

# Rescue Lumbar Drain Placement in a Complex Type B Dissection Repair

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## Introduction

One of the strategies to reduce the risk of spinal cord ischemia (SCI) in patients undergoing thoracoabdominal aortic aneurysm repair (TAAA) is lumbar cerebrospinal fluid drainage. The concomitant use of anticoagulants increases the risk of epidural hematoma during neuraxial instrumentation. We present a case of a patient on clopidogrel therapy, who underwent endovascular stent placement that resulted in complete coverage of the aorta distal to the left subclavian artery. The patient developed symptomatic SCI post-operatively. Although lumbar drain placement was deferred initially, a rescue lumbar drain was placed with complete resolution of symptoms.

## Case Presentation

A 63 year old male with history of complex type B dissection involving the entire descending aorta, who presented with progressively worsening retrosternal chest pain. He was taken to the operating room urgently for an open debranching TAAA.

### Preoperative course

- ❖ Type B thoracoabdominal aortic aneurysm s/p overlapping thoracic stent graft in the descending aorta 3 months prior.
- ❖ Bifurcated infrarenal stent with left renal artery stent 1 month prior.
- ❖ Co-morbidities: Hypertension, chronic back pain.
- ❖ Medications: Clopidogrel (last dose 1 day prior to current encounter), aspirin, enalapril, HCTZ, metoprolol, norvasc, zocor, nexium, catapres.

### Intraoperative course

- ❖ General anesthesia along with invasive monitoring that included an arterial line, central venous catheter and TEE.
- ❖ Lumbar drain NOT placed due to anticoagulation with Clopidogrel.
- ❖ Debranching of the visceral vessels of the abdominal aorta with repair of type B aortic dissection using a thoracic endovascular repair, resulting in complete coverage of aorta with graft. (Figure 1)
- ❖ No re-implantation of segmental arteries performed.
- ❖ Mean arterial pressure within 20% of baseline throughout the case.
- ❖ Estimated blood loss of 2,500ml, for which patient received 11 units of PRBCs, 6 units of FFP, 1 unit of platelets and cryoprecipitate each.
- ❖ Transferred to the ICU intubated, on vasopressor infusion.

### Postoperative course

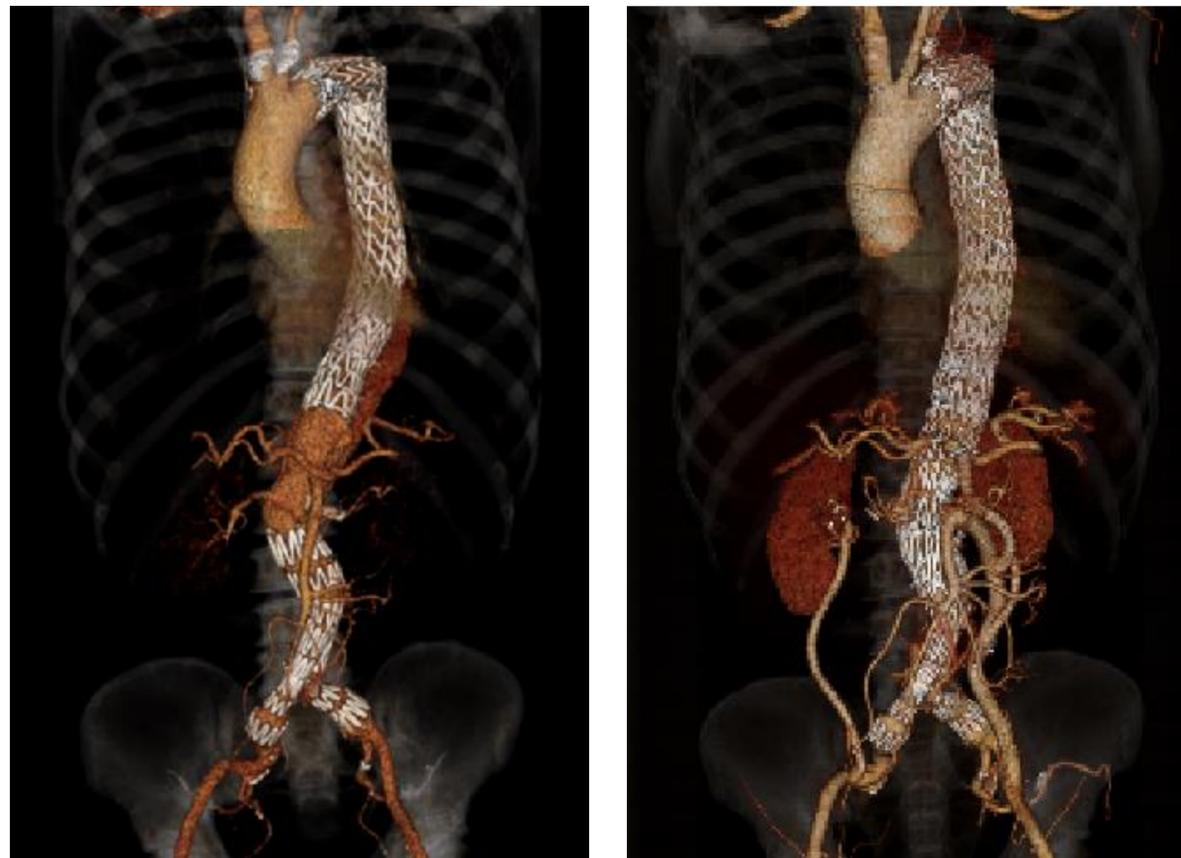
- ❖ Twelve hours post op, patient developed new onset lower extremity weakness.
- ❖ A lumbar drain was placed for rescue therapy.
- ❖ The patient's weakness improved over the course of 24 hours.
- ❖ Lumbar drain was discontinued on POD #4 with no residual weakness or complications.

