

SURGICALLY TREATED PARAPLEGIA AS A RESULT OF EPIDURAL HEMATOMA ASSOCIATED WITH EPIDURAL CATHETER REMOVAL IN A PATIENT RECEIVING ENOXAPARIN

ENOXAPARİN TEDAVİSİ ALAN BİR HASTADA EPİDURAL KATETER İLE BİRLİKTE GÖRÜLEN EPİDURAL HEMATOM SONUCU ORTAYA ÇIKAN PARAPLEJİNİN CERRAHİ TEDAVİSİ

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SUMMARY:

The spinal hematoma, defined as symptomatic bleeding within the spinal neuraxis, is a rare and potentially catastrophic complication of spinal or epidural anesthesia. Spinal hematoma reveals itself with the signs of cord compression. In the case of spinal cord compression, laminectomy seems to be effective in reversing spinal ischemia if performed within the first eight hours. We report a case of epidural hematoma formation after the removal of the lumbar epidural catheter in a patient who underwent total knee replacement surgery and commenced to receive low-molecular-weight heparin (LMWH) postoperatively for thromboembolic prophylaxis.

Key words: Epidural hematoma, epidural anesthesia, laminectomy, enoxoparin

Level of Evidence: Case report, Level IV

ÖZET:

Akson içine semptomatik kanama olan spinal hematom nadir bir lezyondur ve spinal veya epidural anestezinin katastrofik bir komplikasyonudur. Spinal kord basılarının tedavisinde ilk 8 saat içinde yapılacak laminektomi spinal iskeminin geri dönüşünü sağlayacaktır. Bu olgu sunumunda total diz protezi uygulanmış ve tromboemboli profilaksisi olarak düşük molekül ağırlıklı heparin kullanan bir olguda epidural kateterin çekilmesinden sonra oluşan epidural hematom olgusu sunulmuştur.

Anahtar Kelimeler: Epidural anestezi, epidural hematom, laminektomi, enoksiparin

Kanıt Düzeyi: Olgu sunumu, Düzey IV

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INTRODUCTION:

The spinal hematoma, defined as symptomatic bleeding within the spinal neuraxis, is a rare and potentially catastrophic complication of spinal or epidural anesthesia. Continuous epidural analgesia is commonly used for the management of postoperative pain associated with total-joint replacement surgery. Controlled studies, which define the risk of neuraxial bleeding associated with the use of an epidural catheter for postoperative analgesia with concomitant use of low-molecular-weight heparin (LMWH) are not available in the current literature, nevertheless the incidence of spinal hematoma after epidural blockade is documented to be less than 1:150000 and tends to increase with postoperative anticoagulation therapy ⁽⁷⁾. Spinal hematoma reveals itself with the signs of cord compression. The level of hematoma, the degree of cord compression and the spinal canal stenosis can be detected by magnetic resonance imaging. With proper treatment, 38% of patients who had spinal hematoma after epidural blockade achieved recovery with partial or complete neurological improvement are well documented ⁽²⁰⁾. In the case of spinal cord compression, laminectomy seems to be effective in reversing spinal ischemia if performed within the first eight hours ⁽²⁰⁾. We report a case of epidural hematoma formation after the removal of the lumbar epidural catheter in a patient who underwent total knee replacement surgery and commenced to receive low-molecular-weight heparin (LMWH) postoperatively for thromboembolic prophylaxis.

CASE REPORT

A 78-year old female patient with a body weight of 65 kg and height of 155 cm, suffering

from chronic bilateral knee pain was brought to our clinic, who was diagnosed as bilateral gonarthrosis had undergone right total knee arthroplasty under spinal and epidural anesthesia in 1999. The patient was hospitalized in the same orthopedic department for the left total knee arthroplasty. The medical history of the patient included regulated hypertension, hyperlipidemia and Raynaud's disease. Her preoperative laboratory results were within the normal range. Following evaluation of various anesthetic techniques in the light of their potential benefits and the risks, the patient consented to proceed with an epidural+spinal technique for anesthesia and postoperative analgesia which was applied for the previous arthroplasty procedure without encountering any particular problem. After premedication with Midazolam, the patient was brought to the operating room and was monitored. Under sterile conditions, in the lateral decubitus position, an 18G Touhy epidural needle (B/Braun Espocan, Germany) was placed by a CRNA with 10 years of experience without difficulty at L4-5. Another needle sent through epidural catheter needle to the spinal space and the spinal anesthesia was provided by administration of 0.5% bupivacaine into the spinal space. The procedure was accomplished without an apparent problem. After surgery, an epidural infusion of % 0.125 Bupivacaine + 2µg/ml Fentanyl was established in the recovery room, and the patient was transferred to the rounds in stable condition. Postoperative 48 hours were painless and isometric knee stretching exercises and continuous passive motion (CPM) exercises were introduced.

