Cervicothoracic Spinal Epidural Hematoma after Anterior Cervical Spinal Surgery

Dong Yeob Lee, M.D., Sang-Ho Lee, M.D., Ph.D.
Department of Neurosurgery, Woordul Spine Hospital, Seoul, Korea

The purpose of this case report is to describe a rare case of a cervicothoracic spinal epidural hematoma (SEH) after anterior cervical spine surgery. A 60-year-old man complained of severe neck and arm pain 4 hours after anterior cervical discectomy and fusion at the C5-6 level. Magnetic resonance imaging revealed a postoperative SEH extending from C1 to T4. Direct hemostasis and drainage of loculated hematoma at the C5-6 level completely improved the patient’s condition. When a patient complains of severe neck and/or arm pain after anterior cervical spinal surgery, though rare, the possibility of a postoperative SEH extending to non-decompressed, adjacent levels should be considered as with our case.

KEY WORDS: Cervical spine · Complication · Hematoma.

INTRODUCTION

A postoperative spinal epidural hematoma (SEH) is one of the classic complications of anterior cervical spine surgery. Although rare, rapid neurological deterioration caused by a postoperative SEH during the immediate postoperative period may result in profound morbidity, which necessitates emergent revision surgery for hematoma evacuation. A postoperative SEH after anterior cervical spine surgery is mostly localized to the surgical site and thus, a second-look surgery is sometimes performed without any complementary radiological examinations when the patient shows rapid progression of neurological deterioration.

A postoperative SEH extending to non-decompressed, adjacent levels is not an uncommon complication after lumbar spinal surgery. However, it has been rarely reported after anterior cervical spinal surgery. In the present study, we report a case of a postoperative SEH extending from C1 to T4 after anterior cervical spinal surgery at the C5-6 level.

CASE REPORT

A 60-year-old male presented with a 1-month history of severe pain in his right arm, which did not respond to conservative treatment. Physical examination demonstrated no apparent abnormal findings. He had a medical history of hypertension, which was well controlled with medication. The patient did not take antithrombotic agent such as aspirin.

Laboratory examinations did not show any evidence of a coagulopathy. Cervical T2-weighted magnetic resonance imaging (MRI) showed foraminal stenosis due to a herniated disc at the C5-6 level on the right side. He underwent anterior cervical discectomy and fusion (ACDF) at the C5-6 level. During the surgery, active bleeding from an epidural vein was noticed at the C5-6 foramen, which was controlled with hemostatic materials. Anterior cervical fusion was performed with a polyetheretherketone cage filled with cancellous bone graft. The operating time was 120 minutes and blood loss was 200 mL.

When the patient was awake and extubated in the operating room, he coughed several times and approximately 100 mL of blood was suddenly drained. After that event, he was stable in the post-anesthesia care unit with a spontaneous decrease in blood drainage. However, 4 hours after the surgery, he complained of severe pain in his neck and right arm without any motor weakness. Postoperative MRI revealed a
postoperative SEH extending from C1 to T4 with compression of the spinal cord (Fig. 1). The patient complained of severe pain only which seemed to be related to the loculated SEH at the C5-6 level. Therefore, we planned the staged operation for the patient; firstly, anterior revision surgery at the C5-6 level to control the bleeding focus and to drain loculated SEH and secondly, posterior decompressive laminoplasty, if the patient’s symptom did not improve after the first revision surgery. With the removal of the postoperative SEH at the C5-6 level, active bleeding from an epidural vein was noticed at the C5-6 foramen, which was controlled with direct coagulation by bipolar cautery. After anterior revision surgery, the patient completely improved without any neurological sequelae.

**DISCUSSION**

The incidence of symptomatic postoperative SEH is reported between 0.1 percent and 0.2 percent in the literature\(^2\,^3\,^9\). The incidence of postoperative SEH is greater than reported including asymptomatic SEH, between 33 percent and 100 percent in patients who undergo lumbar spinal surgeries\(^2\,^3\,^9\). Extension of postoperative SEH to non-decompressed, adjacent levels is not an uncommon phenomenon in the lumbar spine\(^9\). Sokolowski et al. noted that postoperative SEH extended cephalad-caudally 36 percent of the patients beyond the mean of 1.4 operative levels\(^9\). All of those patients were asymptomatic.

