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Case Report

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Regression of chronic subdural hematoma under antiplatelet treatment: An unusual case

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Abstract

The surgical evacuation is considered as the first choice of treatment for symptomatic chronic subdural hematoma. Since anticoagulation is considered a risk factor and any ongoing anticoagulation treatment is ceased once the diagnosis is confirmed, the data regarding the course of these cases under ongoing anticoagulant treatment remains extremely limited. A 73-year-old female was given antiplatelet treatment for ischemic stroke for right hemiparesis and dysarthria despite an existing, but unrecognized chronic subdural hematoma. The hematoma was discovered after 4 weeks following antiplatelet treatment, but in a smaller volume. As routine practice, antiplatelet treatment was ceased, and the hematoma completely resolved within 2 weeks. This case demonstrates that some patients with chronic subdural hematoma for whom continuation of anticoagulation is essential, can be observed closely if hematoma volume is small, and no midline shift is present since spontaneous resolution may occur.

Keywords: chronic subdural hematoma, spontaneous, resolution, nonsurgical, conservative, antiplatelet

1. Introduction

Chronic subdural hematoma (cSDH) is predominantly a disease of the elderly and its incidence increases as the population ages and anticoagulation/antiplatelet drug usage increases (1). Surgical evacuation is a highly effective treatment and first choice of option for symptomatic cases (1, 2).

The success of nonsurgical treatments varies from case to case leaving the natural history of the disease in oblivion (3). Similarly, since anticoagulant/antiplatelet drugs are among the risk factors and they are discontinued immediately following the diagnosis, there is little known about the natural course of the disease under these drugs (1, 3). The factors affecting the good outcome for nonsurgical management are not completely understood, but small hematoma volume, minimal mass effect and midline shift, frontal placement, and low density on computerized tomography (CT) are said to be predictors for spontaneous resolution (3, 4).

We present a case of chronic subdural hematoma that showed regression despite ongoing antiplatelet treatment.

2. Case Report

A 73-year-old female was admitted with right hemiparesis and dysarthria following 4-days of a headache to the emergency department. She had hypertension for 15 years. Emergency brain CT (Fig. 1a) and diffusion-weighted magnetic resonance imaging (MRI) were performed (Fig. 1b). Though there was a left parietal chronic subdural hematoma (12mm in thickness) on CT, it remained unrecognized at the time. She was hospitalized with a preliminary diagnosis of thromboembolic cerebrovascular disease and put on acetylsalicylic acid (1x100mg p.o. daily) and enoxaparin sodium (2x6000IU s.c. daily) by neurology. Following the improvement of hemiparesis and dysarthria, she was discharged on acetylsalicylic acid after 4 days.

During routine follow-up visit after one month, control brain MRI revealed the regressed left parietal chronic subdural hematoma with a thickness of 8mm, and then the patient was consulted to our department (Fig. 1c). When the patient's records were inspected retrospectively, it was understood that the cSDH was also present at the time of hospitalization. Moreover, the patient had a minor head trauma one month

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before the onset of complaints, for which she was evaluated by a CT scan that turned out to be normal. Although hematoma regressed under 4 days of enoxaparin sodium and one month of acetylsalicylic acid treatments, the antiplatelet treatment is discontinued as common practice. Control CT scan after 15 days revealed a complete resolution of cSDH (Fig. 1d).

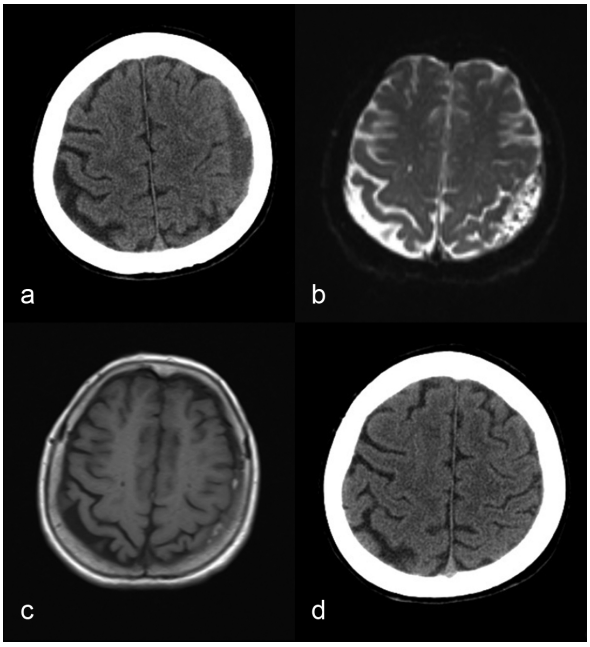

Fig. 1. (a) Nonrecognized parietal chronic subdural hematoma on the left on axial CT scan. (b) The corresponding area on diffuson weighted MRI, which was probably mistaken as cerebrovascular event. (c) Decreased hematoma despite anticoagulation for 1 month on axial T1 weighted MRI. (d) Completely resolved hematoma 2 weeks after anticoagulation was stopped.

