CASE REPORT

Spontaneous iliopsoas muscle haematoma as a complication of anticoagulation in acute cerebral venous thrombosis: to stop or not to stop (the anticoagulation)?

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SUMMARY

Spontaneous iliopsoas muscle haematoma is an infrequent complication of anticoagulation, potentially causing neurological dysfunction through compression of the femoral nerve or lumbar plexus. The authors report the case of a puerperal woman admitted for an extensive cerebral venous thrombosis. Anticoagulation was started, with clinical improvement. The patient later reported low back pain irradiating to the right thigh and developed neurological impairment consistent with lumbar plexus dysfunction. A pelvic CT scan revealed a right iliopsoas muscle haematoma. Considering the risk of anticoagulation suspension, a conservative approach was chosen, with maintenance of anticoagulation. Clinical and functional improvement occurred, with mild right hip and knee flexion paresis as sequelae. Anticoagulation complications are challenging, especially when interruption of anticoagulation may threaten vital and functional outcomes. Therefore, a careful evaluation is essential, since no clinical guidelines are available. In this case, continuing anticoagulation provided a good functional outcome.

BACKGROUND

Cerebral venous thrombosis is a rare type of stroke, mainly affecting young women. Anticoagulation is the mainstay of its treatment and should be initiated as promptly as possible.¹ The occurrence of a spontaneous iliac or psoas muscle haematoma is an infrequent complication of anticoagulation. This condition has been previously accounted for only in isolated case reports and small case series, its incidence being, therefore, little known.² It is most commonly reported as a complication of anticoagulation and, more rarely, in the setting of a clotting disorder or sports injury.^{3–5} Owing to the anatomical proximity of these muscles to the lumbar plexus and the femoral nerve, this can be a source of neurological dysfunction, through the compression of the former or, more frequently, the latter.^{6–7} The optimal treatment for this condition remains controversial.

CASE PRESENTATION

A 42-year-old woman with a history of one spontaneous abortion the year before, underwent a preterm labour (29th week) for suspected pre-eclampsia and fetal suffering. Two weeks later, she developed a persistent and progressively worsening non-throbbing left hemicranial headache, accompanied by nausea

and vomiting that evolved to acute clinical deterioration with altered mental status, mutism and gait impairment. On admission, she was obtunded, with absent verbal response, conjugate gaze deviation to the right, left hemiparesis and left extensor plantar reflex. A head CT scan revealed an extensive cerebral venous thrombosis, involving the superior sagittal sinus and its subsidiary cortical veins, the straight sinus, the great vein of Galen, the basal vein, the left transverse and sigmoid sinuses, the ipsilateral internal jugular vein and the right internal cerebral veins. Brain MRI confirmed the extensive venous thrombosis and showed multiple parenchymal lesions involving the thalamus, basal ganglia and internal capsule bilaterally, and also in the periventricular white matter and the posterior frontal subcortical white matter on the right, these last instances showing restriction in the *apparent diffusion coefficient* sequences, corresponding to venous infarctions (figures 1 and 2). The patient was started on intravenous heparin, which was later changed to warfarin. After clinical stability was attained, the patient also initiated a physiotherapy programme, which was uneventful, and no evidence of trauma or falls occurred during these sessions.

Figure 1 Sagittal T1-weighted MRI documenting signal hyperintensity in the superior sagittal sinus, the straight sinus and the vein of Galen (arrows), corresponding to a venous thrombosis and, therefore, demonstrating an involvement of both the superficial and deep venous system of the brain.

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Figure 2 Axial T2-weighted MRI showing extensive bilateral parenchymal lesions involving the thalamus and lenticulocapsular regions (arrows), and intraventricular haemorrhage in the right lateral ventricle (arrowhead).

A gradual and marked clinical improvement was observed in the following days, with nearly complete neurological recovery, aside from a slight left hemiparesis (grade 4+/5 Medical Research Council scale (MRCs)).

An extensive investigation for prothrombotic conditions was performed, including: antithrombin III, protein S and protein C measurements; genetic testing for factor V Leiden G1691A and prothrombin G20210A gene mutations; lupus anticoagulant, anticardiolipin and anti- β -2-glycoprotein 1 antibodies detection; autoimmune disorders screening with sedimentation rate, anti-nuclear antibodies and anti-double-stranded DNA antibodies; and homocysteine measurement. These investigations led to the diagnosis of a protein S deficiency (protein S activity on admission: 49% (normal range: 63–140%)).

Figure 3 Axial CT scan with contrast showing an extensive left iliopsoas haematoma, with a transversal diameter of approximately 7.7 cm (arrow). There is a predominant involvement of the iliacus muscle and significant displacement inwards of the psoas muscle (arrowhead).

One week after admission, the patient reported low back pain irradiating to the anterior portion of the right thigh. In the following days she developed weakness of the right thigh flexion and adduction (grade 4/5 MRCs) of the right knee extension (grade 1/5 MRCs), right quadriceps muscle atrophy, diminished right patellar tendon reflex and hypoesthesia in the inner surface of the right thigh. A lumbar CT scan suggested an increase in the volume of the right iliopsoas muscle, and a pelvic CT scan showed an extensive right iliopsoas muscle haematoma, with a diameter of 7.7 cm, extending longitudinally through the whole length of the muscle until its insertion (figures 3 and 4). In this setting, a lumbar plexus compression by the haematoma was admitted. The value of the *International Normalised Ratio* (INR) was within the target therapeutic range (namely, 2.1).

