery diameters were measured via magnetic resonance imaging angiography (MRA) and computerized tomography cerebral angiograms (CTA), and VAH was defined as a diameter  $\leq 2$  mm and marked side-to-side asymmetry (1). Clinical variables included migraine chronicity (chronic vs episodic), presence of aura, headache laterality, Numeric Rating Scale (NRS) pain score, presence of MOH, smoking and alcohol use, history of stroke or atherosclerotic disease, and presence of mood/anxiety disorders. Categorical variables were compared using the Mann-Whitney U test; continuous variables with the t-test as appropriate. Correlation analysis and binary logistic regression were performed.

**RESULTS:** In the present study, 72 of the migraineurs were women, and the mean age was  $43.3 \pm 12.6$ . VAH was observed in 58% (40% right, 13% left) of migraine patients. Right VAH was associated with migraine chronicity ( $p = 0.024$ ) and MOH ( $p=0.033$ ). Right vertebral artery diameter demonstrated a significant correlation with chronic migraine ( $p=0.028$ ,  $r=0.230$ ) and MOH ( $p=0.032$ ,  $r=0.225$ ). No significant associations were found between VAH and aura, headache laterality, or NRS. Binary logistic regression did not identify VAH as an independent predictor of migraine chronicity or MOH after adjusting for potential confounders. Vascular risk factors and psychiatric comorbidities were not significantly different between VAH and non-VAH groups.

**CONCLUSION:** In this migraine cohort, VAH—particularly right-sided hypoplasia—was common and associated with migraine chronicity at the univariate level, but not as an independent predictor after multivariate adjustment. VAH was observed at a higher frequency than population-based reports. Right-sided hypoplasia was more prevalent than left-sided hypoplasia, comparable to previous reports. Studies have reported a higher incidence (40%) of VAH in patients with posterior circulation stroke, demonstrating that changes in posterior circulation hemodynamics are significant in these patients. Although our cohort included a small number of stroke patients (4.3%, only 1 of them had VAH), a high ratio of VAH was observed, suggesting that changes in posterior circulation

hemodynamics may also occur in migraine patients and may have an impact on pain chronicity and MOH. Further studies in larger populations are required to confirm the effect of posterior circulation hemodynamics on migraine chronicity and MOH

**Key words:** vertebral artery hypoplasia, migraine, chronic migraine, medication overuse headache, posterior circulation.



**Figure 1.** Vertebral Artery Hypoplasia in Episodic Migraine, Chronic Migraine and MOH patients

**OP-3**

## **ICTAL ANODAL TDCS OVER CONTRALATERAL S1 ALTERS MEDIAN-NERVE SSEPS AND PAIN IN MIGRAINE: A RANDOMIZED CROSSOVER STUDY WITH 24-HOUR TRACKING**

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**BACKGROUND:** Sensory processing abnormalities are a hallmark of migraine. Somatosensory evoked potentials (SSEPs) provide a mechanism-proximal biomarker, yet few ictal neuromodulation trials incorporate SSEPs as endpoints.

**METHODS:** In a single-centre, randomised, double-blind, sham-controlled, two-period crossover trial, adults with migraine with aura (MwA) or without aura (MwoA) presented within  $\leq 60$  minutes of attack onset on two occasions, receiving active anodal tDCS over contralateral S1 during one attack and sham during the other (washout  $\geq 7$ –14 days). Primary endpoints were change in SSEP N20 latency immediately post-stimulation ( $\Delta N20$ ) and change in pain intensity at 2 hours ( $\Delta VAS$ ). SSEP tracking continued at predefined intervals up to 24 hours. Secondary outcomes included N20–P25 amplitude, habituation slope, rescue medication use, HIT-6, and MIDAS. Analyses used repeated-measures ANOVA and mixed models; aura status and baseline N20 were prespecified moderators.

**RESULTS:** Thirty-two participants were randomized (MwA n=16; MwoA n=16); 30 completed both periods. Active tDCS shortened N20 latency relative to sham (MwA:  $-1.2 \pm 0.6$  ms; MwoA:  $-0.8 \pm 0.5$  ms), increased N20–P25 amplitude, and reduced pain at 2 hours (MwA:  $-4.1 \pm 1.2$ ; MwoA:  $-3.4 \pm 1.1$ ). Rescue medication use was lower with active stimulation (0% vs. 53%). Baseline N20 and active allocation independently predicted  $\Delta N20$ ;  $\Delta N20$  predicted  $\Delta VAS$  after adjustment for baseline VAS and aura. Adverse events were mild and transient; blinding remained intact.

**CONCLUSION:** Ictal anodal tDCS over S1 produces convergent electrophysiological and clinical benefits in migraine, with larger effects in MwA. The  $\Delta N20$ – $\Delta VAS$  relationship supports N20 latency as a practical biomarker of target engagement and potential predictor of response. Replication in multicentre trials and biomarker-guided optimisation are warranted.

**Key words:** tDCS, SSEP, migraine, headache

**OP-4**

## **COGNITIVE SYMPTOMS AND PAIN-RELATED FEAR IN MIGRAINE: A COMPARATIVE STUDY ACROSS EPISODIC, CHRONIC, AND MEDICATION OVERUSE HEADACHE**

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**BACKGROUND:** Migraine is a common primary headache disorder with substantial cognitive, functional, and psychological impact during and between attacks. Deficits in attention, memory, and processing speed can further impair quality of life. The Migraine Screen of Cognitive Symptoms (Mig-SCog) assesses cognitive symptoms, while the Fear of Pain Questionnaire-III (FPQ-III) measures pain-related fear. Data comparing these domains across episodic migraine (EM), chronic migraine (CM), and medication overuse headache (MOH) are limited. This study aimed to compare cognitive symptoms and pain-related fear in EM, CM, and MOH using the Mig-SCog and FPQ-III.

**METHODS:** Eligible participants were evaluated by a neurologist and classified into episodic migraine (EM), chronic migraine (CM), or medication overuse headache (MOH) according to ICHD-3 criteria. An online survey via online survey included the Migraine Screen of Cognitive Symptoms (Mig-SCog) and the Fear of Pain Questionnaire-III (FPQ-III). Patients were instructed to indicate their headache group based on the clinical diagnosis. Descriptive statistics were presented as mean  $\pm$  SD or n (%). Group differences were analyzed using the Kruskal-Wallis and Mann-Whitney U tests, with  $p < 0.05$  considered significant.

**RESULTS:** A total of 260 participants were included, comprising 124 (47.7%) with episodic migraine (EM), 60 (23.1%) with chronic migraine (CM), and 76 (29.2%) with medication overuse headache (MOH). Of the participants, 229 (88.1%) were female and 31 (11.9%) were male. The mean age was  $33.7 \pm 10.6$  years, and the mean VAS pain score was  $8.6 \pm 1.36$ . Mig-SCog total scores were significantly higher in participants with photophobia compared to those without ( $p = 0.009$ ). Item-level analysis revealed significant impairments in the ability to maintain a train of thought ( $p = 0.001$ ), planning ability ( $p = 0.024$ ), and sentence formulation ( $p = 0.047$ ). Comparison between EM and MOH groups demonstrated a significant difference in the Mig-SCog item assessing difficulty maintaining a train of thought ( $p = 0.007$ ). There was no significant difference in total pain-related fear scores among three groups ( $p=0.251$ ). Similarly, no significant differences were found between groups when FPQ-III subscales minor pain fear, severe pain fear, and medical pain fear—were analyzed separately ( $p = 0.158$ ,  $p=0.328$ ,  $p=0.616$ ).

**CONCLUSION:** Photophobia in migraine was associated with higher overall cognitive symptom burden, particularly affecting abilities related to maintaining a train of thought, planning, and sentence formulation. Differences in specific cognitive functions were observed between EM and MOH, while pain-related fear did not differ significantly across migraine phenotypes or FPQ-III subscales. These findings highlight the relevance of targeted cognitive assessment in migraine management, especially in patients presenting with photophobia. The absence of significant differences in pain-related fear suggests it may be a pervasive psychological feature in migraine, independent of attack frequency or medication overuse. Adaptation or habituation to recurrent pain in chronic forms may also contribute to similar scores across groups.

**Key words:** migraine, cognitive symptom, pain-related fear, Mig-SCog

**OP-5**

## **EFFICACY OF ULTRASOUND-GUIDED GREATER OCCIPITAL NERVE AND SUPERFICIAL CERVICAL PLEXUS BLOCKS IN CHRONIC MIGRAINE**

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**BACKGROUND:** Migraine is a neurological disorder characterized by recurrent unilateral, pulsating, and severe headache attacks that significantly impair quality of life. The trigeminocervical complex (TCC) is thought to play a crucial role in its pathophysiology. Due to the convergence of sensory inputs from cervical and trigeminal afferents at the level of second- order neurons in the trigeminal nucleus caudalis (TNC), interventional procedures targeting cervical sensory nerves are utilized in treatment. In this study, we aimed to evaluate the efficacy of combining the superficial cervical plexus block (SCPB), targeting the sensory branches of the C2-C4 cervical nerves, with the greater occipital nerve (GON) block in patients with chronic migraine.

**METHODS:** This study included 26 patients aged 18-65 years who met the diagnostic criteria for chronic migraine according to the International Classification of Headache Disorders, 3rd edition (ICHD-3), between March 2024-2025. Patients who underwent a single-session, bilateral GON block or a single-session, bilateral GON + SCPB were evaluated. All blocks were performed under ultrasound (US)-guidance using 2 mL of 0.25% bupivacaine for each site. Demographic variables including age, sex, educational status, body mass index (BMI), and symptom duration were recorded. Headache frequency, duration, severity (Numerical Rating Scale, NRS), and Headache Impact Test-6 (HIT-6) scores were evaluated at baseline and at 1 month after treatment.

**RESULTS:** Of the 26 patients, 17 were female and 9 were male, with a mean age of  $38.46 \pm 10.99$  years and a mean BMI of  $28.46 \pm 3.74$  kg/m<sup>2</sup>. Compared to baseline, both groups showed significant reductions in headache frequency, duration, and severity, as well as HIT-6 scores, at 1 month ( $p < 0.05$ ). Between-group comparison at 1 month revealed significant differences in headache frequency and severity, whereas no significant differences were observed in headache duration or HIT-6 scores.

**CONCLUSION:** In chronic migraine management, interventional procedures are employed in patients who fail to achieve adequate pain relief with conservative therapies. The addition of ultrasound-guided SCPB to GON blockade may provide greater pain reduction in affected patients. Further large-scale, controlled studies are warranted to confirm and expand upon these findings.

**Key words:** migraine, trigeminocervical complex, greater occipital nerve, superficial cervical plexus

**OP-6**

## **THE RELATIONSHIP BETWEEN CLINICAL AND RADIOLOGICAL PARAMETERS AND DEMOGRAPHIC FACTORS IN MIGRAINE SUBTYPES**

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**BACKGROUND:** Migraine, the third most common neurological disorder worldwide, is linked to subclinical white matter lesions (WMLs). The underlying mechanisms remain unclear. This study aimed to examine the association between clinical/demographic factors and brain MRI WML burden in migraine subtypes, focusing on sex, age, and medication overuse.

**METHODS:** This single-center cross-sectional study included 71 migraine patients diagnosed with migraine according to ICHD-3 criteria. Age, sex, migraine type (episodic/chronic), monthly headache days, intensity (0-10 visual analog scale), location, aura, and medication overuse headache (MOH) were recorded. At diagnosis, all patients underwent brain MRI to exclude secondary causes. T2 hyperintense lesions were classified as periventricular, cortical, juxtacortical, subcortical, brainstem or cerebellar and counted. Categorical variables were compared with chi-square, continuous with t-test; correlations used Spearman's coefficient; multiple linear regression identified lesion count predictors ( $p<0.05$ ).

**RESULTS:** Episodic migraine patients (mean age  $39.1\pm11.3$  years) were younger than chronic group ( $45.3\pm14.0$  years). Females comprised 92.6% of the episodic and 75.6% of the chronic group, with no significant sex distribution difference ( $p= 0.133$ ). Monthly headache frequency and total T2 lesion count were higher in chronic migraine group ( $p<0.001$ ,  $p= 0.023$ ), with more cortical lesions ( $p= 0.022$ ) and borderline more juxtacortical lesions ( $p= 0.059$ ) (Table 1). Other lesion sites and headache intensity showed no group differences. In episodic migraine, periventricular lesions were more frequent in females ( $p= 0.020$ ). In chronic migraine, MOH was more common in females; MOH patients had higher headache frequency ( $p= 0.001$ ) and borderline higher intensity ( $p= 0.055$ ) than non-MOH group, but no differences in total lesion count or distribution. No sex-based differences in lesion count or locations were observed in MOH or non-MOH group (Table 2). Age correlated with total T2 lesion count in both chronic migraine and non-MOH groups ( $p<0.001$ ). Aura status did not affect lesion counts.

**Table 1.** Lesion Distribution in Chronic vs Episodic Migraine

Lesion Type	Chronic Migraine	Episodic Migraine	p-value
Periventricular	$0.80\pm1.13$	$0.54\pm1.06$	0.362
Cortical	$1.75\pm2.66$	$0.67\pm1.13$	<b>0.022</b>
Subcortical	$2.93\pm3.99$	$2.12\pm3.94$	0.426
Juxtacortical	$1.00\pm1.67$	$0.42\pm0.83$	<b>0.059</b>

**Table 2.** Comparison of Clinical and Radiological Parameters Between MOH and Non-MOH Groups

Variable	MOH Mean	Non-MOH Mean	p-value
Lesion Count	4.14	6.27	0.236
Headache Frequency (per month)	20.74	13.09	<b>0.001</b>
Headache Severity (VAS)	7.91	7.15	0.055
Periventricular Lesions	0.50	0.80	0.225
Cortical Lesions	0.91	1.58	0.190
Juxtacortical Lesions	0.73	0.82	0.780
Subcortical Lesions	1.86	3.02	0.191

**CONCLUSION:** WMLs are common in migraine regardless of vascular risk factors. Chronic migraine showed higher lesion counts, particularly cortical, but lesion location did not define subtype. Age-related accumulation was evident, supporting the hypothesis that migraine, especially in chronic form, may be associated with progressive white matter involvement over time. The absence of a clear relationship between lesion distribution and migraine phenotype suggests that lesion development may be more influenced by cumulative disease burden and patient-specific factors, such as age and comorbidities, rather than migraine subtype alone.

**Key words:** migraine, medication overuse headache, hyperintense lesions

**OP-7**

## **MIGRAINE WITH AURA IMPROVED AFTER WARFARIN USE IN A THROMBOPHYLIC PATIENT: CASE REPORT**

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**BACKGROUND:** There are some studies reporting that migraine with aura (MA) and hypercoagulability are comorbid conditions. However, there is no consensus regarding its physiopathology yet. In one of these studies, a significant relationship was shown between MA and Factor 5 Leiden (FVL) and Factor 2 Leiden (FIL) mutations. In another similar study, genetic protein C deficiency, activated protein C resistance and protein S deficiency were found to be significantly higher in MA patients than in healthy control group. These genetic disorders in migraine patients may facilitate the formation of aura. If it is accepted that thrombophilic factors can trigger microthrombotic events, it is possible that the microthrombi formed may trigger the development of migraine with the contribution of environmental factors. In this case report, the possible cause-effect relationship between MA and hypercoagulability will be discussed.

**CASE:** A 36-year-old male patient is being monitored for INR (INR=International Normalized Ratio) in the neurology outpatient clinic of our hospital because the use of warfarin was recommended after cerebral vein thrombosis (CVT). In his history, he was diagnosed with MA at the age of 18. He had 6-8 attacks a month, each lasting 1-2 days. Numbness in the hands and blurred vision begin 15-20 minutes before the pain, followed by pain. The pain was pulsatile, mostly unilateral, and caused nausea and vomiting. According to the visual pain scale (VPS), the pain level was 9-10. At age 21, he was admitted to the hospital with headache and confusion and was diagnosed with CVT. In the etiological investigation for CVT, homozygous FVL polymorphism, homozygous C677T polymorphism in the MTHFR gene, and 4G/5G polymorphism in the Plasminogen activator inhibitor-1 (PAI-1) gene were found and lifelong anticoagulant treatment was recommended. He experienced no aura after anticoagulant therapy. The number of migraine attacks decreased to 1 per month; VPS decreased to 7. He has not received any migraine prophylaxis treatment since the use of warfarin, and the moderate migraine pain that occurs once a month is relieved with paracetamol.

