

Steroid-Responsive Late Symptomatic Perihematomal Edema In Intracerebral Hemorrhage

Bahar AKSAY KOYUNCU¹, Selen GÜR ÖZMEN², Sedef TAVUKÇU ÖZKAN³, Ebru ALTINDAĞ⁴, Reha TOLUN⁵, Yakup KRESPI¹

¹Unit of Stroke Rehabilitation and Research, Memorial Şişli Hospital, İstanbul, Turkey

²Clinic of Neurology, Iğdır State Hospital, Iğdır, Turkey

³Unit of Intensive Care, Memorial Hizmet Hospital, İstanbul, Turkey

⁴Clinic of Neurology, Florence Nightingale Hospital, İstanbul, Turkey

⁵Unit of Acute Stroke, Memorial Hizmet Hospital, İstanbul, Turkey

Dear Editor,

Intracerebral hemorrhage (ICH) is the most devastating subtype of stroke (1). One of the causes of late neurological deterioration (LND) in ICH is thought to be perihematomal edema (PHE), which arises early during the course of an acute ICH mostly within the first week after onset (2,3,4,5,6). Herein we report three patients of acute ICH who developed very late neurological deterioration associated with an increasingly late PHE without hematoma enlargement shown on control cranial CT taken approximately two weeks after ICH. The patients showed remarkable clinical recovery with steroid treatment. A 55-year-old man presented with aphasia and right hemiplegia. Baseline cranial CT revealed a left putaminal hematoma (28 cc) (Figure 1a). Sixteen days later, a progressive decline of consciousness was detected. The Glasgow Coma Score (GCS) declined from 15 to 13. The only possible explanation was a large PHE that formed around a spontaneously resorbing hematoma shown on repeated cranial CT (Figure 1b, c). Initiating steroid therapy resulted in a remarkable clinical improvement with a progressive decrease in PHE and its mass effect in the control cranial CT (Figure 1d). A 76-year-old man presented with right hemiplegia and aphasia. Admission cranial CT showed a left putaminal hematoma (18 cc) (Figure 2a). Eighteen days later, a slow and progressive decline of consciousness and progression of hemiparesis were detected. GCS declined from 15 to 12. The sole abnormality was a large PHE despite reduction in the hematoma volume shown on repeat cranial CT (Figure 2b-d). After initiating steroid treatment, a dramatic clinical recovery was noted. A 70-year-old man presented with a left focal motor seizure followed by secondary generalized tonic-clonic seizure. His past medical history noted a left intracerebral hematoma probably due to amyloid angiopathy. An acute right frontal lobar hematoma (15 cc) was detected in his admission cranial CT (Figure 3a). He became somnolent 24 h after clinical onset, and this late neurological deterioration led to hematoma evacuation (Figure 3b). His consciousness improved. On the 12th day after the onset, a second progressive decline of consciousness was observed, and this was associated with an increased PHE shown on repeated cranial CT (Figure 3c, d). In the absence of another likely explanation of LND, steroid treatment was initiated, which led to a remarkable clinical recovery. The significant feature of our patients is that LND and PHE arose in the "very late phase" of acute ICH. Another notable feature is that a symptomatic late PHE appeared around a surgically evacuated ICH within the first 24 h of onset as shown in our third case, which was unexpected. We started intravenous dexamethasone 16 mg daily and decreased the dose by 4 mg every three days. With regard to clinical improvement, the result was dramatic and was detected soon after the initiation of steroid treatment, reminding us of the steroid treatment effect on peritumoral brain edema cases. There are studies showing evidence that PHE of ICH may have a vasogenic nature during the acute or subacute phase of hematoma, but pathophysiological explanations are speculative (3,7). To our knowledge, this is the first study that reports steroid responsive symptomatic late PHE in cases of ICH.

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Correspondence Address: Dr. Selen Gür Özmen, İstanbul Üniversitesi, Deneysel Tıp ve Araştırma Enstitüsü (DETAE), Sinirbilim Anabilim Dalı, İstanbul, Türkiye E-mail: drselenozmen@gmail.com

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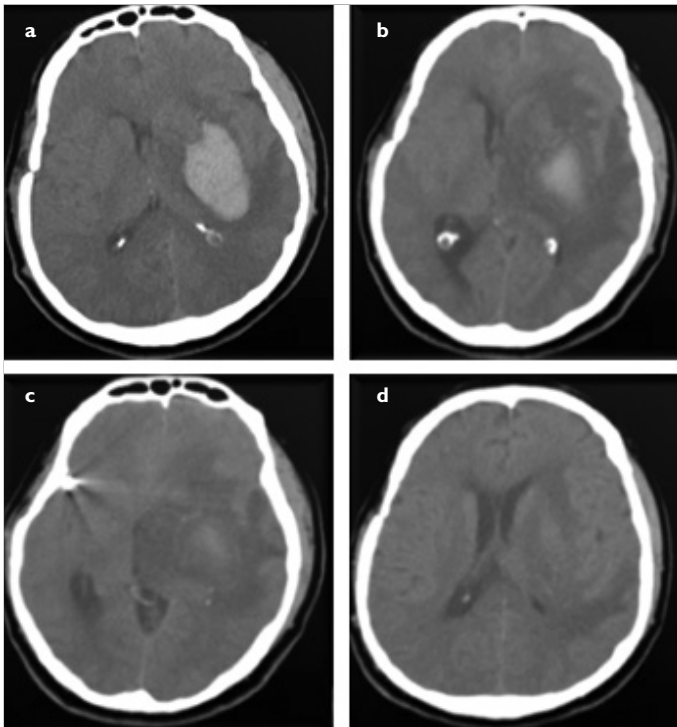