**Table 1** Risk factors for paraplegia after TAAA repair

| OPEN TAAA REPAIR                                 | ENDOVASCULAR TAAA REPAIR                               |
|--|--|
| More extensive aneurysms (Crawford type I or II) | More extensive coverage of the thoracic aorta by graft |
| Emergency presentation                           | Occlusion of the left subclavian artery                |
| Prolonged aortic cross-clamp time                | Occlusion of hypogastric arteries                      |
| Extent of segmental arteries sacrificed          | Injury to the external iliac artery                    |
| Previous abdominal aortic aneurysm repair        | Previous abdominal aortic aneurysm repair              |
| Severe atherosclerotic disease                   | Severe atherosclerosis of the thoracic aorta           |
| Hypotension                                      | Hypotension  |
| Advanced age / Diabetes                          |  |

**Figure 1. 3-D reconstruction of CTA of chest, abdomen, and pelvis of patient.**

Figure on the left is prior to final TAAA repair, while figure on the right is 1 month post op. One can appreciate complete coverage of thoracic and abdominal aorta with debranching of vessels, and absence of blood flow through aneurysmal sac.



## Discussion

### Paraplegia after thoracoabdominal aortic aneurysm (TAAA) repair

- ❖ 5 – 43.8% after open TAAA repair.<sup>1</sup>
- ❖ 4 – 8 % after thoracic endovascular aortic repair (TEVAR).<sup>1</sup>
- ❖ Risk factors include emergency surgery, extent of aneurysm surgery, previous AAA surgery, intra and post-operative hypotension, left subclavian artery coverage and external iliac artery injury (Table 1).
- ❖ Interruption of blood flow through segmental arteries (SA) either secondary to surgical ligation during an open repair, or coverage with endovascular stent during endovascular repair results in SCI.
- ❖ The extent of SA sacrifice is a predictor of paraplegia risk. Patients with more than 13 SAs sacrificed have a higher rate of SCI (12.5%), as compared to those who have fewer than 8 SAs sacrificed (1.2%).<sup>2</sup>