**CONCLUSION:** We presented a case reporting that after starting warfarin treatment at an effective dose, the auras completely ended and the frequency, severity and duration of migraine decreased. We thought that hypercoagulability might have a role in the pathogenesis of MA. There are case reports in the literature reporting a decrease in attack frequency and auras during anticoagulant use in migraineurs with thromboembolic risk factors. These few case reports show that anticoagulant drugs are beneficial, especially in auras that describe visual impairment and headaches. These data are important in terms of showing the cause-effect relationship. We would like to emphasize that new anticoagulants with high confidence intervals that can be used in migraine prophylaxis should be investigated.

**Key words:** migraine with aura, hypercoagulability, comorbidity

**OP-8**

## **BEYOND THE PAIN: POSTDROME SYMPTOMS AS DETERMINANTS OF COGNITIVE IMPAIRMENT AND QUALITY OF LIFE IN MIGRAINE**

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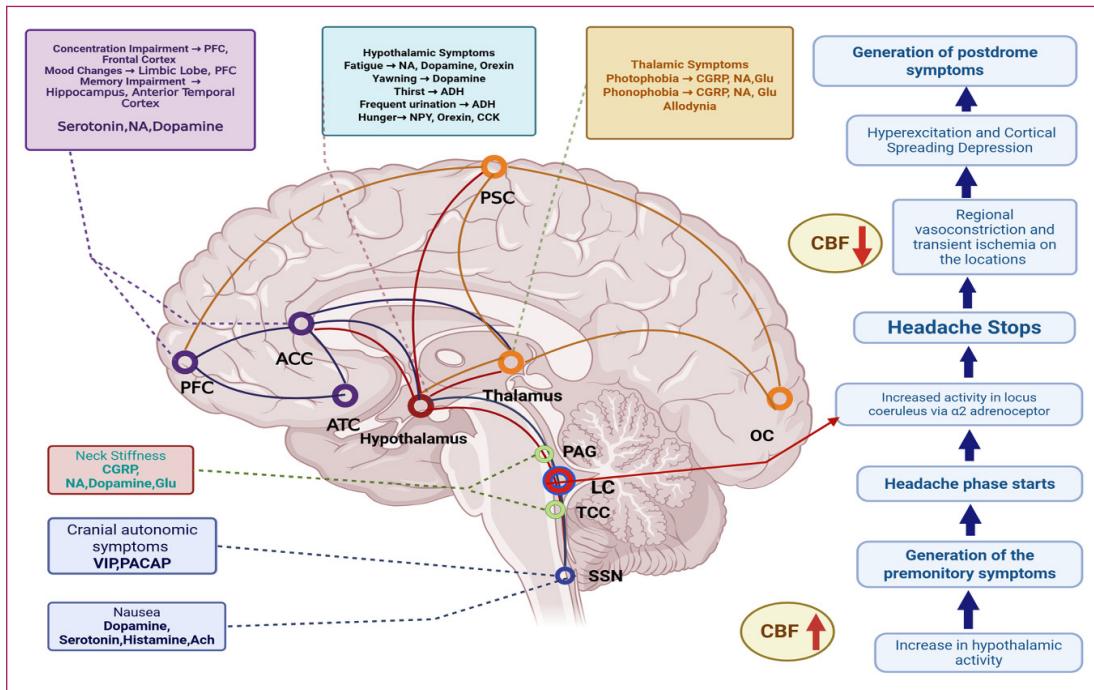
**INTRODUCTION:** Migraine is a primary headache syndrome characterized by attacks of headaches preceded and followed by specific symptoms under appropriate exogenous and endogenous conditions. According to the Global Burden of Disease 2019 Study, (1) migraine is the second most common cause of disability worldwide after stroke. Migraine broadly consists of four non-obligatory phases: the premonitory phase, the aura, the headache phase, and the postdrome phase.

Migraine postdrome is a series of symptoms that begin after a migraine attack and can last for days. These symptoms were vaguely defined in the International Classification of Headache Disorders, 2nd edition (ICHD-2) (2). Still, in ICHD-3 (3), they were clearly defined as symptoms that can last up to 48 hours following the end of a headache. These symptoms begin after the headache attack and may last for several days. Despite highly heterogeneous results, the average duration is 12-18 hours (4,5). Although the pathophysiology of postdromal symptoms (PS) is not yet fully understood, studies are ongoing to elucidate possible mechanisms. Due to the general similarity between migraine prodromal symptoms and PS (6, 7), it is reasonable to suggest that similar mechanisms are involved; however, functional studies have shown that the mechanisms are different (8, 9).

Prodromal symptoms are transient symptoms associated with a decrease in cerebral blood flow (CBF) in certain anatomical regions, such as the periaqueductal gray matter (PAG), trigeminal nucleus, anterior temporal region, hypothalamus, and the thalamus as a result of alpha 2-related vasoconstriction by the locus ceruleus during a headache (Figure 1) (10). In a study, CBF values were examined at the onset of premonitory and postdromal symptoms in a model similar to spontaneous migraine attacks (11). An increase in CBF was observed during premonitory symptoms, while a decrease in CBF was observed during PS (11, 12). Cortical hyperexcitability (CH) in migraineurs also contributes to these symptoms through cortical spreading depression (CSD) (13). Functional imaging studies in recent years have shown a decrease in CBF and the effect of CH, but the impact of CSD has not been demonstrated.

Postdrome is the least studied phase of migraine. Especially in the last decade, studies on the symptomatology and prevalence of this phase have increased. In the few studies available, most of the migraine postdrome studies were retrospective; hence, we know little about this subject. Recent studies on premonitory symptoms have shown that these symptoms may increase the burden of the disease and contribute to migraine-related disability (14, 15). In a multicenter study, it was reported that both premonitory symptoms and PS could cause an increase in MIDAS scores (16). We believe that similar anatomical regions may be involved in the formation of premonitory symptoms and PS. Still, different mechanisms may be at play, leading to varying effects on migraine-related disease burden and disability.

Recently, cognitive impairments that occur during both the ictal (migraine attack) and interictal (between attacks) periods of migraines have gained attention (17, 18). These cognitive difficulties are believed to negatively impact the quality of life for individuals suffering from migraines (18).



**Figure 1.** Potential Anatomical Regions and Hypothesized Mechanism for Generation of Non-Headache Symptoms in Phases of Migraine

ACC: anterior cingulate cortex; Ach: acetylcholine; ADH: antidiuretic hormone; ATC: anterior temporal cortex; CBF: Cerebral blood flow; CCK: cholecystokinin; CGRP: calcitonin gene-related peptide; Glu: glutamate; LC: Locus coeruleus; NA: noradrenaline; NPY: neuropeptide Y; PACAP: pituitary adenylate cyclase-activated peptide; PAG: periaqueductal gray matter; PFC: prefrontal cortex; PSC: primary somatosensory cortex; OC: occipital cortex; SSN: superior salivatory nucleus; TCC: trigeminocervical chain; VIP: vasoactive intestinal peptide

Therefore, we aimed to determine the frequency of PS in migraine patients, compare patients with and without PS in terms of quality of life, migraine-related disability, and cognitive dysfunction, and finally, identify the risk factors causing PS and the parameters affecting the decline in quality of life.

## METHODS

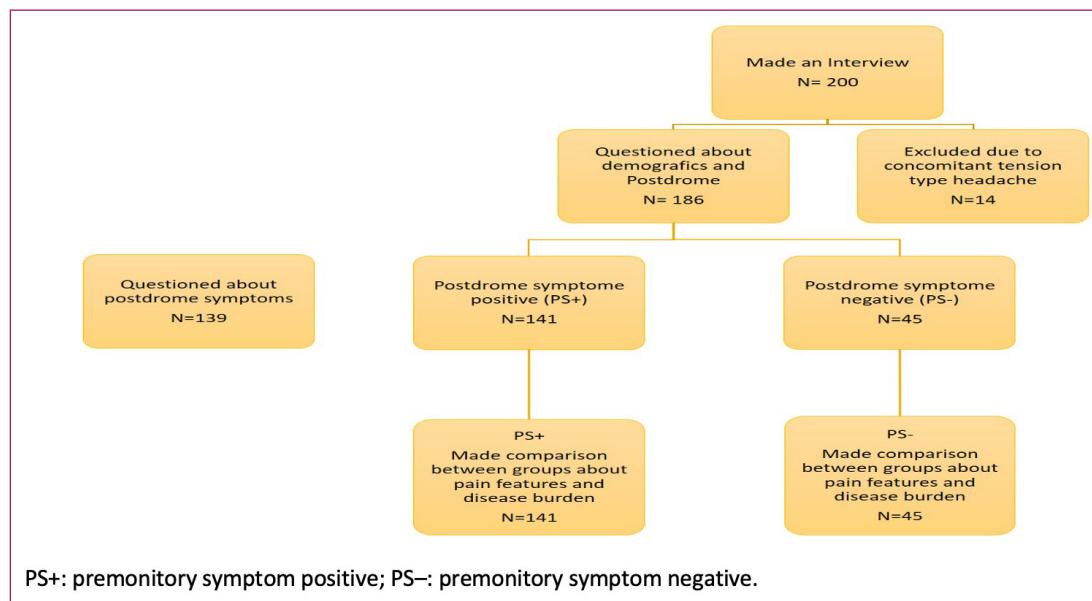
### 1. Study design and setting

This cross-sectional study was conducted at Mersin University Hospital, adhering to the principles of the Declaration of Helsinki (19). Approval was obtained from the Mersin University Clinical Research and Ethics Committee (approval No. E-78017789-050.01.04-1740527). We included a total of 186 patients aged 18–70 years who presented with migraine with or without aura, who applied to Mersin University Hospital between September 2021 and October 2022, and met the diagnosis of migraine with aura and migraine without aura according to ICHD-3[3]. We informed all the patients and obtained their consent before conducting the procedure. Exclusion criteria were cluster headache, hemiplegic migraine according to ICHD-3[3]; suspected migraine diagnosis, medication-overuse headache, and tension-type headache added to migraine. Power analysis determined that a sample of 180 participants was required to achieve 80% power, considering the expected prevalence and effect size detected in PS.

We categorized 11 PS (fatigue, GI symptoms, urinary symptoms, cognitive difficulties, neck stiffness, photophobia, phonophobia, osmophobia, mood changes, hunger, dizziness) and identified and modified these symptoms according to the recent electronic diary study of PS (7).

## 2. Procedure

Patients who met the inclusion and exclusion criteria and provided consent were examined to determine if they had migraines. The face-to-face interview included demographic information, number of monthly headache days (MHD), visual analogue scale (VAS) to assess pain intensity and the duration of migraine attacks. The potential overuse of medication alongside migraines, the presence of tension-type headaches, and whether participants were using prophylactic or interventional treatments for their migraines were also asked. After this preliminary interview, participants were accepted into the study (Figure 2).



**Figure 2.** The study design (flow chart)

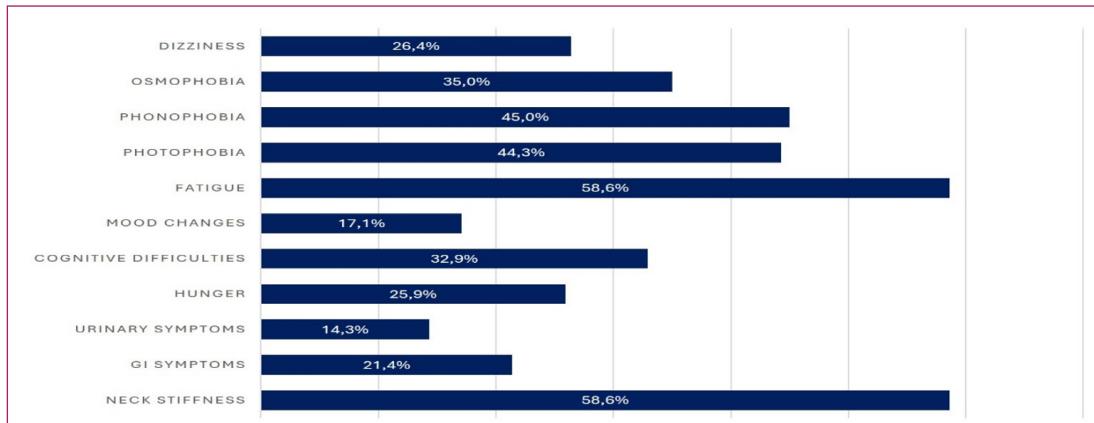
We asked participants if they experienced any symptoms after their headaches ended. Those who reported one or more PS were classified as PS positive (PS+). We then inquired which of the 11 identified PS were experienced by the PS+ participants. Additionally, we asked those who reported PS whether these symptoms began after more severe headaches or lighter ones. Next, we applied three different scales to assess disability and quality of life for patients with and without PS: the Migraine Disability Assessment (MIDAS), [20] the European Health Impact Scale (EUROHIS-8), [21] and a shortened version of the World Health Organization Quality of Life scale, as well as the Migraine Attack Related Subjective Cognitive Scale (Mig-SCOG). [22] .MIDAS scores are calculated as the total number of days lost due to headaches over the past three months, without applying grades to the scores. The EUROHIS-8 scale consists of 8 questions that assess quality of life, with each question scored from 1 to 5. The overall score ranges from 8 to 40, where lower scores indicate a lower quality of life. The Mig-SCOG is a subjective measure used to evaluate cognitive impairment during migraine attacks, comprising nine questions that assess various cognitive domains. Scores on this scale range from 0 to 18, with higher scores indicating greater cognitive impairment.

## 3. Statistical analysis

We conducted the statistical analysis of the study in three stages. In the first stage, we processed the demographic data of the patients using the statistical program 'TIBCO STATISTICA ver. 13.5.0' (TIBCO Software Inc.). We then calculated the PS proportion for the entire group using descriptive

statistical analysis. In the second stage, for the questions directed to the patients with PS, we selected only the patient group with PS and determined the PS rates, when PS started, and how often they occurred. We try to find a relationship between the presence of PS and other parameters by using regression analyses. In the third stage, we divided the patients into two groups based on the presence of PS: those with postdrome symptoms (PS+) and those without (PS-) (PS+: n=141, PS-: n=45). We then compared pain severity, the number of headache days per month, and the results of various scales between these two groups. For normality analysis, we used the Kolmogorov-Smirnov test. We applied a t-test to compare numerical data with a normal distribution and the Mann-Whitney U-test for numerical data that did not follow a normal distribution. Afterward, we conducted regression analyses to identify potential factors that may influence quality of life measures. Statistical significance was determined using p-values, with  $p < 0.05$  considered statistically significant.

**RESULTS:** A total of 186 participants, including 159 (85.0%) women and 27 (14.4%) men, were included in the study. Of the participants, 38 (20.4%) had chronic migraine, and 148 (79.6%) had episodic migraine. One hundred forty-one patients (75.8%) reported postdromal symptoms following a headache. The most common three postdromal symptoms were neck stiffness, fatigue/weakness, and phonophobia, respectively. The symptom frequencies among PS+ patients are summarized in Figure 3.



**Figure 3.** Proportion of individuals with PS experiencing each postdromal symptom (n=141).

The numbers in each bar represent the proportion of individuals with postdromal symptoms who experienced these symptoms.  
PS: Postdromal symptom

### **Postdromal Symptom Analysis**

Patients experiencing postdromal symptoms (PS) were asked to compare the pain they felt during episodes of these symptoms with the pain they experienced when these symptoms were absent. Among those who could make this comparison, 47.0% (31 out of 66) reported that their symptoms emerged after experiencing more severe pain. Additionally, 36.4% of patients with postdromal symptoms experience these symptoms after every headache attack.