3. Discussion

Chronic subdural hematoma is considered to occur following tearing of bridging veins between the dura mater and arachnoid mater, mostly as a result of minor head trauma (2). An inflammatory process is triggered by blood in the subdural space and after a few weeks a neomembrane forms (2). Maintenance or growth of the hematoma is attributed to the fragility of neocapillaries in the membrane that result in further microbleeding, accelerated fibrinolysis, increased concentration of fibrin degradation products, and vascular endothelial growth factor in the subdural space (2, 4).

Since most cSDH have been treated surgically which interrupts the disease process, the natural course of the disease is unclear (2). Many neurosurgeons are familiar with the concept of spontaneously resolved cSDH, however, there are no widely accepted criteria that predict spontaneous resolution at the moment. Minimal neurological deficit, small hematoma volume, low or isodensity on CT, ventricular dilation, frontal hematoma are hypothesized to be related to spontaneous resolution (2, 3). In one of the largest series on this topic, authors suggest considering close observation when hematoma volume, thickness, or midline shift is less than 43mm^3, 13mm, and 5mm, respectively (4).

The association of anticoagulant/antiplatelet drugs with cSDH is well documented. It is thought that anticoagulant/antiplatelet drugs inhibit platelet plug formation in the microcapillaries which otherwise reduce bleeding (5). However, the data regarding disease course under ongoing anticoagulant/antiplatelet treatment is very limited, probably because anticoagulant/antiplatelet treatment is immediately ceased when cSDH is detected.

A recently published report of two cases showed a stable course and decrease in cSDH where patients had to receive anticoagulant therapy after middle meningeal artery embolization (6). In one case, the patient was not given any anticoagulation for the first 8 days and anticoagulation was re-initiated after regression in the hematoma volume was confirmed (6). The cSDH continued to regress during sequential treatment with low molecular weight heparin, warfarin, and enoxaparin (6). The second case was associated with warfarin use which was discontinued once the cSDH was detected. During the course he was given enoxaparin, then warfarin for venous sinus thrombosis (6). The hematoma didn't increase in 5 days and the patient remained symptom-free at the 8th-week follow-up (6). Unfortunately no control imaging was performed (6). In another case with a non-traumatic cSDH, antiplatelet drug treatment (acetylsalicylic acid) had to be initiated and continued due to accompanying cerebellar ischemia (5). The cSDH thickness showed an increase during the first 4 weeks, but then spontaneously resolved within the next 5 months (5).

In our case, the patient was given an antiplatelet drug for a month while accompanied by an unrecognized cSDH. During this period, the hematoma decreased in size, and once it was detected, it was smaller in volume compared to the initial imaging studies. However, not all cases under ongoing anticoagulant/antiplatelet drugs showed resolution or regression. Yang et al described a patient with cSDH that continued to receive acetylsalicylic acid and clopidogrel (both reduced by half) for ischemic heart disease (7). She didn't accept surgical treatment and mannitol treatment is initiated (7). Though the hematoma volume didn't increase significantly, it became a mixed type and all the antiaggregant treatment had to be stopped (7). The hematoma spontaneously resolved thereafter (7). It must be noted that all the cases that showed spontaneous resolution under anticoagulation treatment had a relatively thin hematoma with no midline shift.

In conclusion, though anticoagulation is a risk factor for cSDH, there is a chance of spontaneous resolution or stable course for cSDH that are small in volume and not accompanied by midline shift in cases where the anticoagulation can not be discontinued. Further controlled studies are needed to better understand the course of the disease under anticoagulation, to

assess safety of anticoagulant use in these patients, and to find out which factors are associated with this phenomenon.

Informed Consent

The patient's relatives have consented to the submission of the case report for submission to the journal.

Conflict of interest

The authors have no conflict of interest.

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Authors' contributions

Concept: S.B, F.T., Design: S.B, F.T., Data Collection or Processing: F.T., S.B., Analysis or Interpretation: S.B, F.T., Literature Search: S.B, F.T., Writing: S.B, F.T.

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