### Role of lumbar CSF drainage

- ❖ Augment spinal cord perfusion pressure (SCPP) by decreasing CSF pressure since  
 $SCPP = MAP - CSF \text{ pressure or } CVP \text{ (whichever is greater)}$
- ❖ Randomized control trial (RCT) of 145 patients undergoing TAAA repair showed 80% reduction of incidence of postoperative neurologic deficits (13.0% vs 2.6%,  $P = 0.03$ ).<sup>3</sup>
- ❖ A pooling of 3 RCTs of 289 patients showed the number needed to treat of 9 for prevention of SCI (95% CI [5–50]; odds ratio 0.35, 95% CI [0.12–0.99]).<sup>4</sup>

### Role of Rescue lumbar drainage for delayed paraplegia

- ❖ CSF drainage institution after SCI development in patients that did not have lumbar drain placed intraoperatively has been shown to be effective in preventing paraplegia.
- ❖ Delayed paraplegia is believed to be secondary to cord edema, postoperative hypotension, thrombosis or hematoma formation.<sup>5</sup>

### Lumbar drain placement in the presence of anticoagulation

- ❖ ASRA recommends instrumentation of the neuraxis be avoided in patients with preexisting coagulopathy.<sup>6</sup>
- ❖ The actual risk of spinal hematoma with concomitant clopidogrel therapy is unknown.
- ❖ Risk benefit ratio of placement of lumbar drain and development of spinal hematoma has to be weighed on a case by case basis.
- ❖ Rescue lumbar drain placement in the presence of SCI, even with concomitant antithrombotic therapy is easier to justify.

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## **Persistent sciatic and saphenous neuropathy after a single shot nerve block**

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### Learning Objectives:

Upon completion of this learning activity, participants should be able to

1. Recognize the risk factors that can predispose to neuropathy following peripheral nerve blocks.
2. Comprehend the likely mechanisms that lead to neuropathy following peripheral nerve blocks.
3. Outline the appropriate follow-up, testing and treatment strategies to maximize outcome in a patient with neuropathy following peripheral nerve block.

A 32-year old male presented for peroneal tendon repair. The patient was otherwise healthy with no neurologic deficits on pre-block exam. He underwent popliteal and saphenous nerve blocks, under ultrasound guidance, with a mixture of 0.5% bupivacaine, dexamethasone and epinephrine. Block placement was unremarkable with no evidence of nerve swelling. The patient was noted to have prolonged sensory-motor weakness that prompted neurophysiologic testing which revealed axonal injury to tibial, common fibular and saphenous nerves. Although the etiology of post-block nerve injuries is hard to elucidate in most cases, the use of dexamethasone in this case could have been a contributing factor.

**Liver transplantation in a patient with moderate portopulmonary hypertension: when to proceed versus cancel.**

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Upon completion of this learning activity, participants should be able to:

1. Describe the pathophysiology of portopulmonary hypertension.
2. Predict the risks of liver transplantation in a patient with varying degrees of severity of portopulmonary hypertension
3. Identify pre-operative, intraoperative and postoperative treatment strategies for portopulmonary hypertension

A 56 year-old man with a history of hepatocellular carcinoma and mild portopulmonary hypertension (POPH) presented to the operating room for orthotopic liver transplantation. Prior to incision, a transesophageal echoradiogram was performed and a pulmonary artery catheter was placed. The mean PA pressures were measured in the low 40's, which is moderate to severe. The patient's PA pressures were successfully lowered using inhaled nitric oxide and intravenous epoprostenol. Postoperatively, the medications were continued and he had no major complications. The dilemma of proceeding versus cancelling an orthotopic liver transplant prior to incision in the setting of POPH will be discussed.

## INTRODUCTION

As ultrasound guided regional anesthesia becomes more widely used in clinical practice, we have come to appreciate the extent of anatomic variability, pathology and artifacts that may pose challenges to the clinician with respect to proper image interpretation. We present a case that illustrates these points and led to confusion in properly interpreting the location of the femoral nerve.

## CASE

A 57-year-old female was scheduled for surgical removal of an infected total knee prosthesis. Her past medical history was complicated by asthma, diabetes, hypertension, chronic renal insufficiency, morbid obesity and peripheral vascular insufficiency.