In the regression analysis conducted with patients experiencing postdromal symptoms, cognitive difficulties and hunger were identified as factors contributing to higher scores on the Migraine-Specific Quality of Life (MIGSCOG) scale. Furthermore, mood changes and neck stiffness were found to decrease quality of life, while mood changes and dizziness were associated with higher scores on the Migraine Disability Assessment Scale (MIDAS). Table 1 summarizes the linear regression analyses between each postdromal symptom and parameters.

**Table 1.** Causative relation between postdromal symptoms and MIDAS, MIGSCOG, WHQOL-8 scores, respectively, via multivariate linear regression analysis

	Multivariate linear regression parameters (with Dummy variables)	β coefficient (standardized)	%95 CI for B	P
MIDAS	Mood Changes	13,673	5,394-21,951	<b>&lt;0,001</b>
	Dizziness	9,334	2,036-16,632	<b>0,013</b>
MIGSCOG	Cognitive Difficulties	3,043	1,624-4,462	<b>&lt;0,001</b>
	Hunger	1,831	0,307-3,356	<b>0,019</b>
WHOQL-8	Mood Changes	-0,438	-0,728 - -0,149	<b>0,003</b>
	Neck Stiffness	-0,295	-0,518 - -0149	<b>0,010</b>

CI: Confidence Interval; MIDAS: Migraine Disability Assessment Scale; MIGSCOG, Migraine Attack Related Subjective Cognitive Scale; WHOQL-8: European Health Impact Scale.

When examining risk factors for the development of PS using simple regression analysis, the presence of aura, the presence of premonitory symptoms, high MIDAS scores, and high MIGSCOG scores were all found to contribute significantly to this condition ( $p<0.001$ ). Multivariate analyses revealed that premonitory symptoms and aura directly influence the presence of PS, even within complex multivariate models. The regression analyses are detailed in Table 2.

**Table 2.** Risk factors for the generation of postdrome symptoms

	Univariate Regression Analysis		Multivariate Regression analysis	
	OR (CI 95%)	P	OR(CI95%)	P
Age	1.01(0.97-1.04)	0.778		
Male	0.59(0.24-1.41)	0.234		
MwA	<b>4.77(1.77-12.88)</b>	<b>0.002</b>	<b>3.25(1.13-9.38)</b>	<b>0.029</b>
Chronic Migraine	1.37(0.61-3.04)	0.444		
MHD	0.99(0.95-1.05)	0.811		
Presence of Postdrome symptoms	<b>6.76(3.22-14.22)</b>	<b>&lt;0.001</b>	<b>5.04(2.19-11.61)</b>	<b>&lt;0.001</b>
VAS	0.99(0.80-1.23)	0.935		
MIDAS	<b>1.02(1.00-1.04)</b>	<b>0.048</b>	1.01(0.98-1.03)	0.617
MIGSCOG	<b>1.16(1.06-1.25)</b>	<b>0.001</b>	1.09(0.99-1.20)	0.077
Oral preventatives	2.09(0.75-5.77)	0.155		

CI: Confidence Interval; MIDAS: Migraine Disability Assessment Scale; MIGSCOG: Migraine Attack Related Subjective Cognitive Scale; OR: Odds ratio; MwA: Migraine with Aura; MHD: monthly headache days

### Comparison between the PS+ and PS- groups

Demographic data for the PS+ and PS- groups are summarized in Table 3. The groups have similar distributions in terms of age and gender. No significant differences were found between the groups in terms of pain intensity and MHD. There are significantly more patients with MwA in the PS+ group. Additionally, premonitory symptoms were significantly more in the PS+ group compared to the PS- group.

**Table 3.** Comparison of demographic features between the PS+ and PS- groups

	<b>(PS+ N:141)</b>	<b>PS- (N:45)</b>	<b>p</b>
Age ±SD	35,88 ± 0,88	35 ± 1,69	0.780
Female gender (%n)	123(87.2%)	36(80.0%)	0.230
Chronic Migraine(%n)	27(19.1%)	11(24.4%)	0.443
MHD (IQR)	5(3-10)	6(4-10)	0.535
VAS (IQR)	8(7-9)	8(7-9)	0.718
MwA (%n)	54(38.6%)	5(11.6%)	<b>&lt;0.001</b>
Presence of premonitory symptoms (% n)	121(85.8%)	19(42.2%)	<b>&lt;0.001</b>
On Oral preventatives (%n)	29(20.7%)	5(11.1%)	0.148

IQR, interquartile range; MHD, monthly headache days; MwA: Migraine with Aura; PS+: postdrome symptom positive; PS-: postdrome symptom negative; SD: Standard Deviation; VAS: visual analog score.

\*For parameters given as mean±standard deviation was used Independent-t test, for parameters given as median (IQR), the Mann-Whitney U-test was used to obtain p-values. For parameters given as frequency, the chi-square test was used to obtain p-values.

MIDAS and MIGSCOG scores were significantly higher in the PS+ group. WHOQL-8 scores were significantly lower in the PS+ group (Table 4). Based on these results, the PS+ group has more migraine attack-related cognitive problems and migraine-related disabilities. The quality of life of the PS+ group is significantly lower than that of the PS- negative group.

**Table 4.** Comparison between PS+ and PS- groups in terms of quality of life, migraine-associated disability, and migraine headache-related cognitive dysfunction

	<b>PS+ (n:141)</b>	<b>PS- (n:45)</b>	<b>p *</b>
MIDAS (IQR)	25.5(14.0-40.0)	17.0(8.5-30.0)	<b>0.028</b>
MIGSCOG ± SD	10.21 ± 4.39	7.44± 4.40	<b>&lt;0.001</b>
WHOQL-8 ± SD	3.13 ± 0.69	3.51± 0.64	<b>0.001</b>

MIDAS: Migraine Disability Assessment Scale; Mig-SCOG, Migraine Attack Related Subjective Cognitive Scale; PS+: postdrome symptom positive; PS-: postdrome symptom negative; WHOQL-8: European Health Impact Scale

\*For parameters given as mean ± standard deviation, an independent t-test was used; for parameters given as median (IQR), the Mann-Whitney U-test was used to obtain p-values.

### Effects on quality of life in migraine patients

After determining that PS+ patients had a lower quality of life than PS- patients, we compared the factored that higher MIGSCOG scores, higher MHD, and the presence of MwA significantly reduced quality of life ( $R^2 = 0.240$ ). The Table-6 provides a detailed multivariate linear regression analysis. In conclusion, the most important factor affecting quality of life seems to be migraine attack-related cognitive dysfunction. Considering migraine-related cognitive dysfunction is more prevalent in PS+ patients, we can suggest that the presence of PS indirectly contributes to the decrease in quality of life in migraine patients.

**DISCUSSION:** In our study, we found that postdromal symptoms (PS) were present in 75.8% of patients. A meta-analysis examining the current proportion of postdromal symptoms reported a rate of 86% (23). However, this analysis had a very high heterogeneity index ( $I^2$ ), indicating variability among the included studies. Additionally, the small number of studies considered in the meta-analysis may have influenced the findings. In a recent study, the prevalence of PS was observed to be 80.7% (14).

Determining the proportion of PS in patients can be challenging due to methodological issues. Although the concept of PS is relatively new, similar methodological problems related to assessing premonitory symptoms have been discussed previously. Among these issues is the retrospective nature of many studies, as well as the practice of asking patients about symptoms without directing their attention to specific symptoms, which has resulted in a lower than expected number of patients reporting premonitory symptoms (24, 25). We believe that comparable issues may be present in studies aimed at identifying postdromal symptoms. Furthermore, in a multicenter pre-post headache study conducted in 2021, we noted the absence of a symptom checklist, resulting in a reported postdromal rate of only 60.2% (16).

In our study, fatigue, stiffness in the neck, and phonophobia were quite common in the PS+ patient group. Fatigue was the most common symptom observed in the studies conducted (5, 14, 23, 26). The symptom of neck stiffness observed in our research is more common compared to other studies. (5, 14, 23, 26). This may be because patients describe the neck pain symptom during the attack as if it were a PS. Although neck stiffness is a common symptom in premonitory symptoms, the rate may be lower in postdromal symptoms (27, 28). More studies are needed to confirm this.

In our study, cognitive impairment and hunger symptoms in our PS patients may be related to cognitive problems seen in patients with headaches. We did not find any previous study in the literature investigating the relationship between PS and these types of parameters. Only one study has examined the relationship between specific premonitory symptoms and pain intensity, pain duration, and the number of painful days, suggesting that some premonitory symptoms may be associated with these parameters (14). In another study, mild increases in MIDAS scores were observed in patients with neuropsychiatric and sensory PS (26). The interesting point here is that both premonitory symptoms and postdromal symptoms occur, common anatomic regions (Fig. 1). For example, the symptom of neck stiffness emerges as a premonitory symptom as a result of increased activity in the periaqueductal grey matter (PAG). In contrast, it emerges as a postdromal symptom secondary to decreased CBF in the PAG (9, 11, 29). Since the same anatomical regions do not show activity at the same rates in every migraine patient, these symptoms may manifest differently in patients. These different anatomical localizations may influence chronicity, migraine-related disability, or quality of life in migraine patients to varying degrees. Although no definitive study has been conducted on this topic, we believe that further clinical research on these symptoms will shed light on this issue.

Potential risk factors contributing to the presence of PS include premonitory symptoms, higher MIGSCOG scores, and migraineurs with aura. In a recent study aimed at identifying risk factors for PS, female gender, the presence of premonitory symptoms, and the number of monthly headache days were observed (14). Even when considered separately, the number of monthly headache days did not emerge as a risk factor in our study. However, MIDAS scores were a risk factor in univariate analyses; nevertheless, since the significance value was insufficient in multivariate analyses, we believe that pain frequency or MIDAS scores are not risk factors for the occurrence of PS.

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Although we did not find significant differences in pain intensity and MHD between the PS+ and PS- groups, an increase in migraine-related disability and migraine attack-related cognitive impairment can be noted in the PS+ group. Although there were no significant differences in the number of headache days and pain intensity, quality of life was significantly reduced in this group. We believe that the main reason for this is the contribution of non-pain-related ictal and interictal migraine features (30). Although we do not know all of these ictal and interictal migraine features, we believe that the MIGSCOG score contributes significantly to this condition. In a study, the WHODAS scale was used to assess quality of life, and both premonitory and PS were found to have a weak correlation with a reducing effect on quality of life (14). Another multicenter study reported that patients with premonitory symptoms and postdromal symptoms experienced more migraine-related disability and a higher disease burden. In contrast, those who experienced both phases had lower quality of life (16).

In our study, we summarized the factors contributing to reduced quality of life in Table 5. When considered individually, the presence of postdrome and premonitory symptoms was associated with a decrease in quality of life. However, in multivariate analyses, we determined that auricular migraine, MIGSCOG, and MHD were associated with a reduction in quality of life, and that this model could have high explanatory power when combined with the adjusted R<sup>2</sup> value (Table 6). We did not find any studies in the literature that directly observed the effect of PS on quality of life.

**Table 5.** Simple linear regression (univariate) analysis in quality of life measures

Parameter	B Coefficient (unstandardized)	B Coefficient (standardized)	t	p	R <sup>2</sup> (Adjusted)
Age	-0.006	-0.09	-1.21	0.229	0.002
MIGSCOG	-0.056	-0.36	-5.27	<b>&lt;0.001</b>	0.126
MIDAS	-0.111	-0.31	-4.30	<b>&lt;0.001</b>	0.091
MHD	-0.030	-0.28	-3.90	<b>&lt;0.001</b>	0.071
VAS	-0.107	-0.24	-3.28	<b>0.001</b>	0.050
Oral preventions	-0.023	-0.013	-0.18	0.860	0.000
MwA	-0.245	-0.165	-2.26	<b>0.025</b>	0.022
Presence of Postdrom	-0.424	-0.26	-3.66	<b>&lt;0.001</b>	0.063
Presence of Premonitory	-0.408	-0.26	-3.59	<b>&lt;0.001</b>	0.060

MIDAS: Migraine Disability Assessment Scale; MIGSCOG: Migraine Attack Related Subjective Cognitive Scale; MwA: Migraine with Aura; MHD, monthly headache days; VAS: Visual Analogue Scale

**Table 6.** Multivariate modeling to assess the factors that contribute to a reduced quality of life.

Parameter	B Coefficient (Unstandardized)	Beta (Standardized Coefficient)	t	p	Lower Bound 95% CI	Upper Bound 95% CI	R <sup>2</sup> (Adjusted)
MwA	-0.186	-0.126	-1.82	<b>0.070</b>	-0.389	0.016	<b>0.240</b>
MHD	-0.023	-0.218	-2.794	<b>0.006</b>	-0.039	-0.007	
VAS	-0.059	-0.131	-1.861	0.064	-0.122	0.004	
MIDAS	-0.002	0.049	-0.604	0.547	-0.007	0.004	
MIGSCOG	-0.039	-0.256	-3.534	<b>0.001</b>	-0.061	-0.017	
Presence of Premonitory	-0.158	-0.099	-1.316	0.190	-0.395	0.079	
Presence of Postdrome symptoms	-0.189	-0.115	-1.532	0.127	-0.433	0.055	

CI: Confidence Interval; MIDAS: Migraine Disability Assessment Scale; MIGSCOG: Migraine Attack Related Subjective Cognitive Scale; MwA: Migraine with Aura; MHD: monthly headache days; VAS: Visual Analogue Scale

Although there was no significant difference in quality of life between the PS+ group and the PS- group in terms of MHD, there was a noticeable decrease in the PS+ group in quality of life. We believe that this is due to the increased MIGSCOG scores in the PS+ group affecting quality of life. Approximately 30% of migraine sufferers experience cognitive problems in areas such as complex attention, working memory, immediate memory, and executive function during migraine attacks, before and after attacks, and during the interictal period (17, 31). Recent studies have identified co-occurrence between ictal and interictal cognitive dysfunction (18, 32). Although we did not measure interictal cognitive dysfunction, the cognitive impairments during the ictal period may reflect those during the interictal period.

Cortical hyperexcitability plays a significant role in the development of PS (33, 34). Increased cortical hyperexcitability and cortical spreading depression (CSD) contribute to the persistence of these symptoms even after the pain has subsided (29). Although the exact role of CSD is not fully understood, it is associated with the transient ischemic response that occurs due to decreased cerebral blood flow (CBF) and contributes to the development of symptoms (11, 12). Research indicates that cortical hyperexcitability is heightened in migraine patients compared to the healthy population, and this increase is even more pronounced in those with chronic migraine. Some migraine patients experience symptoms even when not having a migraine attack; these symptoms may include interictal photophobia, difficulties with concentration, mood disorders, and fibromyalgia (32).

Additionally, a subset of migraine sufferers experiences a loss of habituation to pain. In these individuals, the likelihood of chronic migraine is high, and they are also at a higher risk for disorders such as fibromyalgia, depression, and anxiety, which can negatively impact quality of life, other than migraine itself (35, 36). These changes are thought to be related to alterations in serotonergic and dopaminergic pathways in the brain (34). One of the mechanisms responsible for the formation of these biochemical changes, the loss of habituation, development of pain behaviour, and pain anxiety is cortical hyperexcitability in migraine sufferers (36). Previously associated only with aura, the importance of this condition has increased with functional studies. In our study, we hypothesized that cortical hyperexcitability may be increased in the group of patients with increased MIGSCOG scores. Therefore, the quality of life of this group may be reduced due to both migraine-related and non-migraine-related causes. More studies are needed to investigate whether these problems persist in the interictal period, especially in migraine patients with cognitive issues. There are no related studies evaluating cortical hyperexcitability in individuals with predominant PS. However, we believe that not only migraine but also migraine-related conditions and conditions that develop outside of headache attacks may contribute to a decrease in quality of life.

**CONCLUSION:** In the PS+ patient group, quality of life was reduced and migraine-related disability was increased compared to the PS- patient group, independent of pain frequency and severity. An increase in the disease burden in the PS+ patient group may be migraine attack-related cognitive dysfunction. This cognitive dysfunction that occurs during migraine attacks may also present in the interictal period and may emerge as a parameter explaining the disease burden in migraine patients. Some symptoms that occur in the postdromal period may affect quality of life or disease burden. These symptoms, which arise from dysfunction in different anatomical regions, may assist in developing different therapeutic approaches for migraine patients in the future.