In the postoperative 24th hour, a low molecular weight heparin (LMWH) injection -

Enoxaparine 0.6 ml/60 mg 1*1– was made subcutaneously. Epidural catheter was removed in the 47th postoperative hour after routinely checked laboratory results were encountered.

Second dose of LMWH (Enoxaparine 0.6 ml/60 mg) was given 1^{1/2} hours after the catheter removal. In the third hour following the removal of the catheter, (50th postoperative hour) the patient started to complain about back pain, which was worsening by time. The examination of the patient by the orthopaedic surgeon revealed edema in the operated leg and peripheral cyanosis in the legs bilaterally. Cardiovascular surgery (CVS), anesthesiology, and neurology consultations were ordered by the orthopaedic surgeon because of the edema on the operated side and cyanosis of the toe phalanges, bilaterally. CVS consultation concluded with the suspicion of deep venous thrombosis and the consultant ordered to increase the doses of LMWH (Enoxaparine 0.6 ml/60 mg 2*1) and to commence 100mg acetylsalicylic acid.

On call anesthesiologist ordered an MRI and a neurology consultation because back pain might be related to the epidural catheter removal since it is one of the cardinal symptoms of spinal hematoma. The physical examination by the neurology consultant revealed normal muscular strength, absence of pathologic reflexes, normal sensation not necessitating an MRI.

As a result of these consultations the dose increment of the LMWH (enoxaparine 0.6 ml/60 mg 2*1) and 100mg acetylsalicylic acid treatment was started. After the therapy mentioned above commenced, the patient started to complain of her back pain

worsening; anesthesiology consultation was repeated in the postoperative 72nd hour.

In order to rule out the deep venous thrombosis (DVT) which was suspicious due to the peripheral cyanosis Doppler ultrasonography (USG) was performed at the 66th hour. Doppler USG eliminated the DVT diagnosis and DVT treatment was stopped afterwards. At the same time MRI for the lumbar region was performed. MRI revealed an epidural hematoma within the T12-L2 levels of the spinal canal with cord compression at the L1 level (Figures 1-2). Neurological examination at the 72nd hour revealed muscular strength of 2/5 on the previously operated leg (right) while the left side was considered to be of normal motor strength, absence of sensory deficit, bladder with a catheter, and normal functioning anal sphincter, absence of the pathologic reflexes and recommended follow up with physical examination with four hour intervals.



Figure 1. Coronal MRI section demonstrating the spinal canal compression at the L1 level.



Figure 2. Sagittal MRI section demonstrating the epidural hematoma within T12-L2 levels

In the 90th hour (45 hour after catheter withdrawal, or 6 hours after the loss of right ankle movement) the physical examination of the patient showed right ankle extension and flexion muscle strength 1/5, knee extension and flexion muscle strength 1/5, hip flexion muscle strength 1/5 with paraesthesia on right lower extremity up to the inguinal level. At the L1 level utilizing the posterior approach laminectomy and evacuation of the epidural hematoma was performed by orthopaedic surgeon (E.S.)

After laminectomy and hematoma drainage for the first two days the patient could not move her leg, but started plantar and dorsiflexion on the third and knee flexion on the sixth day postoperatively. She started

walking on the tenth day and was discharged from the hospital on the fifteenth day without any neurologic deficit.

DISCUSSION:

Spinal hematoma is a rare but an overwhelming complication of spinal or epidural anesthesia and analgesia. Neuroaxial anesthesia and analgesia are referred to be related with the superior analgesia and diminution of the surgical stress response which, in turn is associated with reduced thromboembolic complications approved to be aggravated by the stress response^(6-9,11). Deep venous thrombosis is shown to be reduced by neuroaxial anesthesia and analgesia but the frequency of thromboembolism is substantial and necessitates application of primary prophylaxis with low-molecular weight heparins or warfarin⁽⁶⁾. The management of pain is a significant concern in major orthopedic surgery. Pain relief is approved to be associated with the improved joint range of motion and facilitates the rehabilitation of the patients who had undergone total knee replacement⁽³⁾.