In order to provide post-operative analgesia in this patient, an ultrasound-guided continuous femoral nerve catheter placement was planned prior to surgery. During our pre-procedure ultrasound scan of the target area, we were initially unable to visualize the femoral nerve lateral to the patent vascular structures identified via color Doppler flow (Figure 1 & Figure 2).

Further examination revealed a femoral artery occluded by thrombus, with an unusual sonographic circumferential appearance, which was later determined to be an unknown previously placed metallic stent (arrow Figure 2). Once the occluded femoral artery was correctly identified, the location of the femoral nerve became apparent, and was confirmed by its relationship to the iliopsoas muscle and fascia iliaca. Under ultrasound guidance, a 17 gauge Tuohy needle was advanced adjacent to the hyperechoic structure identified as the femoral nerve, and a perineural administration of 20 mL of 0.5% ropivacaine was injected, followed by insertion of a catheter. The patient developed a complete motor and sensory block in the femoral nerve distribution, and the subsequent spinal and surgery proceeded uneventfully.

## PICTURES

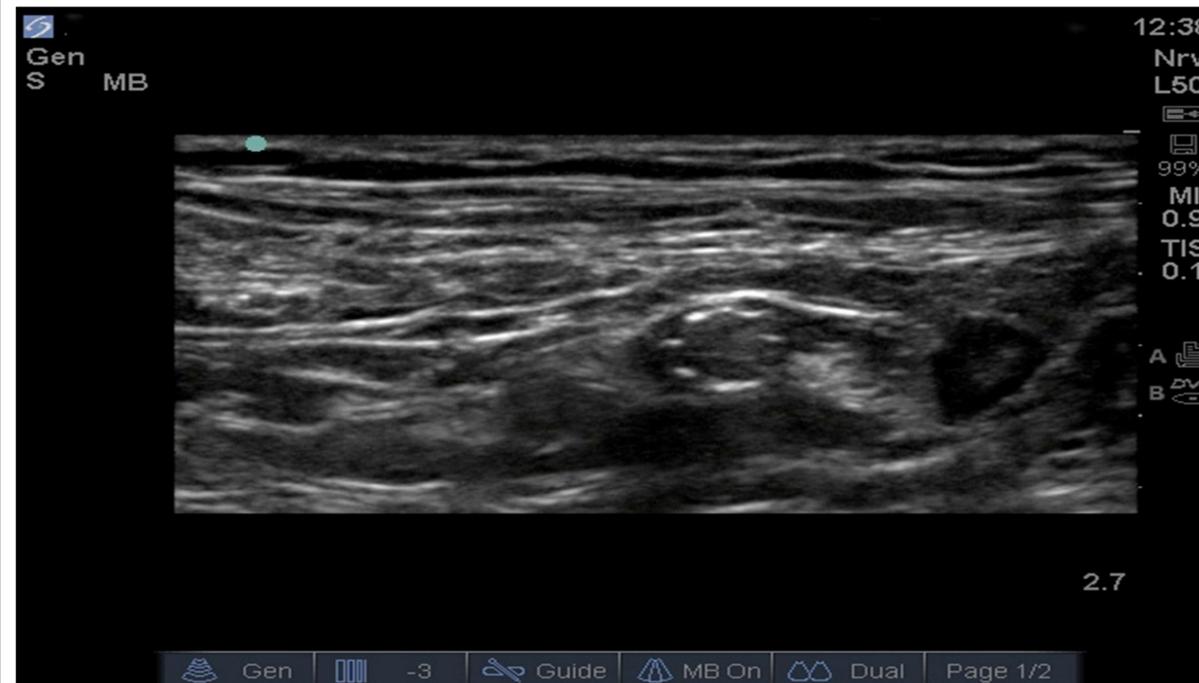


Figure 1:

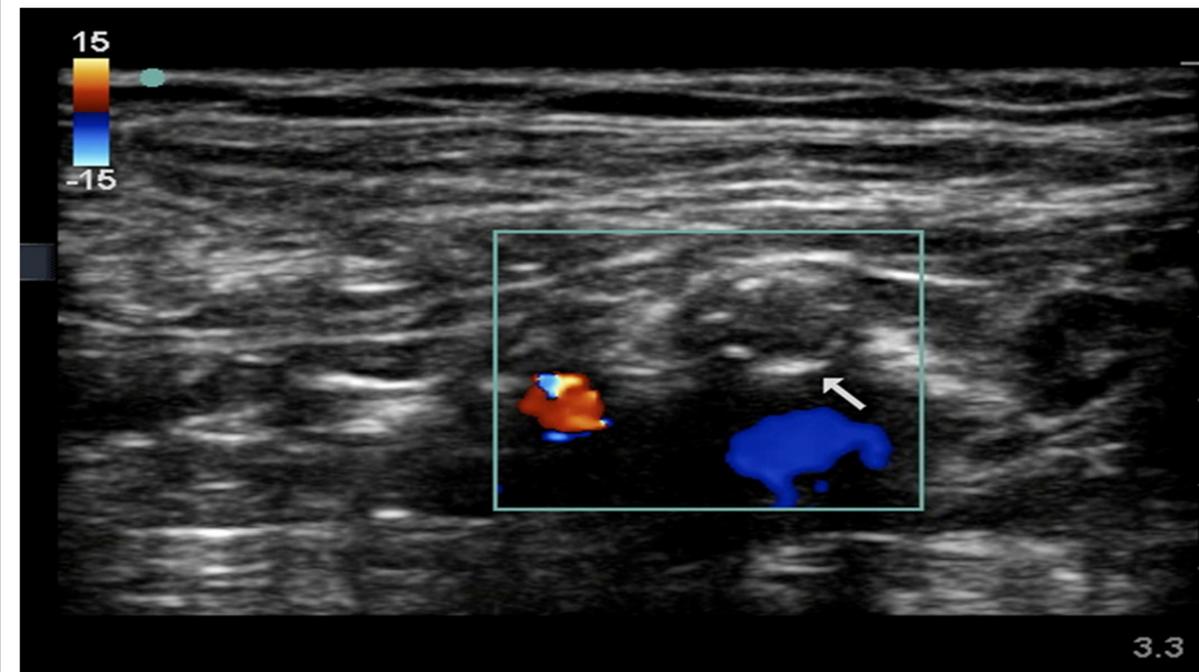


Figure 2:

## DISCUSSION

The importance of a pre-procedural scan has been suggested by other authors<sup>1,2,3</sup> in the recent literature, as an opportunity to identify unrecognized pathology in the area of the intended peripheral nerve block. In our case, this patient's pathology interfered with our initial attempt to correctly identify the femoral nerve using ultrasound guidance, and highlights the importance of appreciating the anatomic relationship of the femoral nerve to the iliopsoas muscle, rather than focusing primarily on what appears to be the femoral artery. Pitfall errors where anatomical structures are misidentified may be more common in patients with underlying vascular disorders, due to thrombotic lesions in vessels that completely occlude flow and prevent positive identification through the use of color flow Doppler imaging.

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# Atypical Presentation of Epidural Hematoma After Attempted Thoracic Epidural Placement

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## INTRODUCTION

The reported maximum risk of epidural hematoma after thoracic epidural placement is estimated at 0.07% and the frequency is reported in some studies to be 1 out of 150,000 to 190,000 procedures. Most present clinically with pain at the site, with progressive neurological symptoms such as weakness and paresthesias. In most cases, risk factors such as coagulopathy or anticoagulation treatment were present. This case presents a patient that was found to have an extensive epidural hematoma after attempted thoracic epidural placement.

## CASE REPORT

A 58 year old female with rectal cancer, hypertension, hypothyroidism, and ulcerative colitis was scheduled for low anterior resection, loop ileostomy, and possible intraoperative radiation therapy. A thoracic epidural was requested by the surgical team for post-operative pain control. The patient was of normal body habitus and had no history of back pain or back problems such as degenerative disease of thoracic spine, scoliosis, and no history of spine instrumentation or trauma. The patient was not on any anticoagulants, NSAIDs or herbal supplements known to increase risk of bleeding.