**LIMITATIONS AND STRENGTHS:** Firstly, the sample size in this study is small compared to recent studies. Therefore, the power of the study may be low. Due to the cross-sectional design of our study, recall bias may occur, especially in determining PS symptoms. Electronic diary studies conducted to prevent this recall bias could overcome this issue. Another issue is that we divided our study into groups based solely on the presence or absence of PS symptoms, without considering the frequency of these symptoms. This may weaken the support for the hypothesis we presented.

The strengths of the study include conducting interviews with patients. Therefore, we carefully and meticulously established migraine diagnoses based on symptom frequencies and other parameters. As a result, we were able to overcome confirmation bias. We asked our patients about their symptoms using a checklist, which allowed us to identify symptom-related categorization and symptom counts without facing any additional bias other than recall bias. The best aspect of the study was that the data adhered to distribution, enabling us to strengthen our hypothesis through parametric tests and regression analyses.

**Key words:** cognitive dysfunction, migraine disability assessment questionnaire, migraine disorders, migraine prodrome, quality of life

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**ORAL PRESENTATIONS**

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**OP-9**

## **SHORT-TERM EFFICACY OF GREATER OCCIPITAL NERVE BLOCK IN CHRONIC MIGRAINE: INSIGHTS FROM A SINGLE-CENTER EXPERIENCE**

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**BACKGROUND:** Chronic headaches, affecting 1.4-2.2% of the global population for  $\geq 15$  days/month, substantially impair daily functioning and impose a socioeconomic burden. Among treatment options, greater occipital nerve block (GONB) has emerged as a promising intervention, especially for occipital neuralgia, cervicogenic headache, cluster headache, and migraine. Blocking the greater occipital nerve (GON) can relieve pain, though response varies. This study aimed to evaluate the short-term efficacy of GONB in chronic migraine, assessing changes in pain severity, attack frequency, and pain-free duration, and exploring the influence of migraine type, headache laterality, and lifestyle factors.

**METHODS:** This single-center retrospective study included 30 chronic migraine patients treated with bilateral GONB between 12/2024-06/2025. Demographic data, migraine characteristics (type, laterality, duration, baseline attack frequency), and lifestyle factors (smoking, alcohol, exercise) were recorded. All patients received bilateral GONB using 0.5% bupivacaine diluted with saline, plus bilateral trapezius trigger-point injections with 2% lidocaine. Treatment was administered weekly for four sessions, and, based on patient symptoms, continued monthly thereafter for up to a maximum of seven sessions. The interval to the first headache recurrence after the last block was documented. Pre-post comparisons were made with the Wilcoxon signed-rank test; subgroup differences were analyzed with Mann-Whitney U or Chi-square/Fisher's exact test; correlations were analyzed using Spearman's rho ( $p < 0.05$ ).

**RESULTS:** Mean age was  $43.2 \pm 9.6$  years; 96.7% were female. No alcohol use was reported; 16.7% smoked, and 13.3% exercised regularly. Pain intensity and attack frequency significantly decreased after GONB (both  $p < 0.001$ ). Patients with bilateral migraine had higher baseline attack frequency ( $p = 0.050$ ), but this difference disappeared post-treatment. Unilateral migraine patients had a longer pain-free interval before recurrence ( $p = 0.043$ ). The median recurrence interval after the last block was 15 days (range: 1-120). Non-significant trends suggested longer relief in exercisers, non-smokers, and those not using acute headache medication.

**CONCLUSION:** Bilateral GONB with trapezius trigger-point injections produced significant short-term reductions in pain and attack frequency, consistent with prior studies. The median 15-day recurrence interval is at the lower end of published ranges. Pain laterality emerged as a potential predictor: unilateral migraine was associated with longer relief, while aura status showed no significant effect. Observed trends with exercise and smoking suggest lifestyle may influence the durability of the benefit. In this cohort, GONB yielded substantial short-term improvement in chronic migraine, with more durable relief, especially in unilateral cases. The variability in recurrence intervals and lifestyle associations highlights the need for individualized re-block scheduling and integration with non-pharmacological strategies. Prospective studies should confirm laterality as a predictor, separate GONB effects from co-interventions, and explore whether combined lifestyle approaches can extend the pain-free period.

**Key words:** migraine, headache, neuralgia

## OP-10

# **MIGRAINE EPIDEMIOLOGY AND DISEASE BURDEN IN TURKEY BASED ON DATA FROM 81 PROVINCES: A PILOT STUDY**

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**INTRODUCTION:** Migraine is a chronic neurological disorder characterized by recurrent episodes of moderate to severe headache, often accompanied by nausea, photophobia, and phonophobia. According to the Global Burden of Disease Study, migraine is one of the leading causes of disability worldwide and significantly affects the quality of life, particularly among individuals in their most productive years (1). While global data suggest a high prevalence and substantial disease burden, there remains a lack of nationally representative data from Turkey that can guide healthcare planning and resource allocation. The aim of this pilot study is to provide preliminary insight into the epidemiology of migraine in Turkey. By collecting data from all 81 provinces, this study explores the prevalence, severity, and geographic distribution of migraine and its impact on daily life using the MIDAS questionnaire (3). The findings aim to inform the development of future nationwide studies and evidence-based public health policies.

## **METHODS**

### **1. Study Design and Participants**

This study employed a cross-sectional design with data collection conducted between January and June 2025. Participants aged 18 years or older were recruited through a combination of online platforms and face-to-face community outreach efforts. Informed consent was obtained from all participants prior to inclusion.

### **2. Data Collection Instruments**

**The survey instrument consisted of six main sections:** demographic data (age, gender, province, education level, marital status, income status, urban vs. rural residence, and employment status), migraine screening questions aligned with the International Classification of Headache Disorders (ICHD-3) criteria (10), the Turkish version of the MIDAS scale (3), headache characteristics, healthcare utilization, and perceived causes and burden. Participants were asked if they had experienced headaches in the last 12 months, whether they had received a formal migraine diagnosis, and who provided the diagnosis (neurologist, general practitioner, other specialist, or self-diagnosed). Additional items included the number of headache days in the last 30 days, headache severity, type (throbbing, pressing, stabbing), duration, and triggers (stress, sleep disturbances, visual stimuli, hormonal changes, dietary factors). Further questions addressed the duration of headaches with and without medication, the most disturbing symptom of the headache, accompanying symptoms (e.g., nausea, photophobia, phonophobia), whether the participant believed the headache had a single cause, and whether attack medication relieved the pain within two hours. Healthcare usage questions covered emergency department visits, outpatient clinic consultations, medication use (acute and preventive), access to preventive therapy, specific types of prophylactic medications used (e.g., beta blockers, antiepileptics, antidepressants), and barriers to accessing preventive treatment.

### **3. Statistical Analysis**

All data were analyzed using descriptive and inferential statistics. Categorical variables such as gender and migraine prevalence were expressed as frequencies and percentages. Continuous variables, including MIDAS scores, were reported as means and standard deviations. Gender comparisons were conducted using independent samples t-tests. Provincial differences were examined to detect regional variability. All analyses were performed using appropriate statistical software.

## **RESULTS**

### **1. Participant Demographics**

A total of 1395 participants were included in the study. Among them, 905 (64.9%) identified as female and 490 (35.1%) as male. The average age was 34.2 years (SD = 12.6), ranging from 18 to 78 years. Participants represented all 81 provinces of Turkiye, with Mersin having the highest number of responses, followed by Istanbul, Ankara, and Izmir. Regarding educational attainment, 41.2% had completed a university degree or higher, 45.6% had a high school education, and 13.2% had primary education or less. In terms of marital status, 58.4% were single, 36.1% married, and 5.5% divorced or widowed. Monthly income distribution showed that 42.3% earned below the national average, 38.9% reported average-level income, and 18.8% had above-average income. Additionally, 68.7% of participants reported living in urban areas, and 31.3% in rural settings. Employment status revealed that 56.2% were employed, 23.5% were students, and 20.3% were unemployed or retired.

### **2. Migraine Prevalence and Diagnosis Confirmation**

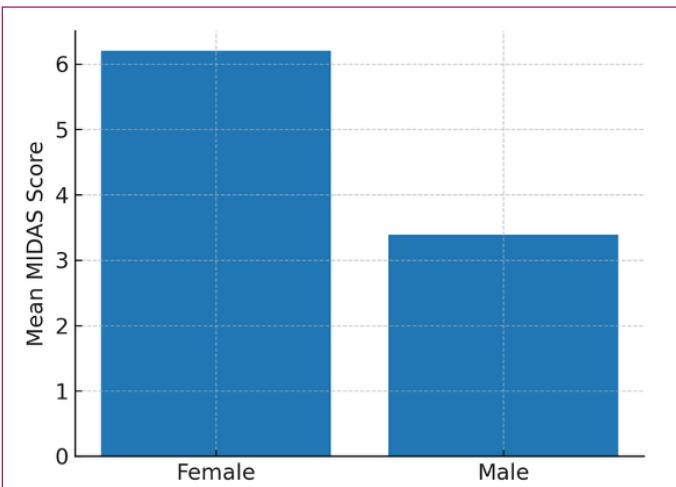
Overall, 34.6% of respondents self-reported a history of migraine. Among them, 86.3% reported experiencing headaches in the last 12 months. Of those, 47.9% had received a formal diagnosis of migraine. Among those diagnosed, 68.5% reported being diagnosed by a neurologist, 21.4% by a general practitioner, and 10.1% either by another specialist or self-diagnosis. Prevalence was significantly higher in females (42.5%) compared to males (20.2%). The highest prevalence was observed in individuals aged 25-44 years.

### **3. MIDAS Scores and Disability Burden**

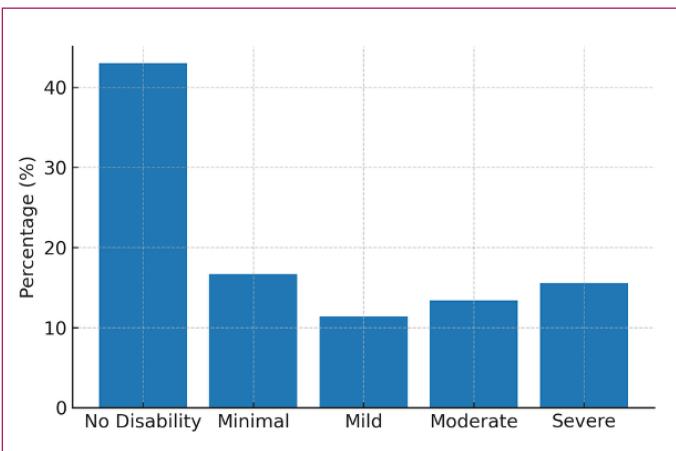
The mean MIDAS score for all participants was 5.25 (SD = 7.52). When stratified by gender, females had an average score of 6.20 (SD = 8.04), while males had a lower average score of 3.39 (SD = 5.97), indicating a greater level of migraine-related disability among women. Disability levels, based on MIDAS grading, were categorized as follows: 43%: No disability 16.7%: Minimal (1-5) 11.4%: Mild (6-10) 13.4%: Moderate (11-20) 15.6%: Severe (>20)

### **4. Headache Characteristics and Attack Features**

Among migraineurs, the average number of headache days in the last 30 days was 7.2 (SD = 4.9). In terms of pain severity, 44.6% reported moderate pain, 34.1% severe pain, and 21.3% mild pain. Regarding headache character, 49.2% described their headaches as throbbing, 29.4% as pressing, and 21.4% as stabbing. Average headache duration was 12.4 hours (SD = 7.6). When untreated, headaches lasted on average 18.1 hours (SD = 8.2), while with medication, duration was reduced to an average of 6.7 hours (SD = 5.3). The most disturbing aspects of the headaches reported were pain intensity (38.5%), nausea (23.7%), and photophobia/phonophobia (18.4%). Accompanying symptoms included nausea (52.3%), photophobia (47.6%), phonophobia (39.2%), and dizziness (26.5%). Approximately 61.4% of migraineurs believed their headaches were caused by a single factor, most commonly stress (49.1%). Among participants using acute attack medication, 42.7% reported that it effectively reduced pain within two hours.



**Figure 1.** Mean MIDAS Score by Gender



**Figure 2.** Distribution of MIDAS Disability Grades

## 5. Health Service Utilization

Among migraineurs, 27.6% reported at least one emergency department visit in the past year due to headache, and 48.2% visited an outpatient clinic. Acute medications were used by 61.5% of migraineurs, with the most common being NSAIDs (38.7%) and triptans (22.8%). Only 18.3% reported using any form of preventive therapy. Among them, the most commonly used medications were beta blockers (31.4%), antiepileptics (24.6%), and tricyclic antidepressants (17.9%). The most frequently cited barriers to preventive treatment were medication side effects (38.1%), cost (27.4%), and lack of access to specialists (19.6%).

## 6. Geographic Distribution

All 81 Turkish provinces were represented in the sample. The number of responses varied by region, with Mersin having the highest count, followed by Istanbul, Ankara, and Izmir. Prevalence rates and average MIDAS scores exhibited interprovincial variability, which may reflect underlying socioeconomic, healthcare access, or cultural differences.

**DISCUSSION:** This pilot study provides a snapshot of migraine prevalence and associated disability across Turkey. The findings reveal that migraine is common and disabling, especially among women

(1,2,6,7). Gender-based disparities in prevalence and severity align with international data (2,9) and may be linked to hormonal factors, stress exposure, and healthcare-seeking behaviors (8). The addition of questions regarding education, marital status, income, and headache characteristics allows for a more nuanced understanding of the migraine burden (6). The frequency of attacks, common triggers, and duration data are in line with existing literature (7,9) and support the development of patient-specific management strategies. Healthcare utilization findings reflect suboptimal access to preventive treatments, despite a high rate of acute medication use and health service contact (4,5,10). Barriers such as medication side effects, cost, and specialist access should be addressed through healthcare system reforms and patient education. The study also identifies regional differences in migraine burden, which warrant further investigation (7,9). These differences could inform regional health policies and resource allocation strategies. While the MIDAS tool has demonstrated utility in assessing functional impairment (3), future research could benefit from incorporating clinical diagnoses and longitudinal follow-up.

**CONCLUSION:** Migraine imposes a significant burden on the Turkish population, with notable gender and regional disparities (1,2,6,7,10). The pilot data underscore the need for nationwide studies employing representative sampling and clinical validation. Health policy initiatives should prioritize early identification, patient education, and access to effective treatment to reduce the individual and societal impact of migraine (4,5).

**Key words:** Migraine, MIDAS, headache burden, Türkiye, epidemiology, gender difference, regional disparities, public health

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**OP-11**

## **THE ROLE OF KYNURENINE PATHWAY METABOLITES IN CLINICAL FEATURES AND CHRONIFICATION OF MIGRAINE**

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**BACKGROUND:** Migraine is a prevalent neurological disorder, yet its pathophysiology remains incompletely understood. While the role of neuroinflammation and metabolic dysregulation in migraine pathogenesis is increasingly recognized, the kynurenine pathway has gained attention due to its involvement in tryptophan metabolism. Our study aims to evaluate the relationship between kynurenine metabolism, the clinical characteristics of migraine, and the process of migraine chronification.

**METHODS:** This study was designed as a prospective, observational study and was conducted with a total of 81 participants including 27 with episodic migraines, 27 with chronic migraines, and 27 healthy controls, aged 18–50 years. Data collected included age, sex, pain type and location of migraine, attack frequency, severity, disease duration, and body mass index. Blood samples taken during the interictal phase were analyzed for Tryptophan (Trp), L-kynurene (KYN), 3-hydroxykynurene (3-HK), and 3-hydroxyanthranilic acid dioxygenase (3-HAAO) levels using ELISA kits.

**RESULTS:** In the comparison between healthy controls and migraine groups, age, sex, and body mass index were similar. However, the patient group exhibited significantly lower levels of Trp, KYN, 3-HK, and 3-HAAO compared to the control group ( $p <0.001$  for each). Laboratory analysis revealed higher levels of HAAO, 3-HK, KYN, and Trp in the episodic migraine group than in the chronic migraine group ( $p = 0.027$ ,  $p <0.001$ ,  $p <0.001$ ,  $p = 0.002$ , respectively). ROC analysis revealed that 3-HK was identified as an independent risk factor for CM (OR=0.403,  $p<0.001$ ). Painful headache days, monthly attack frequency, MIDAS, and HIT-6 scores were negatively correlated with HAAO, 3-HK, KYN, and Trp levels ( $p <0.005$  for each).

