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EPIDURAL HEMATOMA IN A PATIENT WITH ACUTE PANCREATITIS-CASE REPORT

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A 59 year old female was admited to a regional hospital with the diagnosis of acute pancreatitis. In six days, epidural catheter was placed to treat the abdominal pain. Twelve hours later patient developed paraplegia. Blood tests done after the event revealed INR of 4.69. It was found that the patient received 40 mg of subcutaneos enoxaparin 5 hours before the epidural placement. Patient was transfered to the university clinic where MRI revealed epidural hematoma. She died 45 days after the original admission. Since coagulopathy may develop as the disease progresses, coagulation profile must be checked before epidural placement in patients with acute pancreatitis. *Acta Medica Medianae 2017;56(3):77-80.*

Key words: *acute pancreatitis, epidural hematoma, coagulopathy*

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Introduction

Epidural analgesia is a well established method for pain relief in patients with acute pancreatitis (1). Vascular thrombosis and systemic hypercoagulability are known complications of pancreatitis. Coagulopathy, on the other side, is pretty rare in early stages of this disease. We report the case of a patient suffering from acute pancreatitis who developed an epidural hematoma following epidural analgesia.

Case history

A 59 year old female was admitted to a small regional hospital with the diagnosis of acute pancreatitis. The disease was considered to be mild – there were no overt extra-pancreatic complications and blood tests did not show peripheral organ failure. Following fluid resuscitation and pain management with non-opiate analgesics, patient's general condition improved and she was discharged home after five days of treatment. Four days later, she was readmitted to the same hospital with abdominal pain. This time serum amylase and lipase were significantly elevated. There still was no known bleeding tendency. Despite an aggressive treatment abdominal pain continued to worsen and six days after the second admission it was decided to treat her pain with epidural analgesia. At 12.00 pm an 18-gauge Tuohy needle was inserted at L2-L3 level via a midline approach. The loss of resistance to air (2 ml) technique was used to identify epidural space and a 20 gauge nylon epidural catheter (BBraun) was advanced 3 cm into epidural space. No blood was aspirated through the catheter. Through the catheter 3 ml of 0.25% levobupivacaine were administered, which resulted in significant pain improvement. Additional 4 ml and 6 ml of 0.25% levobupivacaine were given in the epidural space at 17:00 and 23:00 hours respectively. Both doses resulted in complete pain relief. No additional doses of levobupivacaine were given.

The next morning patient complained of total inability to move her legs and lost the ability to control bladder and bowels. Blood tests were done immediately and revealed severe coagulopathy – PTT was 50.2s, PT 50.9s and INR 4.69. - The rest of the blood tests showed decreased albumin (28 g/L), decreased total protein (50g/L), while platelet count was increased (461 109/L). A neurologist was immediately consulted and he confirmed the presence of flaccid paralysis of lower extremities. Fresh frozen plasma was given at 9:00 am and epidural catheter was subsequently removed.

Later investigation discovered that patient had received 40 mg of subcutaneous enoxaparin 5

hours before the epidural placement and that patient was receiving a daily dose of enoxaparin for a total of 5 days. The only other medication that she was receiving was tramadol. The patient was transferred to the university hospital where MRI (done 5 days after the onset of paralysis) confirmed the diagnosis of epidural hematoma (Figure 1). It was spreading from L2 to L4 level, causing stenosis of the spinal channel. As an incidental finding, MRI revealed an ascites with suspected pleural carcinosis (Figure 1).

