

Case Report

Spontaneous Resorption of Chronic Subdural Hematoma with Curative Thrombolysis and Atorvastatin

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Abstract: The aim of this study is to report a case of chronic subdural hematoma with antivitamin K curative dose, treated by Atorvastatin. Clinical case: A 48-year-old man was admitted for motor deficit of the left complete hemiparesis, associated with dysarthria. He was followed in Neurology and cardiology for ischemic stroke due to atrial fibrillation, and was treated with antivitamin K treatment for 2 years. The clinical examination showed a Glasgow score at 15, a left hemiparesis with left facial paralysis. Sensitivity was preserved. Brain CT-scan showed a right fronto-parietal subacute hematoma with mass effect and a recent ischemic stroke of a central branch of the left middle cerebral artery. Angio-MRI shows an occlusion of the right vertebral artery and right communicating artery. A curative anticoagulation was initiated. Atorvastatin was administered at 20 mg per night for 8 weeks associated with functional rehabilitation. The evolution had been favorable, with the regression of headaches and hemiparesis, however the persistence of facial paralysis. After 8 months, the patient was asymptomatic. Cerebral CT control showed complete regression of subdural hematoma. Conclusion: Atorvastatin may constitute a non-surgical and effective treatment of chronic subdural hematoma among the patients whose curative anticoagulation cannot be interrupted. It is very important to really value the report risks / profits of every therapeutic option before making a decision.

Keywords: Chronic Subdural Hematoma, Atorvastatin, Nonsurgical Treatment, Anticoagulant

1. Introduction

The incidence of chronic subdural hematomas is increasing worldwide [1, 2]. More than 80% of patients over 60 years old have a clinical history of head trauma [3-5]. The increase in life expectancy and the use of short and long-term preventive and curative anticoagulation and medication

would explain this increase [1, 6]. The management of these patients carries considerable difficulties due to the risk of hemorrhagic, and recurrence [1, 6]. New therapeutic strategies have been proposed in patients with a high surgical risk or a high recurrence rate [3, 6]. We report a case of spontaneous resorption of chronic subdural hematoma in a patient receiving a curative anticoagulation and Atorvastatin.

2. Clinical Case

This is a 48-year-old who was referred to the Cardiology Department for a left complete hemiparesis and language disorders of sudden onset in a context of dizziness and headache. He had a medical history of high blood pressure, anticoagulant treatment for two years because of chronic atrial fibrillation that was complicated by multiple ischemic strokes. His diabetes was stabilized by diet and an oral antidiabetic medication. At admission, blood pressure was measured at 134/95 mmHg and pulse at 77 /mn. Glasgow's score was estimated at 15. There was left facial paralysis and dysarthria with left hemiparesis. The muscular strength was quoted as 3 on a scale of 5. Sensitivity was preserved. Brain CT-scan showed a right fronto-parietal subacute hematoma 19 mm thick (Figure 1a) associated with mass effect (Figure 1b) and a recent ischemic stroke of a central branch of the left middle cerebral artery (Figure 1c). Brain MRI showed the subdural hematoma that appeared increased and measured 23 mm (Figure 2a) and subacute ischemia in the superficial and deep territories of the left middle cerebral artery (Figure 2b). The study of the vascular vascular-weighted of the Circle of Willis showed a defect in clouding and circulation of the right communicating artery and the right vertebral artery (Figure 2c).

The management of the hematoma was medical. The patient was hospitalized under clinical and biological supervision for 15 days. Atorvastatin was given at a dose of 20 mg per night for 8 weeks. The antivitamin K was stopped and replaced with a low molecular weight heparin curative dose, under international normalized ratio (INR) control (goal 2-3). The relay was carried out with antivitamin K. Analgesic treatment included Nefopam 20 mg. Anti-hypertensive treatment and his heart disease (Perindopril 10 mg-Amlodipine 5 mg, Nebivolol 5 mg) was continued. Functional motor rehabilitation completed the management. The patient's neurological functions were evaluated before treatment and 1 week, 2 weeks, 1 month, 3 months after treatment. The evolution was marked by a recovery of hemiparesis and regression of the headaches. After 8 months, he persisted with residual facial paralysis that is undergoing functional rehabilitation. Brain control CT showed complete resorption of the subdural hematoma (Figure 3). The patient's follow-up continues in cardiology and neurology.

3. Discussion

Conservative treatments for chronic subdural hematoma (CSDH) that use Atorvastatin have not yet been well established. Several studies on the use of Atorvastatin are reported in the literature on studies of Chinese patients with CSDH [6-8]. In our patient, this is a conservative treatment using atorvastatin is administered to a patient with CSDH, on anticoagulant treatment without altering the curative dose for an ischemic vascular pathology. In published series, doses are generally reduced or temporarily suspended during medical or surgical management of subdural hematoma [5, 7]. It is a

common condition that occurs willingly in subjects over 60 years [3, 9] because there is then a certain fragility of the vessels. They are the preserve of subjects undergoing long-term oral anticoagulant therapy (antivitamin K) or antiplatelet medication [7], as these thin the blood and thus promote hemorrhage [7]. The authors reported incidence of CSDH with anticoagulant between 21% and 36% [10].