The initial epidural attempt was performed with the patient in a sitting position via midline approach at the T10 intervertebral level, as identified by using the inferior angle of the scapula as a landmark corresponding to T7 vertebral level (Fig 1). Midline approach was aborted due to inability to access the epidural space secondary ossifications. Subsequently a right paramedian approach was utilized. After proper skin anesthetic a 17 gauge Tuohy epidural needle was inserted 1.5-2 cm lateral from midline and advanced until the lamina was contacted at the depth of 4 cm. The needle was then redirected medially and cephalad and walked off the lamina.

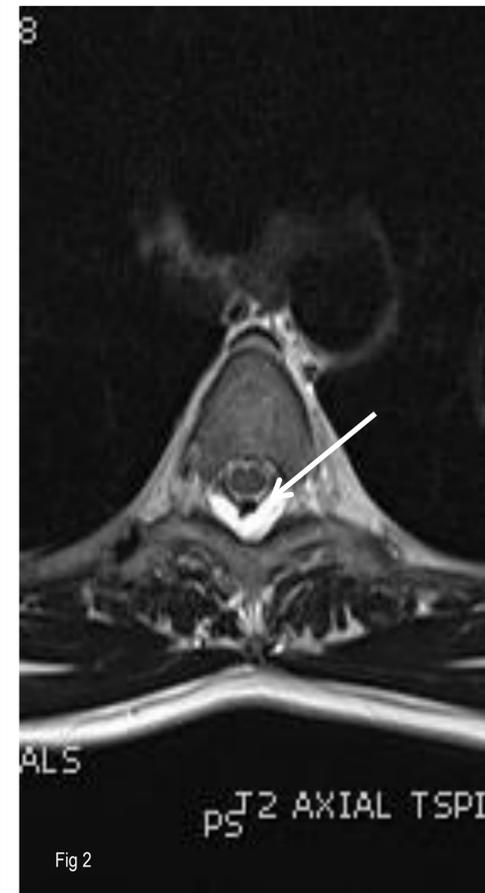
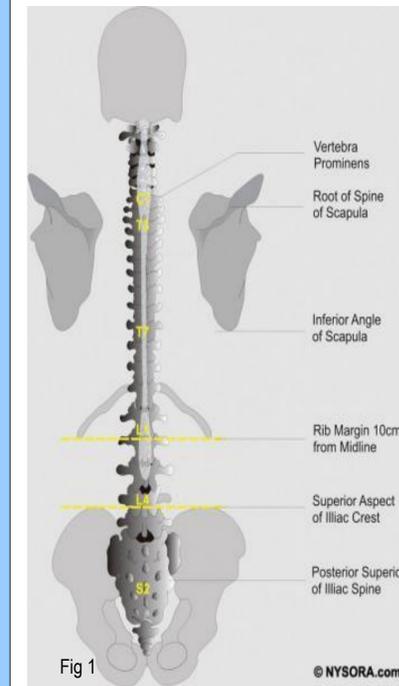


Fig 2

## Thoracic spine MRI with evidence of acute heme in the epidural space

Neurologic examination was performed immediately after the incident. The patient was found to have profound RLE weakness in L4 and L5 dermatomal distribution as evidenced by weak 1st toe dorsiflexion and weak knee extension. The patient also had sensory deficits to touch and pin-prick in a similar distribution and diffusely brisk DTRs. Within minutes the patient felt her pain and heaviness improve substantially and she regained most of her motor function and was able to ambulate with assistance.

At the time of evaluation by neurosurgery, approximately 45 minutes following the procedure, the patient was noted to have an intact neurologic exam. Full spine contrast MRI was obtained within 1 hour of the occurrence. The MRI showed evidence of extensive acute epidural hematoma extending from T1 to the conus and no spinal cord injury (fig 2 and fig 3). The patient remained hospitalized for a period of 2 days and discharged home with only residual groin dysesthesias in approximately L2 distribution. At 1 month follow up the patient reported complete resolution of symptoms and no motor or sensory deficits.



Fig 3

## DISCUSSION

Most if not all reported cases of epidural hematomas present with symptoms of severe acute back pain associated with motor and/or sensory deficits. Muscle weakness and back pain are the most frequent symptoms of epidural hematoma, observed in 46% and 38% of patients, respectively, and they develop slowly, with severity usually increasing with time.

