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# Spontaneous spinal epidural hematoma and spinal cord infarction following orthotopic liver transplantation: Case report and review of the literature

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## Abstract

**Background:** Spinal epidural hematomas are rare conditions. Although the exact cause remains unknown in up to 40% of cases, anticoagulation therapy, neoplasm, thrombolytic therapy, internal jugular vein thrombosis, and prolonged Valsalva maneuvers associated with pregnancy may be contributing factors. The source of bleeding appears to be the dorsal internal vertebral venous plexus (IVVP).

**Case Description:** A 65-year-old female patient with hepatitis C-related cirrhosis underwent orthotopic liver transplantation (OLT). The patient developed SSEH due to congestion of the IVVP in the peri-transplant period. Concurrent spinal cord infarction occurred, likely secondary to hypoperfusion during a cardiac arrest.

**Conclusion:** This case study should increase awareness of SSEH as a complication of OLT.

**Key Words:** Complications of liver transplantation, coagulopathy, internal vertebral venous plexus, orthotopic liver transplantation, spontaneous spinal epidural hematoma, spinal cord infarction

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## INTRODUCTION

The etiology of spontaneous spinal epidural hematoma (SSEH), a rare condition, remains unclear in 40% of cases.[5] Known causes include vascular malformation, anticoagulation therapy, neoplasm, following thrombolytic therapy, in patients with internal jugular vein thombosis, in pregnancy, and in prolonged Valsalva maneuver.[3,4,8,12] Congestion of the valve less posterior internal vertebral venous plexus (IVVP) is thought to account for the development of SSEHs dorsal to the spinal cord.[6] The clinical presentation typically includes the acute onset of neck or back pain followed by a rapid, progressive neurological deficit that may progress to spinal cord infarction.

Here we present a case of SSEH with concurrent spinal cord infarction in a patient who underwent orthotopic liver transplantation (OLT) for hepatitis C-related cirrhosis and hepatocellular carcinoma (HCC).

## CASE REPORT

A 65-year-old female with hepatitis C-related cirrhosis and HCC presented for OLT. The patient previously

underwent treatment for HCC with multiple transarterial chemoembolizations, radiofrequency ablation, and laparoscopic hepatic wedge resection for masses in the right lobe of the liver. Her past history was significant for hypertension and uterine cancer requiring total abdominal hysterectomy with bilateral salpingo-oopherectomy. Prior to transplantation, the patient was neurologically intact.

When a suitable donor organ became available, the patient was taken to the operating room for OLT using conventional techniques and veno-venous bypass. Surgery was complicated, however, by donor liver edema after reperfusion. Secondary to edema and with inability to obtain adequate hemostasis, the abdomen was packed and temporarily closed. Posttransplant Doppler ultrasound was performed and ruled out inferior vena cava (IVC) outflow obstruction. The patient was resuscitated in the intensive care unit and then taken back to the operating room the following day. Approximately 1 L of hematoma was evacuated from the abdomen after which careful inspection showed excellent hemostasis. The abdomen was then closed in standard fashion. The patient was extubated. She did have a fluctuating coagulopathy and thrombocytopenia (INR 1.7 and platelets 21,000 on posttransplant day one); this is not abnormal for patients postliver transplantation.

### Cardiac arrest

On posttransplant day 5 she developed respiratory distress and subsequently suffered a cardiac arrest with pulseless electrical activity. Cardiopulmonary resuscitation (CPR) was administered with eventual conversion to ventricular fibrillation, which was then treated with electrical defibrillation using 300 J. Following the cardiac arrest, the patient required reintubation and suffered from aspiration pneumonitis. Hypotension required the administration of vasopressive medications. Left lower extremity edema prompted a Doppler ultrasound that showed an occlusive deep venous thrombosis (DVT). This warranted the placement of a retrievable IVC filter, during which a nonocclusive thrombus in the infrarenal IVC was noted.

### Paraplegic deficit

When the patient's mental status improved on posttransplant day 10, it became clear that she was plegic in both lower extremities. Magnetic resonance imaging (MRI) of the cervical, thoracic, and lumbar spine [Figure 1] demonstrated epidural hematoma located dorsally at the foramen magnum, and both ventrally and dorsally from C7 to T6. The hematoma was hyperintense on both T1- and T2-weighted sequences, consistent with extracellular methemoglobin (late subacute hemorrhage, >7 days). In the lower thoracic spine there was marked spinal cord edema consistent with infarction characterized by an increased T2 signal from T8-T12.

