Spontaneous resolution of a large chronic subdural hematoma:
a case report and review of the literature

Geniş kronik subdural hematomun spontan rezolüsyonu: 
Olgu sunumu ve literatürün gözden geçirilmesi

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Spontaneous resolution of a chronic subdural hematoma has been reported rarely in the literature. We present herein the case of a 35-year-old patient with spontaneous resolution of a large chronic subdural hematoma, the volume of which was measured as approximately 76.5 ml on magnetic resonance imaging. No pathology was determined on neurological examination. Neither cerebral angiography nor EEG showed any abnormality. We discuss the possible relation between mechanisms of physio-pathogenesis and spontaneous resolution of chronic subdural hematomas, and also present the patient characteristics together with the other literature data.

Key Words: Chronic subdural hematoma; spontaneous resolution.


Anahtar Sözcükler: Kronik subdural hematom; spontan rezolüsyon.

CASE REPORT

A 35-year-old, left-handed man without no chronic illness struck the ball with his head in a football game, after which he complained of transient headache. A month later, he suffered speech disturbance with paraesthesias on his left arm transiently twice a month. He had been asymptomatic for a month when he presented to our facility. We did not find any pathology on neurologic examination. Magnetic resonance imaging (MRI) revealed right frontoparietal chronic subdural hematoma measuring approximately 76.5 ml; there was no midline shift except for mild compression of the right ventricle (Fig. 1). Neither cerebral angiography nor EEG showed any abnormality (Fig. 2). Interestingly, he had been going about his daily activities and had

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also been participating in exhausting sports such as squash and trekking just prior to admission. The patient was observed expectantly since he was asymptomatic, and at the end of a 135-day follow-up period, the hematoma had resolved completely on MRI scans (Fig. 3). A graphical correlation was carried out showing the relationship between time and hematoma volume resolution (Fig. 4).

**DISCUSSION**

Chronic subdural hematomas are usually enlarging and organizing lesions but they may resolve or calcify rarely. By definition, these lesions are at least three weeks old and can be verified when typically dark, liquefied hematoma fluid and a capsule formation are demonstrated at operation.\[^7\] Grumme et al.\[^3\] also classified chronic subdural hematomas in three types according to computerized tomography (CT) findings: hematomas with lower attenuation values than brain tissue, nonhomogeneous hematomas with elements of differing densities, and hematomas with the same density as brain tissue. Some chronic subdural hematomas have arisen from traumatic subdural hygromas and also from acute subdural hematomas.\[^8-10\]

Various theories have been suggested to explain the mechanisms of formation and resolution of chronic subdural hematomas. In 1981, Kawano and Suzuki\[^2\] stated that the modified smooth-muscle

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**Fig. 1.** Right frontoparietal hyperintense images on T1-weighted MRI scans, sagittal (a) and axial (b) views at initial presentation.

**Fig. 2.** MR angiography (a) and digital subtraction angiography with arterial (b) and venous (c) phases are shown.
cells in the outer membrane might play a role in the resolution of chronic subdural hematoma, since the cells produce collagen that reinforces the membrane and reduces its fragility. In 1983, Yamashima et al.\textsuperscript{5} investigated, by electron microscopy, the structure of macrocapillaries, also called ‘sinusoid’, in the outer membrane of subdural hematomas. They suggested that endothelial gap junctions of macrocapillaries in the outer membrane play an important role in the leakage of blood, causing enlargement of chronic subdural hematomas by microhemorrhages and increased fibrinolytic activity. The endothelial gap junctions are sometimes bridged by platelets, reducing microhemorrhages and the size of the subdural hematoma.\textsuperscript{11} Nakamura et al.\textsuperscript{11} stated that the decreased fibrinolytic activity of the hematoma capsule and of the fluid might have caused a spontaneous resolution.

In 1987, Giuffrè\textsuperscript{12} suggested that hormonal factors, in addition to mechanical, hematogenic and vasogenic factors, could play an important role in the pathogenesis of these hematomas. In an experimental study, Glover and Labadie\textsuperscript{12} proposed that corticosteroids inhibited the formation of protein-permeable membrane, decreasing the size of chronic subdural hematoma. Recently, Lee\textsuperscript{13} postulated that maturation of the neomembrane and stabilization of the neovasculature might eventually result in spontaneous resolution.

Some case examples have also been presented in the literature (Table 1). Naganuma et al.\textsuperscript{7} presented four cases and stated that patients who have no or mild clinical signs and lesions with the characteristics of small size, low or isodensity and ventricular dilatation on CT scan, could have shown spontaneous resolution. Horikoshi et al.\textsuperscript{9} observed four cases and all spontaneously resolving lesions were asymptomatic or only caused mild transient headache, were located in the frontal region, and did not show severe mass effect on CT scan. Parlato et al.\textsuperscript{11} presented five patients and postulated that age over 70 years, worsening mental function, the presence of brain atrophy, and the absence of clinical and radiological findings facilitate choosing conservative therapy.

### Table 1. Literature data about spontaneously resolved chronic subdural hematomas

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Cases</th>
<th>Resolution time (day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naganuma, 1986</td>
<td>4</td>
<td>78-231</td>
</tr>
<tr>
<td>Horikoshi, 1998</td>
<td>4</td>
<td>30-360</td>
</tr>
<tr>
<td>Parlato, 2000</td>
<td>5</td>
<td>42-50</td>
</tr>
<tr>
<td>Our case, 2004</td>
<td>1</td>
<td>135</td>
</tr>
</tbody>
</table>
In our case, several clinical features such as mild symptoms, frontal localization and no significant mass effect correlated with the data of other reports; however, our patient was unusual because he was young, athletic, had no chronic illness, and his lesion resulted from only a slight head trauma. Our interest in this case is because of its benign clinical course and the absence of severe mass effect or midline shift despite the large-volume hematoma. Since there was no neovascularization on angiography and the patient could proceed with his daily routine and sports activities, conservative therapy was the logical approach for this pathology, except that it was not cost-effective. The patient also did not have to risk any possible complications of surgery. Finally, unlike classically known lesions that enlarge, chronic subdural hematomas may resolve spontaneously in rare patients having several clinical and radiological parameters.

REFERENCES