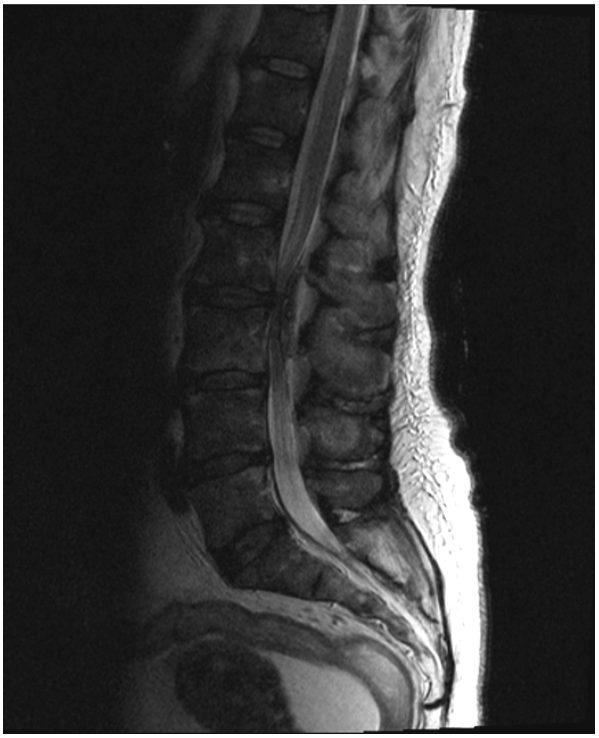

Figure 1.: Epidural hematoma on MRI

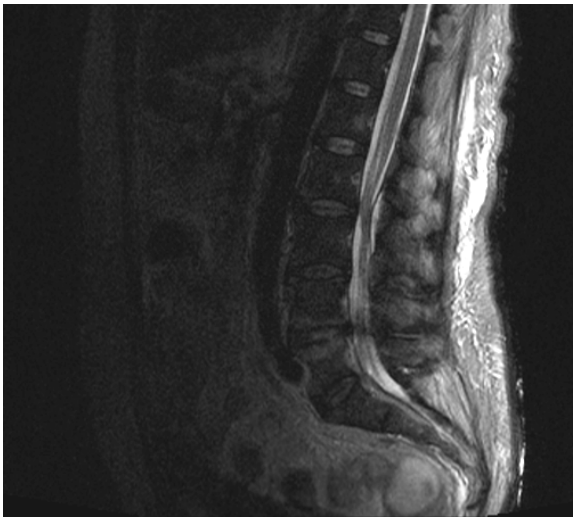

Figure 2.: Mild hematoma reabsorption

The neurosurgical team was consulted but because of severe general condition and the time interval since the beginning of paralysis (5 days), neurosurgeons decided not to perform laminectomy and the patient was transferred to the general surgery floor for pancreatitis treatment. Her neurological status did not change over the next several days and the second MRI was performed 10 days after the first one. It revealed only a mild hematoma reabsorption (Figure 2). Despite aggressive treatment, patient`s condition continued to deteriorate. There was no improvement in lower extremity paralysis and she expired 45 days after the original hospitalization.

Discussion

Patients with acute pancreatitis often suffer from severe visceral pain. Adequate pain relief is therefore one of the most important and urgent treatment goals. In general, good level of analgesia could be achieved combining opiate and non-opiate analgesics. Further, an excellent level of analgesia can be expected with epidural anesthesia in these patients (1). Another advantage of epidural analgesia is that it eliminates the need for systemic narcotics that can delay the return of bowel function in patients with acute pancreatitis. (2)

Neuraxial anesthesia is best avoided in patients with coagulopathy, significant thrombocytopenia, platelet dysfunction, or those who have received fibrinolytic/thrombolytic therapy (3). It has been suggested by Batem et al. that patients with acute pancreatitis are at decreased risk of epidural hematoma compared to the general population because of their relatively hypercoagulable state (4). Vascular thrombosis and systemic hypercoagulable state are known complications of pancreatitis (5, 6). The most probable explanation for hypercoagulability are increased levels of tissue factor (TF). Plasma levels of TF in AP patients have been shown to be higher than those in healthy volunteers (7). When patients with acute pancreatitis were divided into groups by the severity of their disease, the study by Sawa H. et al. did not show any statistically significant difference in the TF levels between the groups (7). It is also known that coagulation cascade is tightly regulated by three natural anticoagulant systems: antithrombin, the protein C pathway, and tissue factor pathway inhibitor. Each of them is often severely deteriorated in patients with AP (18-20)

The study conducted by Radenkovic D et al. has shown a prolonged prothrombin time in patients with AP (8). We have not found any reports of partial thromboplastin time (APTT) elevation in patients with AP.