Figure 1. a-d. (a) Baseline cranial CT of case-1 revealing a left putaminal hematoma. (b, c) Cranial CT taken on the 16th day of admission after decline in consciousness showing a large PHE despite reduction in hematoma volume. (d) Cranial CT taken after starting steroid therapy showing decrease in PHE

CT: computed tomography; PHE: perihematomal edema

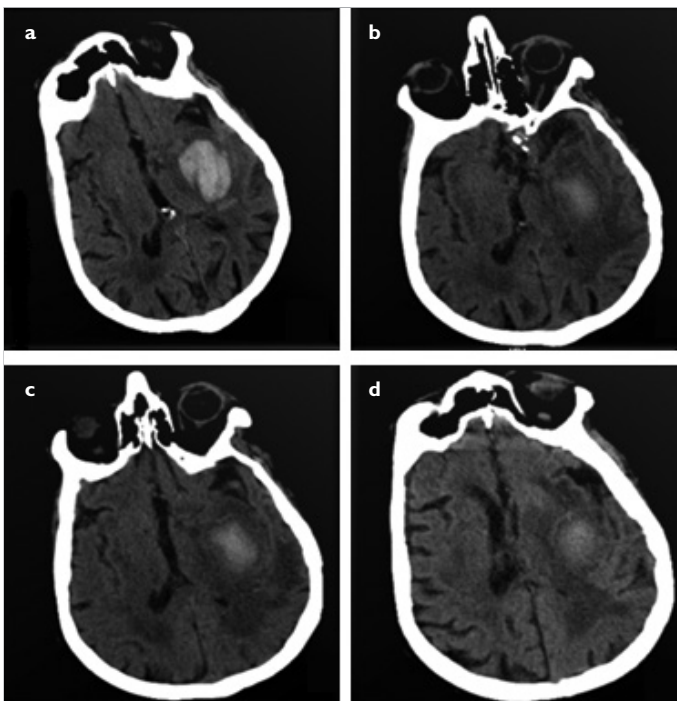


Figure 2. a-d. (a) Admission cranial CT of case-2 showing a left putaminal hematoma. (b-d) Cranial CT taken on the 20th day after clinical deterioration revealing a large PHE despite spontaneous reduction in the hematoma volume

CT: computed tomography; PHE: perihematomal edema

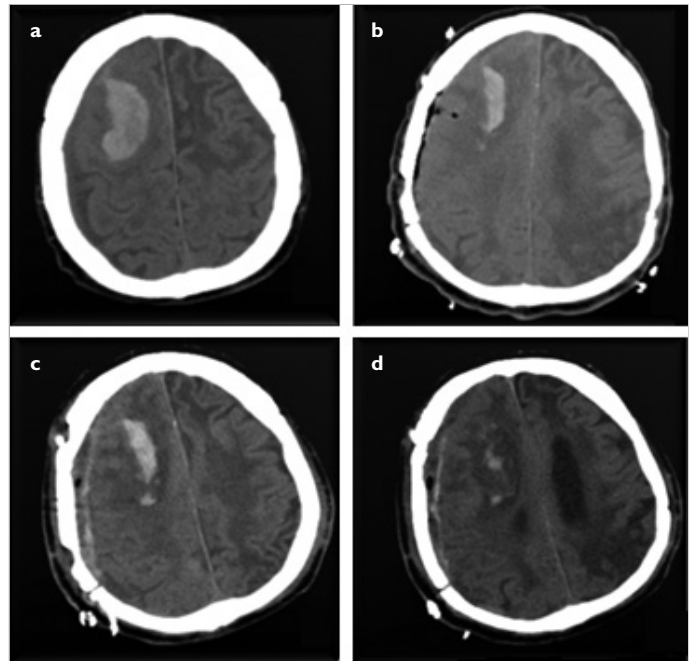


Figure 3. a-d. (a) Baseline cranial CT of case-3 depicting frontal hematoma. (b) Cranial CT taken 24 h after surgery. (c, d) Cranial CT taken on the 12th day after the decline of consciousness showing PHE, while there was no increase in hematoma

CT: computed tomography; PHE: perihematomal edema

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