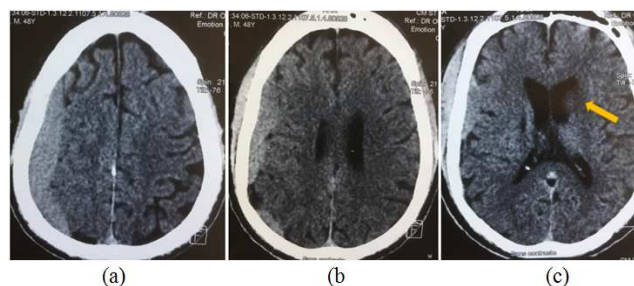


Figure 1. Axial Brain CT-Scan showed a right hemispheric subdural hematoma (Figure 1a), mass effect on cortical mantle (Figure 1b) and showed an acute ischemia in the territory of the superficial and deep territory of the left middle cerebral artery (Figure 1c).

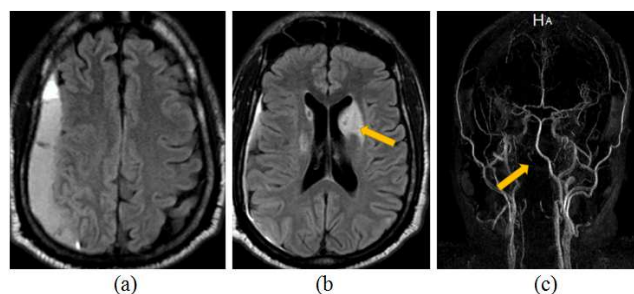


Figure 2. Axial MRI on the T1- weighted showed the subdural hematoma that increased and measured 23 mm (Figure 2a) and on the Flair -weighted (Figure 2b), showed an acute ischemia in the territory of the shallow and deep territory of the left middle brain (Yellow Arrow). The vascular weighted showed an occlusion (Yellow arrow) of the right vertebral artery (Figure 2c).

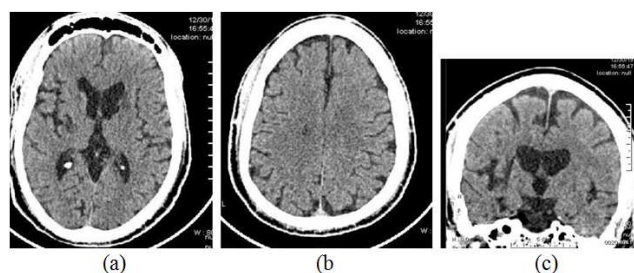


Figure 3. Brain CT-Scan control showed the absence of subdural hematoma on axial (Figure 3a and Figure 3b) and coronal reconstruction (Figure 3c).

In our relatively young patient, it was a spontaneous subdural hematoma occurring during prophylactic anticoagulation medication of rhythmic heart disease, complicated by several ischemic strokes. Initial bleeding can be caused by an often-benign head trauma, which sometimes goes unnoticed [11] not reported in our patient. The hematoma, which develops gradually, will degrade with fibrin degradation products. It will cause local inflammation, with the formation of neo-membranes and neovascularization [11]. The growth of the hematoma will be caused by small

repetitive bleeding of these neo-vessels [12]. The production of plasminogen is responsible for hyperfibrinolysis and therefore increased production of fibrin degradation products that will sustain the hematoma [11]. The long-term anticoagulation found in our patient, intervenes as favorable factors, which by vascular fragility and the increase of hyperfibrinolysis participates in the growth and maintenance of the hematoma [11]. This explains in practice its reduction or even its cessation in CSDH management. The more or less large volume will condition the state of suffering from the parenchyma and be the cause of a polymorphic clinical symptomatology [3, 2]. Clinical presentations are variable progressive in published journals [5, 13]. In our case, headaches were present and associated with the onset of deficit. The initial brutality remains unusual. It could fit into ischemic vascular symptomatology by considering the history of ischemic vascular pathology in our patient with emboligenic heart disease. Sometimes it can be a of higher functions (memory), behavioral problems, hemiplegia or even a severe coma [5, 8]. The first-line brain CT was performed in our case for the diagnosis of CSDH. In general, it is a good diagnostic and monitoring tool. However, given our patient's medical history and the sudden onset of symptoms an MRI had been requested and which had an increase in the size of the hematoma subdural, an of the left middle cerebral artery and a defect of clouding of the vertebral artery and the right communicating artery. ischemias's warranted curative anticoagulation with a risk of increased subdural hematoma. Spontaneous resolution of CSDH, in patients with minor or asymptomatic functional signs associated with a small volume CSDH, may occur within the same time (4 to 8 weeks) [10, 14]. A simple monitoring was not possible for our patient, as spontaneous resolution was uncertain. There was a risk of hematoma increase with lower tolerance. Surgical evacuation is in patients who deteriorate or do not improve after conservative treatment [13]. Surgery is currently the of choice in symptomatic CSDH [13, 14]. Surgical of CSDH generally include burr hole, percutaneous drainage, twist-drill drainage, craniotomy, and endoscopic surgery [12]. However, the risk of hemorrhagic and recurrence is considerable when the patient receives anticoagulant or anti-platelet medication [5] as described for our patient. Strategies for preoperative optimization of platelet function and clotting of these patients have evolved considerably. However, discontinuation of the drug and the inversion strategies (vitamin K) that are suggested in chronic subdural hematomas could not be applied to our patient. Anticoagulant treatment was vital. However, some patients may not need to stop their medications during the traditional 7 days before hole [5]. This could be important, as delays in the start-up of these therapies are associated with thromboembolic risk [5]. Delayed surgery, while managing anticoagulant and antiplatelet therapies, may also be associated with increased morbidity [5]. Various studies have reported positive results non-surgical treatment [3, 10, 12, 13]. It is usually indicated chronic small size subdural hematomas or asymptomatic

