



Spontaneous Resolution of Bilateral Chronic Subdural Hematoma

Bilateral Kronik Subdural Hematomun Spontan Rezolüsyonu

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CASE ILLUSTRATION

A sixty five years old male had presented about two years back with progressive weakness of left upper and lower limb and altered mentation. On examination, as per the hospital records, he was in altered sensorium and had left hemiparesis grade 3/5. Computed tomography (CT) of the brain revealed a hypodense collection in the right frontotemporoparietal region with areas of hyperdensity posteriorly in the parietal region. This was suggestive of a predominant chronic subdural hematoma with an acute component in the parietal region (Figure 1). The hematoma was 1.5 cm in thickness and was causing significant mass effect resulting in compression of the ipsilateral ventricle. Midline shift of 8 mm was noted to the left side (Figure 1). Another hypodense collection was noted in left frontoparietal region with specks of hyperdensity in the left frontal region (Figure 1). Patient was advised surgical evacuation of bilateral subdural hematoma in order to reduce the mass effect and reverse the neurological deficit. However the patient's relatives were not willing to undergo surgery and he was discharged on best possible medications. He was eventually lost to follow up. Two years later he presented to the emergency department with history of fall following an episode of giddiness. On examination he was conscious, oriented in time, place and person. His left hemiparesis had improved and he was ambulant without support. Brain CT revealed complete resolution of the left frontoparietal subdural hematoma (Figure 2). A thin rim of right subdural collection was noted with no evidence of midline shift or mass effect (Figure 2). He was symptomatically treated and discharged.

Chronic subdural hematoma occurs in older patients following trivial trauma (1). Predisposing factors for occurrence of subdural hematoma include alcohol abuse, epilepsy and intake of anticoagulants (1). Chronic subdural hematoma presents with progressive decline in cognition, altered mentation and neurological deficit. Surgical intervention to evacuate the hematoma is necessary in presence of neurological deficit or if imaging reveals significant mass effect and midline shift due to the hematoma. Spontaneous resolution of chronic subdural hematoma is an uncommon event. It is rarer if

the subdural hematoma is bilateral. Predicting which cases are likely to undergo spontaneous resolution may avoid unnecessary surgery. Spontaneous resolution is more likely to occur if the patient is asymptomatic or has mild neurological symptoms, if the hematoma is located in the frontal region, is thin, has minimal mass effect, has low density line indicating CSF between the hematoma and cortex on imaging (2). However spontaneous resolution of chronic subdural hematoma in presence of neurological deficit and evidence of mass effect on imaging is rarely reported (2,3). Acute subdural hematoma very rarely undergoes spontaneous resolution (4). The possible mechanism is that there is redistribution of blood and absorption of blood after coming in contact with cerebrospinal fluid (4). Controversy exists regarding the

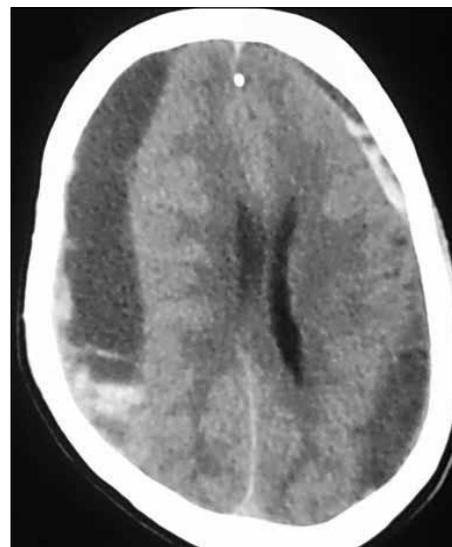


Figure 1: CT of the brain reveals a hypodense collection of 1.5 cm thickness in the right frontotemporoparietal region with areas of hyperdensity in the parietal region, suggestive of a predominant chronic subdural hematoma with an acute component. Significant mass effect resulting in compression of the ipsilateral lateral ventricle and midline shift of 8 mm to the left side was noted. Another hypodense collection in the left frontoparietal region with specks of hyperdensity in the left frontal region is evident.



Figure 2: CT of the brain reveals complete resolution of the left frontoparietal subdural hematoma after two years. A thin rim of right frontal subdural collection is noted with no evidence of midline shift or mass effect.

mechanism for subsequent increase in the size as well as resolution of chronic subdural hematoma. Probably, the same mechanisms which cause increase in size of the hematoma are responsible for its resorption. In chronic subdural hematoma, there are episodes of rebleed which result in increase in size of hematoma. One of the theories proposed is that the hematoma increases in size due to osmotic effect. This was later disapproved as osmolarity between the subdural fluid and cerebrospinal fluid is same (8). The next most accepted theory is recurrent breakdown of the outer membrane with bleeding from the vessels in its wall results in increase in hematoma size (7). This is more evident in patients with ITP where the failure of platelets to plug the openings in the blood vessels leads to recurrent bleeds.

The endothelial gap junctions are sometimes bridged by platelets, reducing microhemorrhage and size of the subdural hematoma (9). Modified smooth-muscle cells in the outer membrane might produce collagen that reinforces the membrane, reducing its fragility, thereby causing resolution of chronic subdural hematoma (5). The vessels of the subdural membrane have leaky walls which may allow for bidirectional flow thereby causing gradual resorption of the hematoma. This may be supported by the fact that burr hole evacuation allows for resolution of the hematoma even when the subdural hematoma is not completely evacuated. Loss of

integrity of the subdural membrane, whether surgically or due to the inherent propensity of the wall to breakdown, may lead to spontaneous resolution in some cases. Corticosteroids inhibit the formation of protein-permeable membrane. This results in maturation of the neomembrane and stabilisation of neovasculature which causes spontaneous resolution of the hematoma (6). However the factors which influence this event in some cases and not in all are yet to be understood.

The present case is unique as it demonstrates that spontaneous resolution of chronic subdural hematoma occurred after two years duration even in the presence of neurological deficit and mass effect and midline shift on imaging. The probable explanation is that the same pathological mechanisms which cause increase in size of the hematoma may be responsible for its resolution. It's a paradox that the same pathological process can lead to diametrically opposite outcome in the same patient over a period of time.

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