**CONCLUSION:** The study suggests that all four molecules are potentially involved in migraine clinical features and its chronification. Their levels decrease as migraine attacks worsen due to their neuroprotective effects. Future studies should explore targeted therapies to modulate kynurenine metabolism to prevent chronic migraine.

**Key words:** Kynurene, migraine, chronic, neuroinflammation, 3-hydroxykynurene

OP-12

# A BIBLIOMETRIC ANALYSIS OF MONOCLONAL ANTIBODY TREATMENTS USED IN MIGRAINE

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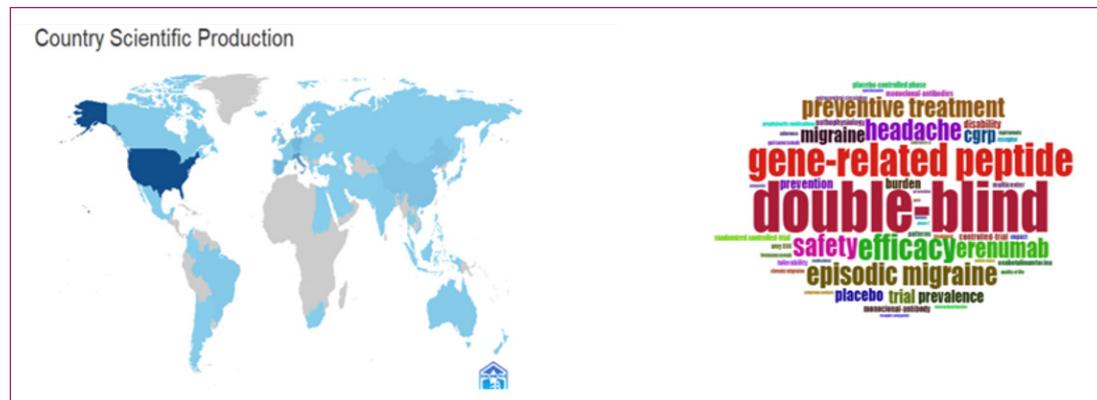
**BACKGROUND:** Migraine is a common neurological disorder, and in recent years, anti-CGRP monoclonal antibodies have emerged as an effective treatment option. This study aimed to assess research trends and scientific productivity on migraine and anti-CGRP monoclonal antibodies by reviewing publications on this topic between 1988 and 2024.

**METHODS:** The study conducted a search of the Web of Science (WoS) database using the keywords "migraine," "monoclonal antibodies," "anti-CGRP antibody," "fremanezumab," "galcanezumab," "epinezumab," and "erenumab." A total of 2873 articles were included in the study. Data were analyzed using Bibliometrix (Biblioshiny) and Excel 365. The distribution of publications by year, the most productive countries, journals, authors, and keywords were evaluated.

**RESULTS:** Our bibliometric study included 2873 publications between 1988 and 2024, with 5837 authors, 23664 references, and 1551 keywords plus. Publications increased particularly after 2016 and reached a peak in the 2020s. The most frequently published journals were: Headache (468), Cephalgia (450), Journal of Headache and Pain (418). The most productive authors were: Cohen JM (253), Ning X (194), Ashina M (183). The countries with the most publications were: USA (4960), Italy (2004), Spain (919), Germany (830), and Denmark (593). (Figure 1). Trending topics included concepts such as sensory neurons, trigeminovascular system, alpha-CGRP, and beta-CGRP.

**CONCLUSION:** This bibliometric analysis demonstrates that monoclonal antibodies are becoming an increasingly important research area in the treatment of migraine. The findings not only contribute to mapping the existing literature in this field but also provide a foundation for future research.

**Key words:** Migraine, monoclonal antibody, anti-CGRP, bibliometric analysis, WoS



**Figure 1.** Country Scientific Production and most relevant words

**OP-13**

## **RED EAR SYNDROME ASSOCIATED WITH CHRONIC MIGRAINE: A CASE REPORT EMPHASIZING TARGETED TREATMENT**

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**BACKGROUND:** Red Ear Syndrome (RES) is a rare clinical entity characterized by episodic erythema, burning, and pain in the external ear. Although often idiopathic, it has been associated with primary headache disorders, particularly migraine. We present a case of RES in a patient with chronic migraine and status migrainosus, highlighting the importance of recognizing and treating this underdiagnosed comorbidity

**CASE:** A 31-year-old male with a history of obsessive-compulsive disorder (OCD), previously operated papillary thyroid carcinoma, and venous angiomas on the body presented with a 15-year history of unilateral pulsatile headaches, predominantly on the right-side, accompanied by nausea, vomiting, and periorbital discomfort. Attacks occurred 6-8 times monthly and lasted 1-2 days. Triptans provided partial relief for acute attacks, but he had a history of medication overuse headache. Despite multiple prophylactic treatments including propranolol, valproic acid, venlafaxine (discontinued due to inadequate control OCD symptoms), paroxetine and agomelatine (for OCD) and botulinum toxin effectiveness was limited. Greater occipital nerve blocks provided transient relief. Two episodes of status migrainosus occurred in the past two years. Over the past three years, the patient developed right-sided auricular redness, burning, and numbness lasting about one hour. Initially mild and monthly, episodes progressed to nearly daily frequency over the past three months, causing disabling pain and functional impairment. Occasionally, they were followed by right-sided migraine attacks. Neurologic examination was normal. Cranial MRI and standard tractography revealed a left-sided 14×11 mm cavernoma in the left posterior superior temporal gyrus, considered likely coincidental as the red ear symptoms and migraines were primarily right-sided. Although a possible contralateral hyperactivation due to left-sided suppression was considered, tractography showed no abnormal activity in pain-related pathways. Routine EEG and prolonged EEG with sleep recording were normal. The clinical presentation was consistent with RES, leading to the initiation of indomethacin 25 mg twice daily. This resulted in a decrease in auricular episodes to once weekly, with significant reduction in pain and burning intensity. No adverse effects were reported. However, the patient declined further dose escalation due to concerns about side effects and satisfaction with his current condition. Venlafaxine was reintroduced for its partial benefit on migraine symptoms and fremanezumab injections are initiated as the next step for migraine prophylaxis.

**CONCLUSION:** This case underscores the importance of identifying RES in patients with difficult-to-control migraine. Ear-related symptoms can be easily overlooked but may independently contribute to disability. Targeted treatment, such as indomethacin, may offer effective symptom control even in the context of chronic migraine. A comprehensive evaluation of all pain and disability sources in migraine patients may improve both quality of life and therapeutic outcomes.

**Key words:** Red ear syndrome, migraine, auricular pain, indomethacin

**OP-14**

## **CAN RETINAL MIGRAINE CAUSE PERMANENT VISUAL LOSS? A CASE-BASED REFLECTION**

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**BACKGROUND:** Retinal migraine is a rare clinical entity characterized by recurrent, transient monocular visual disturbances, typically followed by migraine headache. It is traditionally considered a reversible phenomenon, with visual symptoms resolving completely between episodes. However, the possibility of permanent visual impairment remains uncertain and has rarely been reported. Herein, we question whether retinal migraine can lead to irreversible visual loss, highlighting a case where transient monocular visual disturbances were followed by persistent visual deficits without any other detectable reason.

**CASE:** A 44-year-old female with a history of migraine without aura since age 18 presented with new-onset transient monocular visual loss in her left eye. Episodes occurred exclusively upon awakening in the morning, lasted approximately 30 minutes. The first episode resolved completely. However, the subsequent three episodes (under flunarizin treatment) resulted in partial, persistent visual loss. Ophthalmologic examination after the second episode revealed central retinal artery occlusion in the left eye. Neurological examination was normal. Visual evoked potentials demonstrated a slight P100 latency delay in the left eye compared to the right. Brain MR-Angiography, MR-Venography and MRI showed only a few nonspecific white matter lesions. Secondary causes of transient monocular visual loss, including carotid artery stenosis, cardiac embolic sources, hypercoagulable states (including Factor V Leiden mutation, hyperhomocysteinemia, Protein S, C and anti-Thrombin-III positivity) and inflammatory conditions (including Systemic Lupus Erythematosus and Sjögren syndrome) were ruled out through serologic investigations and rheumatology/cardiology consultations. Cerebrospinal fluid analysis excluded optic neuritis, with negative oligoclonal bands and anti-MOG antibody. Malignancy ruled out via PET imaging. The patient had a history of rhinoplasty three months prior to the first episode, an anxiety disorder related to visual loss and heavy smoking, but no additional vascular risk factors. She was started on rivaroxaban for secondary prevention in addition to duloxetine. No further visual episodes were observed during a six-month follow-up. Notably, her migraine attack frequency decreased by more than 50%.

**CONCLUSION:** Although retinal migraine is generally considered reversible, this patient highlights its potential association with irreversible visual impairment. The occurrence of central retinal artery occlusion following recurrent transient monocular visual disturbances raises concerns about vascular complications in patients with migraine, especially those with additional modifiable risk factors like smoking. The pathophysiological link between retinal migraine and vascular events remains unclear, but this unique presentation contributes to the growing discussion regarding the vascular risks associated with migraine and its rare complications. Retinal migraine should be considered in the differential diagnosis of both transient and permanent monocular visual loss. While traditionally considered benign, clinicians should be aware of the potential irreversible ocular complications in retinal migraine, particularly in the presence of vascular risk factors. Careful exclusion of other causes, close monitoring, and risk factor management are essential.

**Key words:** retinal migraine, permanent visual loss, headache

**OP-15**

## **DIAGNOSTIC VALUE OF FIBRINOGEN ALBUMIN RATIO IN MIGRAINE ATTACKS**

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**BACKGROUND:** Migraine is a primary headache disease that is common in the world and negatively affects the life. Although its etiology is not clear, inflammation and hypercoagulation are involved in its formation. In many recent studies, fibrinogen albumin ratio (FAR) has been found to be an important biomarker in showing inflammation and procoagulation conditions. The aim of this study is to determine the value of FAR in diagnosis migraine attacks.

**METHODS:** This study included 50 patients diagnosed with migraine attacks and a control group of 50 healthy people. Demographic data of the patients and the control group were recorded. Clinical features such as disease duration, attack frequency, attack type (with or without aura) and symptoms accompanying the attack were recorded in migraine patients. Migraine attack severities were determined according to the Visual Analog Scale (VAS). Blood fibrinogen, albumin and FAR values were analyzed in migraine group during the attack and compared with control group.

**RESULTS:** The mean age of migraine patient group was  $36.26 \pm 9.87$  years and 41 were female. The mean age of control group was  $35.22 \pm 11.11$  years and 28 were female. No significant difference was found in fibrinogen and albumin values in migraine attack patients ( $p=0.098$ ), ( $p=0.492$ ). No significant difference was found in FAR in migraine attack patients. No significant relationship was found between disease duration, attack frequency and FAR ( $p=0.806$ ) ( $p=0.978$ ). In the receiver operating characteristic curve (ROC) analysis, FAR was not found to be a significant predictive value to diagnose migraine attacks ( $AUC=0.435$   $p=0.263$ ).

**CONCLUSION:** These findings suggest that FAR is not an adequate biomarker for diagnosing migraine attacks. However, to generalize all these findings, larger patient population studies are needed.

**Key words:** migraine, migraine attack, inflammation, hypercoagulation

## OP-16

### **HEADACHE CAUSED BY MONONUCLEOSIS INFECTION**

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**BACKGROUND:** Headache one of most complication during mononucleosis infection. Cause headache -during mononucleosis infection released cytokines causes inflammation and due to inflammation and swelling pain sensitive structures of the head gets irritated. Patient takes some remedies from headache, but any result, they don't pay attention to origin to headaches and other uncomfortable situation in there healthy. If we take detailed anamnesis, and type of headache, ask about last complications, unordinary things in there body health and during palpitation feel largeness some lymphoid, have to search at first for infections, especially mononucleosis infection. In late stage headache, encephalomalacia in the brain (due to ischemia or encephalitis), especially in the frontal lobe cause some psychiatric character, emotionality, and infiltration give severe headaches. Brain MRI (encephalomalacia area), positive EBV PCR analysis and enlarged lymph nodes, liver, spleen especially are the red flags for this illness.

**CASE:** A 34 year-old female patient suffered from severe headache, nausea, dizziness and emotionality, late thinking. Headache is permanent. She had angina, with high temperature and enlarged lymph nodes on the neck. She took remedies at home and think it is an ordinary sickness, but her condition gets bad. Headache and psychiatric changes, fainting forced her to come to Clinic. She felt some pain and rash in her throat, after some days confusion, she can't give correct anamnesis about her condition during illness. In her family has a conflict, that's why nobody took her to the Hospital. On cranial MRI -in the cerebellum- encephalomalacia, frontal, temporal area - non-specific hyper intensity were found. It forced us to think about rare neurological complication after EBV infection, unfortunately she took therapy at home and we have any correct information about neurological deficits, but as her said after therapy she recovery and some her conditions get better). EEG showed no epileptically signs. EBV analyses were positive. Hgb-9,8g/dl, Hct-31,1%, MCV-66,4fl, MCH21pg, RDW-CV-18%, WBC-normal, ESR-N. Other blood analyses are normal. Ultrasound-enlarged lymph nodes

**CONCLUSION:** Red flags -sore throat, positive EBV analyses, late stage and long lasting illness, lymph nodes with abnormal measures and MRI signs (for Herpes virus fronto-temporal area are characteristic), hemogram is virological in nature. As a viral nature the illness continues longer and show neurological disorders. Inflammation and headache must be in our mind in the first please. Headache and following symptoms are important for differentiation type of headache. Characteristic blood tests, MRI signs, feature of the illness are helpful for the diagnosis. In this case paid attention for the characteristic signs. Detailed anamnesis helped me to think origin of the headache. Also in some cases I saw this picture during most of infections, either mononucleosis infections.

**Key words:** headache, mononucleosis

**OP-17**

## **IS THERE A RELATIONSHIP BETWEEN HEADACHE AND PSYCHIATRIC COMORBIDITIES IN PATIENTS WITH MYASTHENIA GRAVIS? PRELIMINARY FINDINGS**

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**BACKGROUND:** The aim of this study is to evaluate characteristics of headaches in patients with myasthenia gravis (MG) and their relationship with concomitant depression/anxiety. In addition, the possible relationships between headache and psychiatric symptoms with disease severity (MGFA classification), antibody profile, and current treatments were investigated.

**METHODS:** The retrospective data of 13 MG patients with a mean age of 58.2 years were analysed. Patient demographics, disease duration, MGFA classification, and antibody status (AChR, MuSK) were recorded. The presence and severity of headaches were assessed using the International Headache Society criteria and the visual analogue scale (VAS), while their impact on quality of life was assessed using the Headache Impact Test (HIT-6). Depression and anxiety scores were determined using the Beck Depression and Anxiety Scales.

**RESULTS:** Three patients were diagnosed with episodic headache. All of these patients were female; the severity of headache was measured on the VAS between 5 and 7, and the impact on quality of life was measured with the HIT-6 test between 48 and 66. One of the headache patients suffered from severe depression and anxiety, while the other two had minimal psychiatric symptoms. The scores for depression and anxiety ranged from minimal to severe in the entire patient group. One patient with MuSK antibodies, experienced headaches despite the low severity of the disease.

**CONCLUSION:** This preliminary study shows that headache and psychiatric comorbidities are common in MG patients. Headache is more common in women, in patients with a longer duration, of disease and in patients with psychiatric symptoms. The results emphasize the importance of routine assessment of headache and psychiatric disorders in the follow-up of MG patients. Further statistical analyses in larger patient series will clarify these correlations.