The actual incidence of neurologic complications of neuroaxial anesthesia and analgesia is unknown but the incidence was reported to be 1:150 000 after epidural anesthesia and 1: 220 000 after spinal anesthesia^(7,16). Owing to the prominent epidural venous plexus, spinal bleeding most commonly occurs within the epidural space. Epidural hematomas can occur spontaneously while the review of spontaneous epidural hematomas in the literature revealed an association with anticoagulant therapy with an odd's ratio of 20%⁽⁴⁾. Vandermeulen et al.⁽²⁰⁾ reported 61 cases of spinal hematoma associated with neuroaxial anesthesia in his review of the literature between

1906 and 1994. 32 of these 61 patients had an indwelling epidural catheter and 15 among those patients had suffered from the spinal hematoma immediately after the catheter removal. The risk factors identified to be associated with spinal hematoma formation include female gender, increased age, traumatic needle or catheter insertion, epidural (compared to spinal) technique, anticoagulation/coagulopathy⁽⁶⁻⁷⁾. Vandermeulen et al. reported that 53/61 cases had a history of either puncture difficulties or a coagulation disorder⁽²⁰⁾. In our case the patient had not experienced any puncture difficulties such as multiple attempts or bloody tap and her preoperative blood coagulation parameters were completely normal.

Both insertion and removal of the epidural catheter in the presence of low molecular weight heparin accused to be responsible for epidural hematoma formation⁽²²⁾. 30-60% of clinically important spinal hematomas occur after removal of the epidural catheter as is the situation in our case.

Two reviews published in 1992 and 1993 concluded that neurologic complications after spinal or epidural anesthesia in patients receiving low molecular weight heparin were extremely rare and the combination supported to seem safe^(1-2,8). Between May 1993 and December 1997, 25 cases of spinal hematoma after spinal or regional anesthesia in conjunction with the enoxaparin use, in patients who underwent total hip or total knee arthroplasty were reported which provided evidence for the incidence of spinal hematoma being greater than 1:150 000 for this patient group⁽¹³⁾. Enoxaparin is the first low molecular weight heparin approved by the Food and Drug Administration (FDA) in the United States and distributed for general use in May 1993⁽⁸⁾. In 1997, semination of the reports associating

epidural or spinal hematomas to epidural anesthesia and application of low molecular weight heparins, FDA and the German Society of Anesthesiology and Intensive Care Medicine reached a consensus that when low molecular weight heparin is used preoperatively, neuroaxial block should be delayed for 10 to 12 hours after the administration of last dose^(5,10,20). The first postoperative low molecular weight heparin dose should be administered no sooner than 6-8 hours postoperatively⁽⁷⁾. Between administration of low molecular weight heparin and removal of the epidural catheter the recommended time interval is 10-12 hours⁽¹⁰⁾. Following catheter removal, the low molecular weight heparin should be administered minimum two hours later⁽⁷⁾. According to the American Society of Regional Anesthesia and Pain Medicine (ASRA) guidelines, twice daily dosing and regardless of the anesthetic technique, administration of the LMWH after the 24th postoperative hour and removal of the epidural catheter 2 hours before the first LMWH dose was recommended⁽²⁰⁾. The pharmacodynamic properties of low molecular weight heparins include onset of its effect approximately 90 min after injection and a half life of more than 4 hours, with antithrombotic effects lasting approximately 12 hours⁽¹⁹⁾. In our patient, enoxaparin (0.6 ml 1*1) commenced at the postoperative 24th hour. She did not receive preoperative prophylactic low molecular weight heparin. Epidural catheter was removed at the 47th postoperative hour meanwhile no manipulation of the epidural catheter was present. Second dose of LMWH (Enoxaparin 0.6 ml) was given 1^{1/2} hours after the catheter removal which is at least 1/2 hour earlier than recommended dosing time and probably might be responsible for the development of the neurologic complication. Renal impairment also

results in accumulation of the drug but this is irrelevant with our case since she had normal renal function tests preoperatively and also postoperatively. Apart from the total daily dose of the low molecular weight heparin, the dosing regimen of the drug is also related with the epidural hematoma incidence⁽¹⁸⁾. The highest bleeding risk has been shown with 30 mg (0.3 ml) enoxaparin injected twice daily⁽¹⁸⁾. Our patient received 60 mg enoxaparin once a day for two days and on the second day postoperatively the dose of the enoxaparin was increased to 60 mg twice daily in combination with 100 mg acetylsalicylic acid as a result of the cardiovascular surgery consultations which was ordered with a suspicion deep venous thrombosis. Horlocker et al. reported that typical doses of acetylsalicylic acid do not cause an increased risk of spinal hematoma formation⁽⁹⁾. But the combination of acetylsalicylic acid and low molecular weight heparin might increase the risk of bleeding⁽¹⁷⁾. In 1997, a case report of epidural hematoma which developed soon after hospital discharge in an ambulatory surgical patient (underwent arthroscopic lateral meniscectomy) who received epidural analgesia concurrent with aspirin, NSAIDs and low molecular weight heparin was published. Up to our knowledge this is the unique report documenting formation of epidural hematoma in the presence of three anticoagulants⁽¹²⁾. Our patient received 100 mg acetylsalicylic acid and enoxaparin (0.6 ml 2*1) for once and soon after her complaints worsened and meanwhile venous Doppler USG of the lower extremities eliminated deep venous thrombosis which was actually a misdiagnosis.