### Post-SSEH/infarction course

Due to the age of the hematoma, the plegic deficit and presence of spinal cord infarction, and her life-threatening

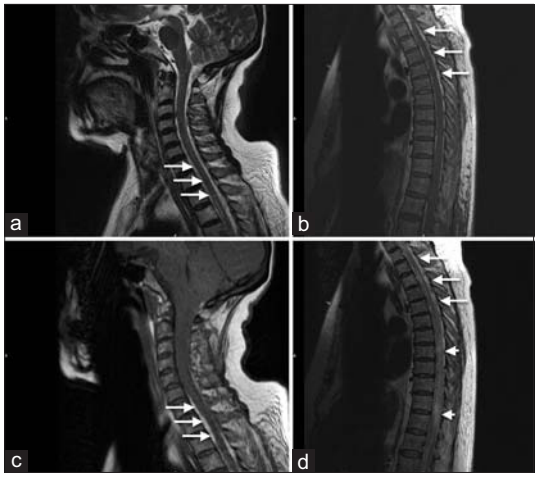


**Figure 1: (a) Cervical spine T1-weighted MRI, (b) cervical spine T2-weighted MRI, (c) thoracic spine T1-weighted MRI, (d) thoracic spine T2-weighted MRI. Long arrows = epidural hematoma, short arrows = spinal cord infarction**

continued coagulopathy, the patient was not considered a candidate for surgery (e.g., decompressive laminectomy for evacuation of the hematoma). She did, however, receive an 8-day course of intravenous methylprednisolone as part of the routine posttransplant immunosuppresion. Although the patient slowly recovered (tracheostomy for prolonged ventilator-dependent respiratory failure with subsequent weaning from the ventilator), she remained plegic in both lower extremities 6 months posttransplantation.

## DISCUSSION

In this case, the most likely etiology of the postoperative SSEH and spinal cord infarction would be attributed to bleeding from an engorged spinal epidural venous plexus exacerbated by thrombocytopenia (platelets 21,000), coagulopathy (INR 1.7), and the cardiac arrest/hypotension with resultant hypoperfusion of the lower thoracic spinal cord.

### Uncommon SSEH

Spinal cord infarction is an uncommon occurrence with SSEH. Park *et al.* described a case of SSEH resulting in spinal cord compression and thrombosis of the anterior spinal artery with subsequent infarction; decompressive laminectomy did not reverse the plegic deficit attributed to spinal cord infarction.[11] In a review of spinal cord infarction at a single institution by Cheshire *et al.*, 2 of 44 patients had epidural hematomas.[1] Both patient's developed epidural hematomata during anticoagulation therapy. One patient had improvement in both motor and sensory function and the other patient was unimproved upon follow up.

### Etiology of SSEH

SSEH is thought to occur as a result of rupture in the dorsal part of the IVVP, a valve less anastomotic system,

wherein blood can flow in either direction depending on changes in the intrathoracic and intraabdominal pressure.[6] Patients with liver disorders (e.g., Budd-Chiari syndrome or HCC) may utilize the VVS as collateral pathway for venous return in cases where there is obstruction of the IVC.[2] However, there are no reports in the literature of SSEH occurring in this setting. While our patient did have HCC, there was no evidence of obstruction of the IVC on preoperative imaging. Rather, the SSEH was most likely a result of compression of the IVC, perhaps secondary to abdominal packing, in the immediate postoperative setting with resulting engorgement of the VVS and bleeding secondary to thrombocytopenia and coagulopathy.

### Recommended management of SSEH

Most authors advocate rapid decompression of spinal epidural hematomas.[13] Lawton *et al.* demonstrated in a study of all patients with spinal epidural hematoma (traumatic, spontaneous, and iatrogenic) that outcome correlated inversely with the time interval between symptom onset, duration of maximal deficit, and surgery.[9] When Groen and Alphen analyzed 330 patients with SSEH from the reported literature and found that in patients with complete sensorimotor deficits for 36 hours of less, or for those with incomplete deficit within 48 hours or less, surgical decompression afforded significant improvement in outcome.[7] Nonoperative management of SSEH appears to only be an option if the patient's neurological deficits spontaneously resolve within 12 hours after onset of symptoms.[10] In this case, the patient's plegic deficit and complete cord infarction had likely been present for over 7 days.

## SUMMARY

SSEH and spinal cord infarction may result in paraplegic deficits for patients undergoing OLT. Increased awareness of the potential for major neurologic complication following OLT should prompt more vigilant and repeated postoperative neurological assessment.

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