**Key words:** myasthenia gravis, headache, depression, anxiety

**OP-18**

## **TRANSITION OF HEADACHE CHARACTERISTICS FROM CHILDHOOD TO ADULTHOOD: A DESCRIPTIVE ANALYSIS OF SINGLE-CENTER FOLLOW**

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**BACKGROUND:** Primary headache symptomatology may differ between childhood and adulthood. Since pediatric and adult neurology services are often organized as separate units in many centers, longitudinal data on how headache characteristics evolve during the transition from childhood to adulthood are limited. This study aimed to evaluate these changes by comparing clinical records obtained before and after this transition.

**METHODS:** We conducted a retrospective review of patients who were followed longitudinally in both the Pediatric and Adult Headache Clinics of our tertiary university hospital. Sociodemographic data, headache diagnoses, treatments, and Visual Analog Scale (VAS) scores at the initial pediatric presentation and during adult follow-up were recorded and analyzed.

**RESULTS:** Episodic migraine was the most common diagnosis, affecting 74.6% of patients. Preventive therapy was initiated in 15% of cases. The mean initial VAS score at pediatric presentation was 7.8, decreasing to 6.0 in adulthood. Headache frequency improved in 72% of patients. Additionally, 15% experienced a diagnostic change from chronic migraine to episodic migraine during follow-up.

**CONCLUSION:** While the overall headache type remained stable in most cases during the transition from childhood to adulthood, both headache severity and diagnostic classification showed notable changes. Further research is warranted to identify the factors influencing these outcomes.

**Key words:** headache transition

**OP-19**

## **DATA-DRIVEN CLASSIFICATION OF PRIMARY AND SECONDARY HEADACHES USING CLINICAL VARIABLES**

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**BACKGROUND:** Headache is one of the most common symptoms encountered in neurological and clinical practice. Accurately differentiating between primary headache disorders, such as migraine and tension- type headache, and secondary causes, including infectious aetiologies and intracranial pathologies, is paramount in the diagnostic process. The diagnostic value of clinical features, including associated neurological symptoms, pain characteristics and triggers, has been extensively investigated as a means of refining this distinction. However, the limitations of conventional diagnostic algorithms in complex or atypical cases, coupled with their reliance on subjective clinical judgement, highlight the need for automated classification models based on machine learning.

**METHODS:** In this study, the classification included only primary headache types (e.g. migraine and tension-type headache) and secondary headache types (e.g. infections and intracranial pathologies). Retrospective clinical data from 3,665 patients presenting to Mersin University were reviewed, with records containing missing observations or cases involving more than one headache type being excluded. Following these pre-processing steps, a total of 601 patients (586 primary [97.5%]; 15 secondary [2.5%]) were included in the analysis. Due to a significant imbalance in the dataset, synthetic samples were generated using the ROSE (Random Over-Sampling Examples) method and class weighting strategies were employed to balance the data and enhance the model's ability to discriminate between classes. For the classification task, XGBoost, a gradient boosting-based machine learning algorithm, was employed. Hyperparameter tuning and 10-fold cross-validation were performed to optimize the model's overall performance.

**RESULTS:** In this study, the XGBoost model achieved a balanced accuracy of 81.62% in distinguishing between primary and secondary headaches. Although the model correctly classified all primary headache cases, it failed to detect 37% of secondary headaches. The most important determining factors include total accompanying symptoms (total number of accompanying symptoms), daily pain pattern score, triggers (total triggers), and unilateral pain localization (headache side). The fact that accompanying symptoms have the highest importance in the model supports the association between secondary headaches and systemic findings in clinical practice. In contrast, variables such as gender (male/female) and pain severity contribute relatively less, indicating that these factors are less decisive in differential diagnosis.

**CONCLUSION:** The model's high accuracy in identifying primary headache disorders suggests its potential as a powerful clinical tool for reducing unnecessary investigations and referrals. However, the risk of missing secondary cases, particularly those associated with rare but serious pathologies, highlights the model's limitations. This shortcoming may be due to class imbalance within the dataset and the inadequacy of the available clinical variables.

**Key words:** headache, classification, machine learning algorithm

**OP-20**

## **DO CHRONIC INFLAMMATORY DISEASES PREDISPOSE TO IDIOPATHIC INTRACRANIAL HYPERTENSION?**

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**BACKGROUND:** Idiopathic intracranial hypertension (IIH) is an elevation of intracranial pressure, provided that the cerebrospinal fluid (CSF) is normal, excluding intracranial pathologies. Its specific symptoms are headache, papilledema, temporary loss of vision, sixth cranial nerve palsy, diplopia, and pulsatile tinnitus. As per its definition, other neurological examination findings should be within normal ranges. It is suggested that obesity, toxins, steroids, oral contraceptives, tetracyclines, hypohypervitaminosis (vitamin A), collagen tissue diseases, endocrine and hematological disorders can have a role in its etiology. Recent studies state that chronic inflammation also plays a role in pathogenesis. In this study, we aimed to investigate the relationship between IIH and inflammatory diseases.

**METHODS:** For this retrospective study, medical data of 124 IIH patients and 109 tension-type headache (TTH) patients were reviewed. During the diagnosis phase, the answers to the question "Do you have any accompanying diseases?" in the medical files filled out by the physician were examined and additional inflammatory diseases were recorded. Laboratory results of the patients were obtained from the electronic data system. Those with inflammatory diseases were re-evaluated by the specialist physician and the diagnosis was confirmed. The prevalence of inflammatory diseases in IIH patients was compared with TTH patients.

**RESULTS:** The IIH group included three patients with ankylosing spondylitis (AS), three with Behçet's disease, four with Hashimoto's thyroiditis, one with rheumatoid arthritis, one with psoriatic arthritis, and one with pemphigus. The TTH group included one with AS, one with lichen planus, and one with Hashimoto's thyroiditis. Some patients had two comorbidities. In gender analyses, 90% of the IIH group and 67% of the TTH group were female (p: 0.001). The mean age of the IIH group was 34 years and 40 years in the TTH group (p: 0.001). The BMI of the IIH group was 32 and the TTH group was 29 (p: 0.001). The rate of inflammatory disease was 11% in the IIH group and 3% in the TTH group (p: 0.039).

**CONCLUSION:** In our study, we found a statistically significantly higher rate of inflammatory disease comorbidity in IIH patients compared to the control group. These findings suggest that inflammatory activation may be a possible factor in the development of IIH. A pathophysiological mechanism explaining the association of IIH with inflammation has not yet been reported. One hypothesis has suggested that inflammatory products and cytokines may impair CSF drainage by causing fibrosis in the choroid plexus or arachnoid granulation tissue. Decreased CSF absorption due to immune-mediated damage to arachnoid villi or vascular occlusion due to hypercoagulability are other proposed pathogenesis of this condition.

**Key words:** idiopathic intracranial hypertension, inflammatory disease, comorbidity, etiology

**OP-21**

## **A CASE OF PAINFUL OPHTHALMOPLEGIC NEUROPATHY**

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**BACKGROUND:** Recurrent painful ophthalmoplegic neuropathy (RPON), formerly termed ophthalmoplegic migraine, is a rare condition characterized by unilateral headache attacks accompanied by involvement of cranial nerves, most frequently the oculomotor nerve, followed by the abducens and trochlear nerves. Oculomotor nerve involvement may present with ophthalmoparesis, ptosis, and delayed light reflex. Most patients achieve full recovery within days to weeks. Differential diagnoses include Tolosa-Hunt syndrome, cranial neuropathies, cavernous sinus lesions, demyelinating diseases, and compressive pathologies.

**CASE:** A 50-year-old male presented to an external center with diplopia and was found to have limited right eye movements. Brain CT, CT angiography, and diffusion MRI excluded cerebrovascular ischemia, hemorrhage, and aneurysm. He was referred with a preliminary diagnosis of myasthenic attack or Miller-Fisher syndrome. In our emergency department, neurological examination showed slightly diminished right light reflex, restricted inward and downward gaze, and ptosis in the right eye; left eye movements were full. Muscle strength and reflexes were normal, and there was no ataxia. Initial diagnosis was not considered. The patient was admitted with third cranial nerve neuropathy. IV methylprednisolone 80 mg was initiated. The patient had a known diagnosis of migraine with visual aura, and unilateral throbbing headache began in the 10th hour of his hospitalization. Contrast-enhanced brain MRI (1.5 Tesla) revealed increased enhancement and thickening of the right oculomotor nerve, raising suspicion for painful ophthalmoplegic neuropathy (RPON). Symptoms improved rapidly after initiating IV prednisolone, with ptosis resolving on day 1 and diplopia on day 2. At follow-up, ocular motility was normal; Migraine headaches recurred during follow-up, but ophthalmoplegia did not.

**CONCLUSION:** In patients with ophthalmoplegia, once cerebrovascular events have been excluded, RPON should be considered as a potential differential diagnosis. Although our patient experienced a single ophthalmoplegia episode, the presence of third cranial nerve enhancement on MRI supported a diagnosis of RPON. According to ICHD-3 criteria, at least two attacks are required for a definitive diagnosis; thus, this case was classified as "probable" RPON. Continued follow-up is warranted, and recurrence would confirm the diagnosis. Given the increasing use of MRI, incorporating imaging evidence of nerve involvement into diagnostic criteria—as previously proposed—may facilitate earlier recognition and classification in patients presenting with a first clinical attack.

**Key words:** Ophthalmoplegic neuropathy, ophthalmoplegia, headache

**OP-22**

## DIPOPIA AND HEADACHE : CLINICAL CORRELATIONS AND DIAGNOSTIC APPROACH

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**BACKGROUND:** Diplopia (double vision) is a common complaint in emergency department (ED) and outpatient clinics. It is defined as 'secondary diplopia' when an organic cause was found. 'Isolated diplopia' identified as the absence of additional signs and symptoms other than diplopia. Some patients exhibit associated signs and symptoms. Headache is one of these symptoms. Diplopia and headache are frequent neurological complaints that may occur independently or together, often indicating an underlying disorder. We aimed to investigate patients with diplopia who presented with headaches as an associated symptom.

**METHODS:** Over a three-year period, patients admitted to the ED and neurology outpatient clinic with diplopia; were retrospectively analyzed. All patients were examined for risk factors and investigated for etiology.

**RESULTS:** Out of the 222(Male: 138); 213 (96%) had binocular diplopia. Headache was identified as an associated symptom in 29 (Male: 12) out of 213 patients. Patients were presented with a pressure-like headache behind their eyes. Secondary diplopia was observed in 16 (55%) cases, while an organic cause was not found in 13 (45%) cases. 11(38%) cases had ocular cranial nerve palsy, most commonly the 6th cranial nerve. Ocular cranial nerve palsy was higher in secondary diplopia.

**CONCLUSION:** Headache patients with visual abnormalities like diplopia need to be evaluated to identify underlying neurologic disease and targeted investigations are essential for accurate diagnosis and timely treatment.

**Key words:** diplopia, headache, underlying neurologic disease

**OP-23**

## **BILATERAL AVASCULAR NECROSIS OF THE FEMORAL HEAD FOLLOWING SHORT-TERM HIGH-DOSE CORTICOSTEROID THERAPY FOR COVID-19: SUCCESSFUL MANAGEMENT WITH AUTOLOGOUS ADIPOSE-DERIVED MESENCHYMAL STEM CELL INJECTION UNDER FLUOROSCOPIC GUIDANCE**

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**BACKGROUND:** Avascular necrosis (AVN) of the femoral head is a progressive ischemic condition that can occur after corticosteroid therapy, even with short-term high-dose administration. Early-stage AVN management aims to preserve joint function and delay or prevent arthroplasty. This case report presents the clinical and radiological outcomes of autologous adipose-derived mesenchymal stem cell (MSC) therapy under fluoroscopic guidance in bilateral steroid-induced AVN following COVID-19 treatment.

**METHODS:** A 46-year-old female developed bilateral hip pain one year after receiving intravenous prednisolone 250 mg/day for 5 days during hospitalization for COVID-19. Preoperative MRI demonstrated bilateral femoral head AVN (Ficat-Arlet stage II), more severe on the left. Under operating room conditions, abdominal adipose tissue was harvested, processed to obtain MSC-rich graft, and combined with 6 cc adipose tissue per hip. Using fluoroscopic guidance, each hip joint was injected intra-articularly with 6 cc of MSC-rich adipose tissue. Follow-up assessments were performed at 2 weeks, 6 months, and 1 year, including clinical evaluation and MRI.

**RESULTS:** Preoperatively, the patient's Numeric Rating Scale (NRS) pain score was 10/10, with marked functional limitation. At 2 weeks, pain decreased to 6/10, with improved ambulation. At 6 months, pain reduced to 3/10 and daily activities improved. At 1 year, pain was 1-2/10, with no functional restrictions. One-year MRI showed decreased abnormal signal intensity in the right femoral head AVN area, consistent with healing. No procedure-related complications occurred.

**CONCLUSION:** Autologous adipose-derived MSC injection under fluoroscopic guidance provided significant pain relief, functional recovery, and radiological improvement in bilateral steroid-induced AVN. This minimally invasive, joint-preserving approach may be considered as an effective treatment option for early-stage AVN, particularly in patients with corticosteroid-induced etiology.

**Key words:** avascular necrosis, femoral head, mesenchymal stem cells, corticosteroids, adipose tissue, COVID-19, fluoroscopic guidance, regenerative therapy

**OP-24**

## **THE UNSOLVED MYSTERY OF TRIGEMINAL AUTONOMIC CEPHALALGIAS: IS TRANSITION BETWEEN SUBTYPES POSSIBLE?**

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**BACKGROUND:** Trigeminal autonomic cephalgias (TACs) are a rare and disabling group of primary headaches in which unilateral pain is accompanied by parasympathetic autonomic symptoms, and subcategorized mainly by their symptom timing, duration and treatment options. We present our patients with more than one TAC to underline the characteristics of the treatment approach.

**CASES:** **Case 1.** A female patient had chronic paroxysmal hemicrania (PH) since age 17, with frequent daily right-sided orbital attacks lasting 15–20 minutes, accompanied by ipsilateral autonomic symptoms and responsive to indomethacin (100 mg/d). At age 37, she developed new attacks at the same location and character, but lasting 2.5 hours once daily and unresponsive to indomethacin. These resolved within two months with verapamil and topiramate, consistent with cluster headache (CH). Over follow-up, CH recurred three times, every two years in spring, while PH attacks persisted year-round.

**Case 2.** A 24-year-old woman, with a history of right-sided CH since age 8, recurring annually for 1–1.5 months in summer, presented with a new headache type. Her CH attacks (20 minutes, twice daily) responded well to subcutaneous sumatriptan. The new attacks were shorter (7–8 minutes), more frequent (6–8/day), and in the same location, with autonomic symptoms. They were unresponsive to verapamil, ergot, lithium, and galcanezumab, but responded completely to indomethacin, consistent with PH.

**Case 3.** A 33-year-old man with left-sided CH since age 24, typically lasting 30–70 days each winter, presented with another attack. Standard CH treatment (prednisolone, verapamil, lithium, weekly nerve blocks) was initiated. On day 43, he developed a second type of headache: 8–10 nightly attacks of orbital pain, lasting 15–20 minutes, with autonomic symptoms, unresponsive to oxygen, triptans and sphenopalatine block. Suspecting transformation to PH, indomethacin (150 mg/day) was started, leading to complete resolution of headache within 10 days.

**Case 4.** A 27-year-old man presented with severe nocturnal headaches for 2 months, lasting 1–2 hours with ipsilateral autonomic symptoms, consistent with CH. Similar attacks, lasting 1.5 months had occurred two years earlier. In the past month, he also developed shorter daytime attacks (5–10 minutes, 4–5 times/day) in the same location. Ongoing treatment with dexamethasone, verapamil, topiramate and triptans or ergot was ineffective. Verapamil was increased and weekly nerve blocks administered, reducing nocturnal CH attacks. However, daytime attacks persisted. Suspecting coexisting PH, indomethacin was initiated, leading to complete resolution of the short attacks.

**DISCUSSION:** Only a few case reports have shown the coexistence of two types of TACs in the same patient. Despite some clinical overlap, their pathophysiologic mechanisms and pharmacological responses differ. The co-occurrence or transformation over time may reflect maladaptation in the pain matrix due to chronic trigeminal-autonomic activation. This complicates diagnosis and treatment, requiring individualized approaches guided by current evidence to optimize outcomes and quality of life.

