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Letter to the editor

To the Editor,

With great interest I read the paper by Dr. Morelli et al. entitled "Spinal epidural hematoma onset with Horner syndrome" (1). The authors described a patient under anticoagulant treatment, presenting with hypoesthesia of the right arm. Thorough neurological examination revealed a Horner Syndrome on the right side as the only clinical symptom. MRI of the spine resulted in the diagnosis *subacute* posterior spinal epidural hematoma at C6-T1. Anticoagulant therapy was disrupted, and neurological signs improved with conservative treatment. The readership remains uninformed about the results (if any) of follow-up MRI. The authors explained that this case illustrates that early discontinuation of anticoagulant therapy may significantly improve the prognosis of patients with SSEH.

At present there is a significant amount of literature about the treatment of patients with spontaneous spinal epidural hematomas (SSEH) (2, 3). Most patients require acute operative decompression, however, also patients with spontaneous resolution have been described. The role of anticoagulants in the etiology of SSEH still remains unsolved, but it is evident that, in general, hematomas in patients with clotting abnormalities are larger in size. However, larger hematomas are not equivalent with more severe neurological deficit (3). On the contrary, in patients with coagulopathy, hematomas were larger in the conservative group($SSEH^{cons}$) when compared with operative cases($SSEH^{oper}$) (3). It has been speculated that spreading of the hematoma along the spinal epidural space in the acute stage of hemorrhage (before clotting) plays a role in decompression of the intradural neural structures, promoting the spontaneous relief of neurological deficit (3).

In Morelli's case, MRI revealed a subacute (S)SEH, which implicates that at the time of diagnosis the hematoma did exist for more than 24 hours (4). Most SSEHs present with acute severe pain (in neck, back, arm or leg) followed by (rapidly) progressive sensory and/or motor deficit. In this patient no initial pain is reported, and other alarming signs or symptoms apparently were absent. The referral to the neurologist most likely has been in the "subacute" stage, which is in line with MRI findings. Therefore, the statement by the authors that discontinuation of anticoagulants has improved the chances for (spontaneous) recovery of their patient is wrong. In case of an ongoing bleeding, symptoms would have been severe and progressive, and discontinuation of anticoagulants without active correction of the clotting time would have been without any effect. Such a patient would end up with a para- or tetraplegia.

In the past, we have analysed a series of 333 patients with SSEH (2). A total of 89 patients suffered coagulopathy (caused by medication in 77, and by disease in 12). In 81 (91%) of these patients solid hematomas were reported during surgery, and in 8 (9%) patients "semi-solid hematomas" were encountered. Of the remaining 244 patients (without coagulopathy), solid hematomas were reported in 238 (97.5%). A "liquid hematoma" was reported in only one patient (5), and in five patients profuse bleeding occurred during the removal of the epidural blood clot. This means that the intra-operative finding of an active spinal epidural bleeding is exceptional (6).

As such, the case reported by Morelli *et al.* is another example of a benign course of a SSEH. It illustrates that also in patients with anticoagulants, spontaneous resolution of SSEH may occur. Nevertheless, most cases of SSEH, as is supported by the large number of cases reported in the literature, require urgent operative decompression. Conservative treatment of SSEH may only be considered in mild cases with a benign clinical course and provided that the patient is closely followed neurologically, as in the present case by Dr. Morelli *et al.* Repeat MRI should always be part of the follow-up procedure in such cases.

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