Unfortunately, PT, PTT and INR were not checked before the epidural placement. Liver function tests (ALB 28g/L; AST 100U/L; ALT 114U/L; GGT 122U/L) done one day after the epidural placement and 5 days later indicated a progressively worse liver function (ALB 27 g/L; AST 373U/L; ALT 394U/L; GGT 414U/L). The patient's medical history was not suggestive of coagulation problems or alcohol abuse. Our patient did have

ascites, but aspiration probe was negative on blood.

It is difficult to speculate about what was the main culprit for the development of epidural hematoma. INR values of 4.69 indicated severe coagulopathy. Both placement and removal of epidural catheter could have caused hematoma formation. A study by Liu SS et al. found no increased risk of hematoma formation with the removal of epidural catheter even in patients with INR in the range of 1.5-7.1 (9). However, these patients had increased INR as a result of coumadine administration and all catheters were removed within 50 hours of coumadine administration (9). Our patient's increased INR was likely caused by liver failure. Enoxaparin that was administered 5 hours before the epidural placement could also be the reason for hematoma. Even an increased intra-abdominal pressure (due to ascites) that was causing engorgement of the epidural veins could have contributed to hematoma formation.

The American Society of Regional Anesthesia (ASRA) consensus statement clearly indicates that anesthesia should not be done if INR values are higher than 1.5 and neuroaxial anesthesia should not be done either sooner that 10-12 hours after prophylactic (40 mg) dose of enoxaparin (10, 11). The reason that prompted us to report this case was not the development of hematoma in the situation where ASRA guidelines suggested that neuroaxial anesthesia should not be placed. The development of epidural hematoma in similar clinical situations has been well documented (12-14). The reason to present the case was the development of coagulation abnormalities in the clinical situation where hypercoagulability is way more common. As said before, Badhal SS. et al. as well as Salmone T. et al. reported vascular thrombosis in patients with acute pancreatitis. (6, 7)

Literature review discovered more cases where patient with AP had an increased tendency to bleed (15-17). All of these cases had disseminated intravascular coagulation as an end result. However, our patient did not have any clinical signs of an increased tendency to bleed.

Conclusion

In conclusion, we can say that coagulation abnormalities are a hallmark of AP. Although the early stages of acute pancreatitis are characterized by microvascular thrombosis, coagulopathy may develop as disease progresses. Coagulation profile must be checked before epidural placement in patients with acute pancreatitis. Radiating back pain, sensory changes and muscular weakness progressing towards paraplegia should arouse suspicion of an epidural hematoma. Neuroradiological examination and decompressive surgery must be promptly performed then. In this case, due to lack of resources, the patient did not receive the treatment that is usually available in the Western world. As far as we know, this is the first reported case of epidural hematoma in Serbia and this case should prompt a better coordination of care between the hospitals which may prevent similar events in the future.

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Prikaz slučaja

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EPIDURALNI HEMATOM KOD BOLESNICE SA AKUTNIM PANKREATITISOM: PRIKAZ SLUČAJA

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Ženska osoba starosti 59 godina primljena je u regionalnu bolnicu sa dijagnozom akutnog pankreatitisa. Posle šest dana, postavljen joj je epiduralni kateter sa ciljem kupiranja bolova u abdomenu. Dvanaest sati kasnije, bolesnica je dobila paraplegiju. Odmah su urađene laboratorijske analize i uočen je INR od 4,69. Otkriveno je da je bolesnica dobila 40 mg enoksaparina potkožno pet sati pre epiduralne punkcije. Odmah je upućena u univerzitetsku kliniku gde je urađeno MRI snimanje i viđen epiduralni hematom. Preminula je 45 dana nakon prvog prijema u bolnicu. Pošto je nastanak koagulopatije moguć sa napredovanjem bolesti, koagulacioni status se mora proveriti pre postavljanja epiduralnog katetera kod bolesnika sa akutnim pankreatitisom. *Acta Medica Medianae 2017;56(3):77-80.*

Ključne reči: *akutni pankreatitis, epiduralni hematom, koagulopatija*