**Key words:** trigeminal autonomic cephalgias, paroxysmal hemicranias, cluster headache

**OP-25**

## **APPLICATION OF CERVICAL EPIDURAL BLOOD PATCH FOR SPONTANEOUS CEREBROSPINAL FLUID LEAKS: TWO CASE REPORTS**

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**BACKGROUND:** Spontaneous cerebrospinal fluid (CSF) leaks are uncommon but significant causes of orthostatic headaches, often resulting from dural injuries. Although lumbar epidural blood patches (EBPs) are commonly used to treat such leaks, cervical EBPs are rarely performed due to the higher risk of complications, including spinal cord injury. This case series presents two patients with spontaneous cervical CSF leaks successfully treated with cervical EBPs under fluoroscopic guidance.

### **CASES**

**Case 1:** The first case involved a 24-year-old female who presented with a two-month history of severe orthostatic headaches and tinnitus, and a Visual Analog Scale (VAS) score of 8. After a cervical EBP with 3 mL of autologous blood, her symptoms resolved.

**Case 2.** The second case describes a 55-year-old male with a history of radiotherapy who presented with a VAS score of 10 and a four-month history of orthostatic headaches. A cervical EBP using 6 mL of blood led to complete symptom resolution.

**CONCLUSION:** Both cases highlight the efficacy and safety of cervical EBPs when performed with careful imaging guidance and minimal blood volumes. These findings contribute to the limited literature on cervical EBPs and suggest that this approach should be considered in cases of refractory cervical CSF leaks.

**Key words:** cervical epidural blood patch, fluoroscopic guidance, spontaneous intracranial hypotension

**OP-26**

## **IDIOPATHIC INTRACRANIAL HYPERTENSION WITHOUT PAPILLEDEMA (IIHWOP)**

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**BACKGROUND:** Idiopathic Intracranial Hypertension Without Papilledema (IIHWOP) is a rare and underdiagnosed condition characterized by elevated intracranial pressure without optic disc swelling. It commonly presents with chronic refractory headaches that mimic primary headache disorders, especially migraine. The absence of papilledema might often lead to diagnostic delay. Recent literature emphasizes the role of neuroimaging findings -such as empty sella and transverse sinus stenosis- and cerebrospinal fluid (CSF) opening pressure in identifying IIHWOP. This case series highlights the clinical and radiological features of four patients diagnosed with IIHWOP to raise awareness of this diagnostic entity.

**CASES:** Between February and July 2025, patients hospitalized with chronic headaches in our neurology ward were prospectively evaluated. IIHWOP diagnosis was based on Friedman criteria. Demographic data, comorbidities, neuroimaging (MRI/MRV), ophthalmologic findings (fundus, OCT, visual field), CSF opening pressure and protein levels, and treatment responses were systematically recorded. Four patients (mean age: 46.5 years; three females) were identified with chronic daily headache unresponsive to standard therapies. Two patients had additional comorbidities, including hypertension, asthma, and hypothyroidism. None of the patients exhibited papilledema on fundus examination or optic nerve edema on OCT. Visual field testing revealed peripheral constriction in 2 patients, while findings were unremarkable in the others. MRI revealed optic nerve tortuosity in 2 patients, distension of the optic nerve sheath in 2 patients, and flattening of the optic disc in 1 patient; some patients exhibited more than one neuroimaging abnormality. All patients received acetazolamide as initial therapy; 2 required adjunctive topiramate, and 1 received corticosteroids with subsequent referral for shunt surgery due to persistent symptoms. Three patients demonstrated a marked reduction in headache frequency and severity with medical management and remained stable during follow-up without visual deterioration. One patient did not respond to pharmacological therapy and was referred to neurosurgical intervention. No new neurological deficits, papilledema, or severe complications were observed in any patient during the observation period.

**CONCLUSION:** This case series highlights that the absence of papilledema should not exclude the diagnosis of idiopathic intracranial hypertension in patients presenting with chronic refractory headaches. Neuroimaging findings and CSF analysis are essential for timely diagnosis. Increased clinical awareness of IIHWOP may prevent misdiagnosis, enable earlier intervention, and reduce unnecessary morbidity in this challenging patient group.

**Key words:** Idiopathic Intracranial Hypertension Without Papilledema

**OP-27**

## **MECHANICAL PROPERTIES OF THE TRAPEZIUS MUSCLE IN CHRONIC NECK PAIN: A COMPARATIVE STUDY WITH AGE-MATCHED ASYMPTOMATIC CONTROLS**

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**BACKGROUND:** Chronic neck pain (CNP) affects nearly 30% of the global population, often involving the trapezius muscle, causing stiffness, tenderness, and reduced mobility. CNP is a leading cause of work-related disability. While changes in pain thresholds are studied, the mechanical alterations of the trapezius in CNP remain unclear. This study aimed to investigate both pain sensitivity and the viscoelastic properties of the trapezius muscle in individuals with chronic neck pain, and to compare the findings with those of an age-matched healthy control group.

**METHODS:** The study included 46 participants, aged 20 to 33, with 23 individuals diagnosed with chronic neck pain (CNP) (mean age:  $23.61 \pm 2.61$  years; BMI:  $22.09 \pm 3.20$ ) and 23 healthy controls (mean age:  $23.70 \pm 2.72$  years; BMI:  $23.82 \pm 3.55$ ). All individuals in the CNP group had neck pain for over three months and a Neck Disability Index (NDI) score above 20%. Pain thresholds and mechanical properties of the trapezius muscle were assessed on the dominant side, where all CNP participants experienced pain. Measurements were taken at the midpoint of the upper, middle, and lower trapezius using two devices. The algometer was used to measure pressure pain threshold, while the myotonometer assessed muscle tension, stiffness, decrement, relaxation time, and creep.

**RESULTS:** The NDI scores in the CNP group were significantly higher ( $26.88 \pm 8.86$ ) compared to the control group ( $10.57 \pm 4.64$ ). Pain thresholds in the upper trapezius were significantly lower in the CNP group ( $p=0.036$ ), as well as in the middle trapezius ( $p<0.0001$ ) and the lower trapezius ( $p=0.009$ ). The CNP group also showed a significant difference in creep in the upper trapezius ( $p=0.047$ ) and middle trapezius ( $p=0.003$ ). Additionally, the middle trapezius exhibited increased tension ( $p=0.018$ ), stiffness ( $p=0.008$ ), and reduced relaxation time ( $p=0.004$ ). There were no significant differences found in decrement or in any mechanical properties of the lower trapezius ( $p>0.05$ ).

**CONCLUSION:** Individuals with chronic neck pain show lower pain thresholds and marked changes in the viscoelastic properties of the trapezius muscle, particularly in the upper and middle regions. Reduced creep suggests limited muscle elasticity and higher injury risk. Compared to healthy controls, these individuals exhibit greater muscle stiffness and tension, especially in the middle trapezius, indicating decreased compliance and altered mechanical function. Elevated muscle tension may reflect deeper cellular-level changes. Additionally, shorter relaxation times observed in this group imply that stiffer muscles recover more rapidly, which might reduce the effectiveness of therapeutic interventions targeting muscle flexibility and endurance.

**Key words:** pain, physical therapy, physiotherapy, physical activity, rehabilitation

**OP-28**

## **COMPARATIVE ANALYSIS OF CORTICAL INTERACTIONS IN HEADACHE SYNDROMES: AN EEG STUDY BASED ON PHASE-AMPLITUDE COUPLING**

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**INTRODUCTION:** Headache is one of the most common neurological complaints worldwide that negatively affects individuals' quality of life and is often associated with loss of workforce, decreased life satisfaction, and an increased burden on healthcare systems. In particular, migraine and tension-type headache (TTH) are among the most prevalent primary headache disorders in the general population. Due to differences in their symptom profiles and underlying etiologies, these two syndromes must be carefully differentiated in clinical practice [1, 2].

Migraine is commonly defined as a neurovascular disorder characterized by unilateral, throbbing pain that worsens with physical activity, whereas tension-type headache (TTH) is typically bilateral, pressing in nature, and of mild to moderate intensity. However, there are significant clinical overlaps between the two headache types, and some studies suggest that these conditions may represent different ends of a shared spectrum [2, 3]. Clinical findings in both headache types also indicate alterations in attention, sensory processing, and cognitive control mechanisms [4-6].

Electroencephalography (EEG) is a valuable tool for understanding the neural underpinnings of headache disorders. EEG analyses have shown that migraine patients exhibit increased cortical excitability, disrupted neuronal synchronization, and alterations in power spectra across certain frequency bands during both interictal and ictal phases [5, 7, 8]. Similarly, in TTH patients, abnormal electrical activity and altered reflex responses related to attention and perceptual processing have been reported [2, 9, 10]. In recent years, it has become evident that EEG signals can be evaluated not only based on power and frequency characteristics but also through the interactions between different oscillatory bands. In this context, phase-amplitude coupling (PAC) enables the measurement of the relationship between the phase of slower rhythms and the amplitude of faster rhythms, thereby allowing for the assessment of coordination and information transfer across distinct brain regions [11, 12]. Such cross-frequency interactions are particularly critical for higher-order cognitive functions such as attention, perception, and motor control [12].

One of the most used methods for measuring PAC is the Modulation Index (MI) analysis developed by Tort et al. [3]. This method has high sensitivity in detecting irregularities in brain oscillations associated with neurological disorders. However, the variation in MI levels in headache disorders has not been sufficiently investigated, and further evidence is needed in this area.

The aim of this study is to perform a comparative analysis of phase-amplitude coupling in EEG signals of individuals with migraine, tension-type headache (TTH), and healthy controls using the Modulation Index calculated via the Tort method. In doing so, the study seeks to identify neurophysiological patterns specific to different headache types and support clinical diagnostic categories with objective biomarkers. The findings are expected to contribute both to a better understanding of disease mechanisms and to the development of EEG-based diagnostic approaches.

It should be emphasized that the current results represent preliminary findings, and the study is still ongoing. Therefore, interpretations should be made cautiously until further data and validation are obtained.

**METHODS:** In our study, a comparative evaluation was conducted on the demographic data, pre-treatment monthly attack frequency, pain severity, and EEG signal analyses of patients who presented to the Neurology Outpatient Clinic of Adana City Training and Research Hospital between March 15, 2025, and July 10, 2025, and were diagnosed with either migraine or tension-type headache (TTH) according to the International Classification of Headache Disorders (ICHD-3 beta) criteria and who had given informed consent to participate in the study.

**Exclusion criteria included:** the presence of chronic systemic diseases (e.g., hypertension, diabetes, chronic liver, kidney, or lung disease, hematological disorders), acute or chronic infectious conditions, intracranial space-occupying lesions, malignancies, current use of anticoagulant or anti-inflammatory medications, systemic corticosteroid use, systemic inflammatory disorders, ongoing prophylactic headache treatment, use of antiepileptic or antidepressant drugs, excessive use of simple analgesics, pregnancy, lactation, and clinical depression.

In this study, resting-state electroencephalography (EEG) recordings were obtained from participants using a 32-channel Cadwell device. Following data collection, preprocessing steps were applied to the EEG signals to eliminate noise and artifacts, including band-pass filtering. The cleaned EEG signals were then decomposed into five standard frequency bands: Delta, Theta, Alpha, Beta, and Gamma. For each frequency band, the Modulation Index (MI), a widely used method for assessing phase-amplitude coupling (PAC), was calculated using the Tort method. Subsequently, the signals were segmented into five cortical regions—frontal, temporal, central, parietal, and occipital—for topographic analysis. In the final stage, a comparative analysis of MI values was performed across the different groups (i.e., migraine, tension-type headache, and healthy controls). The methodological procedure is schematically summarized in Figure 1.

## 1. Participants

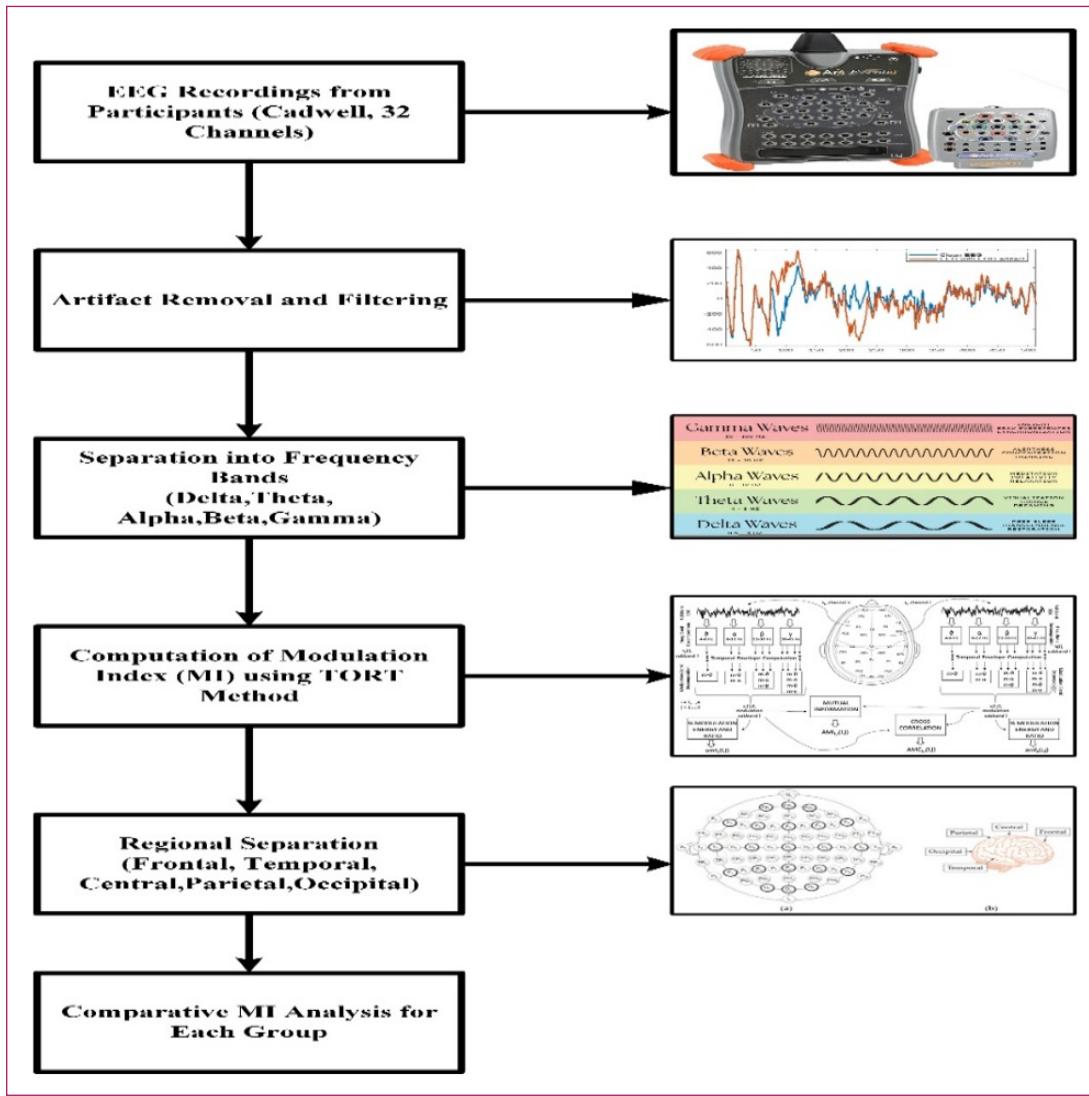
**In this study, a total of 29 individuals were included. Participants were divided into three groups:** individuals diagnosed with tension-type headache (TTH) ( $n = 12$ ), individuals diagnosed with migraine ( $n = 11$ ), and healthy controls ( $n = 6$ ). The mean age and standard deviation for each group were as follows: TTH group  $25.25 \pm 7.67$  years; migraine group  $28.55 \pm 11.03$  years; control group  $29.83 \pm 9.63$  years. The overall mean age across all participants was  $27.45 \pm 9.66$  years. Of the 29 participants, 23 were female and 6 were male. Informed consent was obtained from all participants prior to inclusion in the study. The research was conducted in accordance with the principles of the Declaration of Helsinki and received approval from the local ethics committee.