Hematoma presentation in our case was highly unusual, as the patient had no back pain, her symptoms were extremely acute and improved within minutes and resolved within hours. This raises the question whether or not the patient's symptomatology was in fact caused by the mass effect of the epidural hematoma on the spinal cord, or was it a result of a direct contact of the needle with the neural structures. There is no question that the hematoma was hyper-acute in nature, as it appeared as an iso-intense and homogenous signal on the contrast spine MRI, suggesting less than 4 hrs of heme presence in the spinal canal.

Beyond the unusual presentation, one may also wonder about the rapid and nearly complete resolution of the patient's symptoms immediately after the occurrence, which further supports the notion that the symptoms were not related to the heme mass, but rather to mechanical intrusion of the Tuohy needle on the spinal cord. The residual symptoms of dysesthesias that persisted for a few weeks after might have been caused by minor spinal cord injury. The cause of the hematoma however, still remains unclear, as the CSF flow obtained from the inadvertent dural puncture was heme free. There was no blood aspirated at any time during the procedure. It is also unusual that the subarachnoid space was entered without evidence of LOR, which might have been influenced by relative inexperience of the performing physician, or due to structural epidural space anomalies such as lack of fusion of the ligamentum flavum.

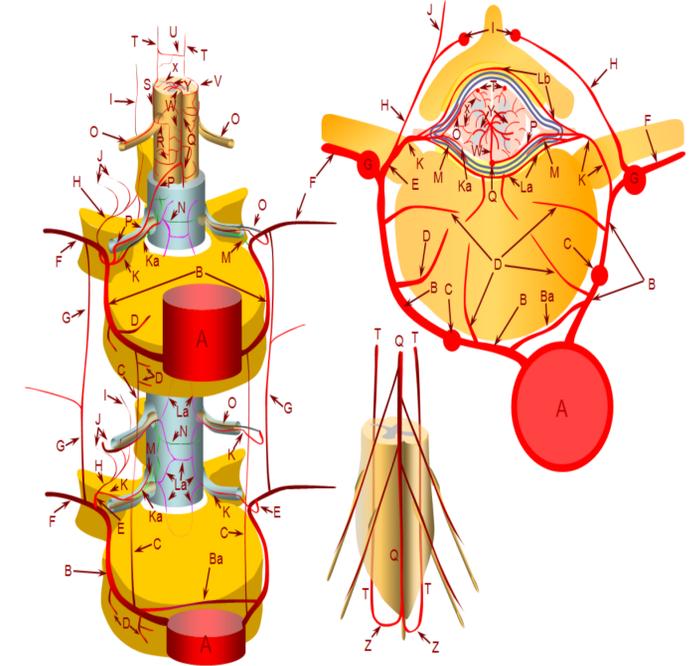


Fig4

It has been postulated that some epidural hematomas may arise from abnormal vascular malformations residing in the epidural space. Other theories suggest epidural hematoma as a result of arterial injury. Taken under consideration the rapid and extensive heme accumulation in this case, we propose that the epidural formation resulted from injury to either dorsal epidural arcade or nerve root sleeve dural branch of the ventral division dorsal spinal artery (fig 4).

The mainstay of treatment of epidural hematoma remains emergent surgical decompression and evacuation via laminectomy. There are only a few reports of successful conservative treatment for epidural hematoma, but none for a hematoma as extensive as demonstrated in this case. Our patient was extremely fortunate to recover spontaneously without sequella.

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6. neuroangio.org. This website is developed and maintained by Dr. Maksim Shapiro of the New York University Langone Medical Center. Copyright 2012

The needle was then advanced slowly in 1 to 2 mm increments using loss of resistance to saline with air bubble technique. At the depth of 4.5 cm the patient complained of sensation in her perineal area which was attributed to discomfort related to rectal cancer. Upon further needle advancement of 1 mm the patient complained of tingling and heaviness in her right leg and groin. After negative aspiration the stilet was inserted to ensure needle patency and upon stilet removal CSF flow was noted with no evidence of heme. The needle was then removed.