patients [13]. Of all the drugs used, steroids were the most common [3, 10, 12]. Its isolated use or as an adjuvant has associated with morbidity [3, 9] especially when combined with surgery [9]. They are associated with side effects and limited indications (diabetes, high blood pressure, Gastric ulcer etc....) [3, 9, 12]. Indeed, the patient was diabetic and had high blood pressure, which contraindicated steroid use. Recent ischemic stroke and occlusion of the vertebral and the middle cerebral arteries required curative anticoagulant therapy. Reducing doses or temporarily discontinuing anticoagulant therapy could have had serious consequences. In general, anticoagulant therapy is given in a reduced dose if suspended [5, 7]. In our case, anticoagulation was pursued a curative dose and a relay by the antivitamins K was performed. This treatment was dictated by the patient's multiple vascular ischemia. The high risk of a new stroke justifies this treatment option. A surgical evacuation of the hematoma therefore presented a post-operative hemorrhagic risk for us. Statin administration was the best option in drawing on favorable results that are reported on large series of Chinese patients [4, 8, 14]. Atorvastatin is an inhibitor of 3-Hydroxy-3-methylglutaryl (HMG)-COA reductase. It was used for the treatment of patients with high cholesterol and heart disease. This molecule improves angiogenesis and reduces inflammation [8]. By reducing vascular leakage induced by local inflammation atorvastatin at the 20 mg dose would prevent formation and accelerate the absorption of hematoma [4, 8]. No documented side effects have been reported on statin [4, 14], and we have also not observed any adverse effects in our patient. The treatment was well supported. Strict regular monitoring was carried out for our patient. The difficulty was the dosage of this drug which was precisely adapted according to the result of regular blood Too low an INR reflects insufficient action and a risk of clots. Too high INR results in an overdose and an increased risk of hemorrhage. In the majority of cases, the INR must be between 2 and 3. The patient's improvement had been rapid the first weeks of treatment. This had been observed in the published journal [4, 8]. Patients with CSDH taking atorvastatin were twice as likely to have improved neurological function and a greater decrease in the volume of hematoma on statin improved faster than placebo patients [4]. In patients with a clinical history of anticoagulant treatment treated with atorvastatin, patients have a better prognosis [7, 15]. CSDH is fully absorbed within 3 months [15]. We that under these particular conditions (patients with CSDH and on anticoagulant), atorvastatin remains the best without risk of recurrence or worsening of the hematoma. prognosis was favorable in the majority of cases. In the Chinese series, only 11,2 to 16% had received additional surgery. Atorvastatin is associated with a low risk of recurrence of CSDH [6, 14]. In this case, it may be associated with surgery, which is associated with a high morbidity rate mortality [8, 14]. It would significantly reduce the risk of recurrence [3, 8]. The use of anticoagulant and anti-platelet medication has been implicated in the development and recurrence of CSDH [5, 10, 12]. In our experience the

evolution was favorable with a complete resorption of the hematoma in eight weeks in a patient with anticoagulation medication. Comparative studies were conducted on patients who had received anticoagulation medication associated with atorvastatin and a control group. The group treated with atorvastatin showed the best prognosis [7, 8, 15]. Several studies on small samples have described a significant reduction in CSDH in the first month after daily oral administration of 20 mg of atorvastatin [7, 8, 14, 15]. The patient was stay in hospital with strict clinical and biological supervision for the first 15 days of treatment. Neurological improvement was noted during this period. Outpatient follow-up was permitted after satisfactory control of clinical and biological parameters. For our patient, the choice conservative treatment with close supervision and surgery a challenge. The development of a therapeutic strategy must be adapted to each patient according to the risks involved.

4. Conclusion

Atorvastatin may be an effective conservative therapy for CSDH in patients whose continuation of curative anticoagulation is vital and cannot be interrupted. It is, therefore, very important to properly assess the risk/benefit ratio of each treatment option before making a decision. The development of a therapeutic strategy must be tailored to each case, and should take into account the comorbidities and possible consequences of stopping anticoagulation in patients with high-risk embolism.

Conflicts of Interest

The authors declare that they have no competing interests.

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Informed Consent: Written informed consent was obtained from the patient for publication of this manuscript and any accompanying images.

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