## 2. EEG Recordings

Electroencephalography (EEG) recordings were obtained using a 32-channel Cadwell Arc EEG system. Electrode placement was performed according to the international 10-20 system. Recordings were conducted while participants were in a resting state with their eyes closed, in a quiet and dimly lit environment. The sampling rate was set at 512 Hz, and electrode impedances were maintained below  $5 \text{ k}\Omega$ .

## 3. Signal Preprocessing

Raw EEG signals were processed using MATLAB 2023b software. First, a notch filter was applied to eliminate 50 Hz power line noise, followed by a band-pass filter in the range of 0.5–100 Hz.



**Figure 1.** Processing Workflow of EEG Data Obtained from Participants for Modulation Index Analysis

Segments containing artifacts were excluded from the analysis based on visual inspection. These preprocessing steps ensured that the signals were suitable for further analysis.

#### 4. Frequency Band Decomposition

The cleaned EEG signals were decomposed into the following five standard frequency bands;

- **Delta:** 0.5-4 Hz
- Theta: 4-8 Hz
- Alpha: 8-13 Hz
- Beta: 13-30 Hz
- Gamma: 30-45 Hz

Each frequency band was analyzed independently.

#### 5. Modulation Index (MI) Calculation - Tort Method

To measure phase-amplitude coupling (PAC) between frequency bands, the Modulation Index (MI) was calculated using the method developed by Tort et al. (2010) [13]. This method quantitatively determines the relative interaction between the phase of a low-frequency signal and the amplitude envelope of a high-frequency signal.

The following steps were applied for each EEG channel:

1. Band-pass filtering in the relevant low and high frequency bands,
2. Extraction of instantaneous phase and amplitude values using the Hilbert transform,
3. Construction of amplitude distribution histograms as a function of phase,
4. Entropy-based computation of the difference between the observed distribution and a theoretical uniform distribution to obtain the MI value.

MI values were calculated separately for each channel and each frequency band

## **6. Regional Analysis**

MI values were evaluated across five anatomical regions based on electrode placement:

- Frontal • Temporal • Central • Parietal • Occipital

For each region, the mean MI value corresponding to each frequency band was calculated. This allowed for a comparative investigation of how PAC characteristics varied across different cortical regions.

## **7. Statistical Analysis**

The obtained data were evaluated statistically. For each group, the arithmetic mean and standard deviation of the MI values corresponding to each region and frequency band combination were calculated. Advanced statistical tests were excluded from the scope of this study; only descriptive statistics were employed at this stage. All computations and analyses were performed using MATLAB 2023b software.

**RESULTS:** In this study, Modulation Index (MI) values calculated using the Tort method were analyzed based on resting-state electroencephalography (EEG) recordings from 12 patients with chronic tension-type headache (TTH), 11 patients with migraine, and 6 healthy control individuals. MI values were computed across five main EEG frequency bands (Delta, Theta, Alpha, Beta, Gamma) and five cortical regions (Frontal, Temporal, Central, Parietal, Occipital). The findings are presented separately for each group as well as in a comparative format.

### **1. Tension-Type Headache (TTH) Group (n = 12)**

In the TTH group, delta band modulation was particularly elevated in the temporal ( $5.65\text{E-}06 \pm 1.32\text{E-}06$ ) and parietal ( $4.47\text{E-}06 \pm 1.33\text{E-}06$ ) regions. The central region exhibited the lowest MI value in this band ( $3.82\text{E-}06 \pm 1.40\text{E-}06$ ). In the theta band, the highest MI value was observed in the central region ( $5.17\text{E-}05 \pm 1.51\text{E-}05$ ), which may indicate the role of the sensorimotor cortex in the pathophysiology of TTH.

Alpha band modulations were relatively similar across all regions, although a slight increase was noted in the temporal cortex ( $6.25\text{E-}06 \pm 2.18\text{E-}06$ ). The beta band showed a markedly high MI value particularly in the parietal region ( $5.53\text{E-}03 \pm 1.40\text{E-}03$ ), suggesting that cognitive-motor interactions may be enhanced in this patient group. The lowest beta MI value was found in the occipital cortex.

In the gamma band, a prominent increase in modulation was observed in the frontal ( $0.051 \pm 0.014$ ) and temporal ( $0.052 \pm 0.013$ ) regions. This may reflect compensatory cortical mechanisms associated

with executive functions and cognitive control processes in TTH patients. Detailed MI values are presented in Table 1.

**Table 1.** EEG Modulation Index (MI) Values in the Tension-Type Headache (TTH) Group (n = 12)

Band	Frontal	Temporal	Central	Parietal	Occipital
Delta	4.83E-06 ± 1.43E-06	5.65E-06 ± 1.32E-06	3.82E-06 ± 1.40E-06	4.47E-06 ± 1.33E-06	4.52E-06 ± 1.78E-06
Theta	4.74E-05 ± 1.20E-05	4.96E-05 ± 1.47E-05	5.17E-05 ± 1.51E-05	4.17E-05 ± 1.34E-05	4.03E-05 ± 1.22E-05
Alpha	5.68E-06 ± 1.70E-06	6.25E-06 ± 2.18E-06	6.06E-06 ± 1.87E-06	5.42E-06 ± 2.10E-06	5.26E-06 ± 2.04E-06
Beta	4.94E-03 ± 1.18E-03	4.81E-03 ± 1.27E-03	4.73E-03 ± 1.31E-03	5.53E-03 ± 1.40E-03	3.84E-03 ± 1.07E-03
Gamma	0.051 ± 0.014	0.052 ± 0.013	0.047 ± 0.011	0.045 ± 0.010	0.050 ± 0.013

## 2. Healthy Control Group (n = 6)

In healthy individuals, delta band modulation was most prominent in the parietal (1.65E-05 ± 1.88E-05) and occipital (5.01E-06 ± 3.10E-06) regions, indicating that slow-wave activity is more pronounced in posterior cortical areas. Theta band MI values were generally low, although slight increases were observed in the temporal (9.32E-07 ± 8.91E-07) and parietal (2.91E-06 ± 2.51E-06) regions.

MI values in the alpha and beta bands were low and stable, which may reflect healthy cortical synchronization during the resting state. In contrast, gamma band modulation was notably elevated in the parietal (3.71E-05 ± 2.65E-05), temporal (1.86E-05 ± 1.65E-05), and frontal (1.52E-05 ± 1.43E-05) regions. These values are presented in Table 2.

**Table 2.** EEG Modulation Index (MI) Values in Healthy Controls (n = 6)

Band	Frontal	Temporal	Central	Parietal	Occipital
Delta	2.06E-06 ± 2.10E-06	1.58E-06 ± 1.35E-06	2.60E-06 ± 1.00E-08	1.65E-05 ± 1.88E-05	5.01E-06 ± 3.10E-06
Theta	1.15E-07 ± 6.42E-08	9.32E-07 ± 8.91E-07	5.94E-08 ± 2.71E-08	2.91E-06 ± 2.51E-06	7.90E-07 ± 3.00E-07
Alpha	3.65E-08 ± 1.40E-08	4.39E-07 ± 4.50E-07	5.63E-08 ± 4.87E-08	3.79E-07 ± 2.61E-07	4.70E-07 ± 3.05E-07
Beta	6.92E-08 ± 4.73E-08	3.56E-07 ± 2.92E-07	1.46E-07 ± 1.39E-07	4.15E-07 ± 1.79E-07	4.23E-07 ± 2.04E-07
Gamma	1.52E-05 ± 1.43E-05	1.86E-05 ± 1.65E-05	1.52E-05 ± 8.76E-06	3.71E-05 ± 2.65E-05	1.87E-05 ± 3.00E-06

## 3. Migraine Group (n = 11)

In the migraine group, delta band modulation was strikingly elevated in the frontal region (3.03E+07 ± 7.42E+07), which may be associated with impaired frontal cortical inhibition. The central delta MI value (3.68E-03 ± 5.18E-03) was also notable.

In the theta band, a high MI value was observed in the frontal cortex (6.39E+06 ± 1.74E+07), supporting the possibility of thalamocortical dysrhythmia. The central region (5.57E-03 ± 6.99E-03) also showed increased theta modulation. Alpha band modulation was most prominent in the frontal region (1.02E+06 ± 2.83E+06).

For the beta band, the highest MI value was found in the central cortex (5.58E-03 ± 6.88E-03), which

may be related to enhanced sensorimotor activity. In the gamma band, an extraordinarily high MI value in the frontal region ( $5.13E+08 \pm 1.43E+09$ ) was interpreted as a sign of increased cortical excitability. Detailed MI values, along with other regional data, are presented in Table 3.

**Table 3.** EEG Modulation Index (MI) Values in the Migraine Group (n = 11)

Band	Frontal	Temporal	Central	Parietal	Occipital
Delta	$3.03E+07 \pm 7.42E+07$	$7.22E-06 \pm 9.14E-06$	$3.68E-03 \pm 5.18E-03$	$1.28E-05 \pm 9.22E-06$	$1.56E-05 \pm 1.26E-05$
Theta	$6.39E+06 \pm 1.74E+07$	$1.24E-06 \pm 1.25E-06$	$5.57E-03 \pm 6.99E-03$	$8.58E-07 \pm 3.56E-07$	$1.50E-06 \pm 4.65E-07$
Alpha	$1.02E+06 \pm 2.83E+06$	$3.43E-07 \pm 3.28E-07$	$5.52E-03 \pm 6.96E-03$	$5.89E-07 \pm 3.59E-07$	$1.08E-06 \pm 6.35E-07$
Beta	$2.26E+06 \pm 6.30E+06$	$1.25E-06 \pm 1.26E-06$	$5.58E-03 \pm 6.88E-03$	$1.09E-06 \pm 6.11E-07$	$1.32E-06 \pm 6.27E-07$
Gamma	$5.13E+08 \pm 1.43E+09$	$2.49E-05 \pm 2.38E-05$	$5.52E-03 \pm 6.74E-03$	$3.78E-05 \pm 1.68E-05$	$2.42E-05 \pm 1.28E-05$

**DISCUSSION:** In this study, phase-amplitude coupling (PAC) was evaluated based on resting-state EEG recordings among individuals with migraine, tension-type headache (TTH), and healthy controls. Analyses performed using the Modulation Index (MI) method developed by Tort et al. revealed remarkably elevated MI values in the delta and gamma bands within the frontal regions of the migraine group. These findings suggest that migraine may be characterized by increased cortical excitability and impaired inhibition even during the interictal period [3, 5, 7]. Similarly, increases in the theta band were consistent with the possibility of thalamocortical dysrhythmia [4, 8].

In the TTH group, prominent delta modulation in the temporal and parietal regions, along with elevated beta MI values in the parietal cortex, suggests the presence of compensatory mechanisms related to perception and motor control. Gamma band MI increases in the frontal-temporal regions may reflect cortical adaptations aimed at regulating executive functions in TTH patients [2, 6, 8].

The findings highlighted significant increases in delta and gamma MI values in the frontal regions of the migraine group, and in the TTH group, significant differences were observed in the delta and beta bands in the parietal and temporal regions. These results indicate that cross-frequency interactions could serve as neurophysiological biomarkers to assist in the differential diagnosis of headache disorders.

Although the results of the present study are largely consistent with prior literature, they offer more detailed and region-specific analyses. For example, Khan et al. (2024) [14] emphasized increased delta activity in the frontal EEG recordings of migraine patients during the interictal period, attributing this to disrupted cortical inhibition. Similarly, our study observed elevated delta MI in the frontal cortex, and additionally reported a striking increase in the gamma band. This gamma modulation may reflect heightened cortical excitability. In this respect, our study extends the findings of Khan et al. by providing new perspectives based on both regional and frequency-specific analysis, thereby making a meaningful contribution to the literature.

Altintop et al. (2017) [15] identified differences in EEG waveforms and power spectra between migraine and TTH patients but did not include advanced methods such as PAC analysis. In contrast, our study utilized a PAC-based approach, moving beyond traditional spectral analysis and more sensitively revealing the neural coordination patterns of different headache types. Moreover, regional comparisons were not only made at the individual channel level but also across five distinct cortical regions, enabling topographical mapping of neurophysiological differences. This methodological

approach provides a more comprehensive and in-depth analytical perspective than that of Altintop et al.

The observed delta modulation in the temporal and parietal regions and increased beta MI values in the parietal cortex of the TTH group suggest compensatory reorganization of cortical mechanisms related to pain perception and motor planning. The increased gamma modulation in the frontal-temporal regions may also reflect compensatory activity in executive functions. This implies that TTH is not merely a passive pain syndrome, but also involves neurophysiological restructuring of certain cognitive processes.

In healthy individuals, the overall low and stable MI values indicate preserved cortical synchronization at rest, while regional increases in gamma modulation highlight that high-frequency coordination is also necessary for physiological cognitive processes such as attention and alertness. These findings underscore the role of gamma band activity not only in pathological states but also in normal cognitive control mechanisms.

Limitations of the study include a relatively small sample size and the use of descriptive rather than advanced statistical comparisons. Additionally, correlations between clinical variables (e.g., attack frequency, disease duration) and neurophysiological markers were not addressed within the scope of this study. Nevertheless, the regional PAC analyses conducted using the Tort method offer valuable methodological contributions to the field.

**CONCLUSION:** This study presented a comparative analysis of phase-amplitude coupling (PAC) in EEG signals across individuals with migraine, tension-type headache (TTH), and healthy controls, revealing neurophysiological differences specific to each headache disorder. Modulation Index (MI) values calculated using the Tort method showed marked increases particularly in the frontal regions of migraine patients, indicating disruptions in cortical excitability. In the TTH group, beta and gamma band modulations in sensorimotor regions were notable, suggesting the involvement of distinct compensatory mechanisms within cognitive-motor networks.

The low and balanced MI levels observed in healthy individuals reflect the regularity of brain activity during the resting state. These findings suggest that EEG-based PAC analysis may provide potential neurophysiological biomarkers for the differential diagnosis of headache syndromes. This approach may contribute to the development of non-invasive methods that enhance diagnostic accuracy in the future. However, to generalize the findings, further studies with larger sample sizes and clinical correlation analyses are required.

**Key words:** migraine, tension-type headache, headache disorders, electroencephalography, phase-amplitude coupling, modulation index, cortical excitability, functional connectivity, resting-state EEG, biomarkers

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**ORAL PRESENTATIONS**

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**OP-29**

## **RARE AND OVERLOOKED BUT CRITICAL: INDOMETHACIN-RESPONSIVE HEADACHE AND LONG-LASTING AUTONOMIC SYMPTOMS**

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**BACKGROUND:** Hemicrania with long-lasting autonomic symptoms (LASH) is a rare, indomethacin-responsive headache disorder considered within the spectrum of trigeminal autonomic cephalgias (TACs), although it is not currently recognized in the International Classification of Headache Disorders, 3rd edition (ICHD-3). It is characterized by unilateral headache accompanied by cranial autonomic symptoms (CAS) that both precede and persist beyond the headache phase.

**CASE:** Male patient with recurrent unilateral headache and autonomic symptoms underwent neurological and ophthalmological evaluation, brain MRI, and vascular imaging. Written informed consent was obtained from the patient for diagnostic procedures and publication of clinical findings. A 32-year-old male presented with a two-year history of monthly headache attacks lasting 30-40 minutes, accompanied by right-sided eyelid edema, ptosis, and ocular pruritus. Notably, the CAS persisted for 1-2 days after headache resolution. Neurological and ophthalmological examinations were unremarkable between attacks. Brain MRI and vascular imaging were normal. Treatment with indomethacin resulted in complete symptom remission. Symptoms recurred upon discontinuation and again resolved with re-initiation.

**CONCLUSION:** The temporal dissociation between headache and CAS, combined with responsiveness to indomethacin, supports a diagnosis of LASH. The clinical features did not fulfill criteria for other TACs, highlighting the need for broader awareness of this rare entity. Including LASH in future ICHD revisions could facilitate timely diagnosis and effective treatment in similar cases.

**Key words:** headache, autonomic symptoms, indomethacin