There was a significant drop of haemoglobin levels (greater than 2 g/dL: haemoglobin on admission was 13.1 g/dL; haemoglobin nadir after the diagnosis of the haematoma was 9.7 g/dL; haemoglobin at discharge was 11.4 g/dL), compatible and expected on the basis of the haematoma volume. However, the patient did not show any associated symptoms or clinical signs of haemodynamic compromise.

The management decision was to maintain the anticoagulation with warfarin (with the same target INR interval of 2–3), under strict clinical vigilance and INR monitoring. The patient was also kept on bed rest and the physiotherapy programme was considerably restricted.

In the following days, the lumbar and right thigh pain progressively subsided, and there was no further neurological deterioration. The patient was discharged 3 weeks later, after being referred to the neurology outpatient clinic. She also resumed the physiotherapy programme, uneventfully.

OUTCOME AND FOLLOW-UP

After discharge, the anticoagulation with warfarin was maintained with strict INR control, and the physiotherapy was progressively intensified. The initial diagnosis of protein S

Figure 4 Coronal reconstruction of the axial acquisition with contrast CT scan showing the length of the iliopsoas haematoma, which extends longitudinally involving the whole muscle, from its origin until its insertion (arrows). A significant displacement inwards of the psoas muscle, and also the iliac artery, is again seen (arrowhead).

deficiency, with a first measurement taking place on admission, hence, in the acute phase of the thrombotic event, was confirmed during follow-up, with a second measurement occurring 6 months later, for which the warfarin had been previously suspended for approximately 3 weeks (protein S activity on this second determination: 39% (normal range: 63–140%)). A potential indication for long-term anticoagulation was then considered. The anticoagulation with warfarin was, thus, maintained, and the patient was kept under follow-up. The patients' siblings were investigated for protein S deficiency, however, it was not found in any of them.

In the weeks following discharge from the neurology ward, the patient had significant clinical and functional improvement, maintaining only a slight right hip and knee flexion weakness (grade 4+/5 MRCs) and slight quadriceps muscle atrophy, which remained as sequelae but, however, not causing functional impact.

DISCUSSION

Anticoagulation-related haemorrhagic complications pose a considerable challenge, particularly when halting anticoagulation may worsen the primary disease. In a patient with cerebral venous thrombosis, an iliopsoas haematoma causing femoral nerve or lumbar plexus compression is an additional challenge, as in a first approach it may be mistaken for new brain lesions due to thrombosis progression and, if undiagnosed and unaddressed it can also result in permanent neurological impairment.⁸ This condition has not been previously reported as a complication of anticoagulation in a patient with cerebral venous thrombosis.

According to current guidelines addressing management of anticoagulant therapy, patients with high risk or active bleeding should be closely monitored concerning haemodynamic parameters and full blood count, including haematocrit, and, when presenting with major bleeding due to vitamin K antagonist use, rapid reversal of anticoagulation is indicated, particularly if the bleeding is life-threatening. Several treatment options are available for this purpose, the most consensual being combining vitamin K with one of prothrombin complex concentrate, fresh frozen plasma, or recombinant factor VIIa.⁹ In the case of anticoagulation-related retroperitoneal haematomas, treatment management may additionally include evacuation procedures.¹⁰

Regarding iliopsoas haematomas, the optimal treatment remains controversial, as current evidence favours neither the conservative management nor surgical or percutaneous drainage. All these therapeutic measures had good outcomes in previously published case reports. Generally, a conservative approach is chosen in smaller haematomas in haemodynamically stable patients with slight-to-moderate neurological impairment, and a drainage procedure is the option in larger haematomas with severe neurological impairment and/or haemodynamic instability.¹¹ However, stopping anticoagulation was always indicated in the reported cases.^{2 3 6 7 11}

In this patient, the lumbar plexus lesion could possibly lead to a permanent neurological impairment, particularly if the volume of the haematoma were to increase in the setting of maintenance of the anticoagulation. On the other hand, stopping anticoagulation in the subacute phase of an extensive cerebral venous thrombosis could be more functional and life-threatening, due to a greater potential neurological deterioration in the context of a central nervous system lesion than a

lumbar plexus compression. Considering the above, and the fact that the patient did not have any significant clinical evidence of a haemodynamic compromise due to the haematoma, the authors' decision was to favour conservative management and to maintain anticoagulation with warfarin, under strict clinical vigilance.

In similar cases involving haemodynamically stable patients, when halting anticoagulation may threaten the patients' vital or functional outcome, maintaining the anticoagulation may be considered. In our case, under strict clinical and INR monitoring, we report a favourable and eventless clinical outcome.

Learning points

- ▶ Given the broad indications to prescribe anticoagulation, it is important to be aware of the iliopsoas haematoma as a potentially serious complication.
- ▶ Until further evidence is available, the treatment approach should be chosen according to the clinical and haemodynamical stability of the patient, the severity of the potential neurological sequelae, and the risks associated with the suspension of anticoagulation.
- ▶ In patients for whom suspending anticoagulation may threaten vital outcomes, maintaining anticoagulation, in a setting of close clinical monitoring, may be a reasonable option.

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Competing interests None.

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