Typical presentation of spinal epidural hematoma is with sharp back pain and associated motor and sensory deficit progressing to paraplegia and bowel/bladder

dysfunction^(7,14). Vandermeulen mentioned that in a patient with a spinal hematoma formation in 46% of the cases, the first symptom was muscle weakness which was followed by back pain (38%) and a sensory deficit (14%)⁽²⁰⁾. The first complaint of our case was a sharp back pain started 3 hours after the removal of the epidural catheter and by time the back pain worsened. Neurologic examination of the patient which was performed both by the orthopaedist and the neurology consultant did not reveal any loss of muscular strength or sensory deficit. There was an in situ foley catheter in the bladder and anal tonus was present.

Postoperative utilization of anticoagulants in the presence of an indwelling epidural catheter has a greater risk of producing a spinal epidural hematoma and such cases necessitates regular neurologic examination to detect signs of a developing hematoma⁽¹⁵⁾. The complaints of a patient with a developing hematoma might be misinterpreted as being due to the action of local anesthetic infusion. In case of a spinal hematoma, the delay in the diagnosis may result in poor neurologic outcome. Lumbar spinal MRI which was performed at the 66th postoperative hour revealed a spinal epidural hematoma extending from T12 to L2 with a spinal cord compression at the level of L1. Due to the severe back pain, the patient asked to lie in the lateral decubitus position on her right side and soon after the positional change at the 72nd hour, the neurologic examination is consistent with functional motor loss in her right leg (the previously operated leg). In the literature it is noted that active movement of these patients should be minimized in order to prevent the migration of the epidural catheter causing vessel damage⁽¹⁵⁾. But in our patient the neurologic deficit occurred following her positional change while lying down in the bed which was absolutely

25 hours after the catheter removal. By means of medline search we could not meet any case noting the aggravation of neurological findings due to the positional change of an immobilized patient.

The treatment of choice is laminectomy and surgical evacuation of the hematoma in case of an epidural hematoma associated with neurological deficit ⁽⁵⁾. Prompt establishment of the surgical treatment is strongly correlated with the neurologic outcome but it is also dependent on preoperative neurological function, hematoma size and location and the speed of hematoma formation (20). Spinal cord ischemia tended to be reversible in patients who underwent laminectomy within the first 8 hours after the onset of paraplegia ^(7,15). We performed L1 laminectomy and surgical evacuation of the hematoma 6 hours after the onset of paraplegia but 40 hours after the onset of back pain and 18 hours after the onset of paresis in her right lower extremity. As a result of their review of spinal hematoma cases, Horlecker and Wedel assessed that the median time interval between the initiation of low molecular weight heparin treatment and neurological dysfunction was 3 days and the median time to onset of symptoms and laminectomy was more than 24 hours ⁽⁹⁾. Among those patients less than one third of them achieved fair or good neurologic recovery. Our patient achieved full recovery; after the second postoperative day she succeeded in moving her extremity and on the sixth postoperative day regained full motor function and on the tenth day she started to walk. She was discharged from the hospital on the postoperative 15th day with a full neurologic recovery.

In summary, we report a case of an elderly female patient underwent left total knee arthroplasty under continuous epidural analgesia and spinal anesthesia, who had

received postoperative low molecular weight heparin therapy in conjunction with aspirin as a result misdiagnosis. She had suffered from spinal epidural hematoma developed soon after the catheter removal, extending between T11-L2 with a presenting symptom of sharp back pain. Administration of anticoagulant after the epidural catheter removal minimum 30 minutes earlier than suggested in ASRA guidelines may be responsible for the development of the spinal hematoma. Probably as a result of positional change of the patient in her bed functional motor deficit developed. On call conditions lead to an inadequate differential diagnosis in our patient who had a Raynaud's disease preoperatively and the patient had a treatment for DVT with a concomitant increase in the neurologic findings of the spinal hematoma after repeated doses of anticoagulation. Eventually, the patient regained her complete neurological function after a surgical decompression performed without wasting much time after the spinal hematoma diagnosis was accurate.

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