A postoperative SEH extending to non-decompressed, adjacent levels has been seldom reported after cervical spine surgery. Hans et al.\(^2\) performed a second-look surgery via the anterior route under the suspicion of a loculated postoperative SEH for a patient presenting with respiratory distress and flaccid tetraplegia after ACDF at the C6-7 level. However, the patient’s condition did not improve after the revision surgery. Then they performed follow-up MRI, which revealed a postoperative SEH extending from C3 to T3. They performed a posterior cervical laminectomy with evacuation of the hematomata thereafter and the patient completely recovered after the posterior decompressive surgery. In the present study, the patient with postoperative SEH complained of severe pain only without any neurological deterioration. Based on the clinical symptom of the patient, we considered the loculated SEH at the C5-6 level as main lesion, though follow-up MRI showed significant compression of cervical spinal cord due to cervicothoracic SEH. The patient’s symptoms totally relieved after anterior removal of loculated SEH at the C5-6 level without removing the whole cervicothoracic SEH. Ikuta et al.\(^9\) reported a 33 percent overall incidence of postoperative SEH after microendoscopic posterior decompression for lumbar stenois, and only three developed symptoms of radicular pain. MRI demonstrated spontaneous regression of the SEH in all patients at 3 months after surgery\(^5\). However, they observed less expansion of the dural sac after 1 year despite sufficient widening of the osseous spinal canal in patients with postoperative SEH. Postoperative SEH also caused a delay in the patient’s recovery and led to a poor clinical improvement. They assumed that transient compression of the nerve roots and poor expansion of the dural sac might prevent the initial recovery of the nerve damage; fibrosis induced by SEH permanently prevents the expansion of the dural sac and might cause nerve root irritation. Therefore, it was suggested that prevention of postoperative SEH might be required to prevent not only neurological deterioration but also a delay in the patient’s recovery\(^9\). In the present case, remnant SEH at non-decompressed, adjacent levels would have resolved spontaneously. However, further follow-up evaluation seems necessary since postoperative SEH in the cervicothoracic spine might cause a delay in patient’s recovery.

An age of more than 60 years old, multi-level procedure, and/or preoperative coagulopathy have been suggested as risk factors for postoperative SEH\(^5\,\,^9\). One important factor for a postoperative SEH is the completeness of the hemostasis during the initial surgery\(^2\,^3\,^9\). Bleeding form the fusion bed might be one cause of postoperative SEH after ACDF. The authors try not to prepare the fusion bed vigorously, because it can cause troublesome bleeding and furthermore delayed subsidence of cage or graft. If there is significant bleeding from the fusion bed, the authors usually control bleeding with a small amount of hemostatic material such as, bone
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wax. Another important cause of postoperative SEH is bleeding from the epidural vein at the foramen. When it is not feasible to coagulate epidural vein directly, the authors use commercially available hemostatic products, such as thrombin-soaked gelfoam, surgicel, and/or avitene. Though effective in control of epidural bleeding, these products can embolize, induce a localized inflammatory response, be easily washed away, and result in rebleeding. In the present case, hemostasis was considered adequate during the initial surgery since there was no evidence of active bleeding before closure. Rather, coughing during wake-up, which is associated with sudden elevation of the venous pressure, probably caused rebleeding from the epidural vein at the foramen. Straining, sneezing, and lifting have been previously suggested as probable predisposing factors for a spontaneous spinal epidural hematoma, all of which are also related to a sudden elevation of the venous pressure. Therefore, it may be helpful to induce elevation of the venous pressure using a Valsalva maneuver before closure. When rebleeding happens with Valsalva maneuver, additional hemostasis of epidural vein is necessary before closure. Care also should be taken to prevent severe coughing during wake-up from general anesthesia, since it can cause rebleeding of epidural vein as with our case.

The pathogenesis of a postoperative SEH extending to non-decompressed, adjacent levels still remains unclear. MRI is the most effective diagnostic tool for detecting a postoperative SEH extending to non-decompressed, adjacent levels. Second-look surgery without any complimentary radiological examination may result in misdiagnosis and inadequate treatment.

CONCLUSION

When a patient complains of severe neck and/or arm pain after anterior cervical spinal surgery, though rare, the possibility of a postoperative SEH extending to non-decompressed, adjacent levels should be considered. In the present case, removal of loculated hematoma at the index level was sufficient to relieve patient’s symptoms without removing the whole cervicothoracic SEH.

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References