**Case
Report**

Spinal Epidural Hematoma during Anticoagulant Therapy for Pulmonary Embolism: Postoperative Complications in a Patient with Lung Cancer

Yoshihisa Kobayashi, MD,¹ Junya Nakada, MD,² Hiroaki Kuroda, MD,¹
Noriaki Sakakura, MD,¹ Noriyasu Usami, MD,^{1,3} and Yukinori Sakao, MD¹

Spinal epidural hematoma (SEH) is rare but causes neurological disorders. Rapid diagnosis and treatment maximize neurological recovery. We present the case of SEH after lung cancer surgery under epidural and general anesthesia. A 64-year-old man underwent right upper lobectomy. Pulmonary embolism occurred on postoperative day 2. Anticoagulant therapy with fondaparinux and warfarin was started 2 hours after epidural catheter removal and he gradually recovered. On postoperative day 13, the level of prothrombin time-international normalized ratio reached 1.47 and fondaparinux administration was stopped. The next day, he developed back pain and paraplegia, and magnetic resonance imaging revealed a mass between Th4 and Th7 compressing the spinal cord. Emergency decompression laminectomy and hematoma evacuation were performed. After 2.5 months of rehabilitation, he regained almost all motor function and sensation. Late after epidural anesthesia, attention should be paid to possible SEH even though appropriate anticoagulant therapy had been initiated after epidural catheter removal.

Keywords: lung cancer surgery, pulmonary embolism, spinal paraplegia, anticoagulant therapy

Introduction

Spinal epidural hematoma (SEH) is extremely rare but can cause severe neurological dysfunction and even permanent neurological deficits. Rapid diagnosis and emergency surgical decompression maximize neurological

recovery; the complete recovery rate of patients treated within 12 hours and later than 12 hours were 65.9% and 29.1%, respectively.¹⁾ Epidural anesthesia, which is widely employed during lung surgeries, is one of the etiological factors for SEH. Here, we present the case of a patient who developed SEH after lung cancer surgery under general and epidural anesthesia.

¹Department of Thoracic Surgery, Aichi Cancer Center Hospital, Nagoya, Aichi, Japan

²Department of Anesthesiology, Aichi Cancer Center Hospital, Nagoya, Aichi, Japan

³Department of Thoracic Surgery, Nagoya University Graduate School of Medicine, Nagoya, Aichi, Japan

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Corresponding author: Yoshihisa Kobayashi, MD. Department of Thoracic Surgery, Aichi Cancer Center Hospital, 1-1 Kanokoden, Chikusa-ku, Nagoya, Aichi 464-8681, Japan

Email: ykobayasi@aichi-cc.jp

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Case Report

A 64-year-old man presented with dyspnea on exertion. Computed tomography (CT) showed a pulmonary nodule in the right upper lobe. Transbronchial lung biopsy revealed adenocarcinoma. He had no family history of coagulation disorder. His body mass index was 26.3 kg/m². Physical examination showed no varicose veins of the legs, and venous ultrasonography of the legs revealed no

Fig. 1 The treatment for pulmonary embolism and the clinical course. Anticoagulant therapy consisting of fondaparinux and warfarin was initiated on postoperative day (POD) 2. The oxygen demands were gradually decreased. The amount of warfarin was gradually increased according to the level of prothrombin time-international normalized ratio (PT-INR). On POD 13, the PT-INR reached 1.47 and fondaparinux administration was stopped.

clots. The preoperative D-dimer level was 0.7 $\mu\text{g/ml}$. Intermittent pneumatic compression (IPC) was selected as a prophylaxis against deep vein thrombosis (DVT).

An epidural catheter was inserted with an 18-gauge needle at the level of Th5–Th6 over 11 minutes. Under epidural and general anesthesia, right upper lobectomy with mediastinal lymph node dissection was performed. The operative duration was 3 hours. The blood loss was 20g. The histologic diagnosis was stage IA adenocarcinoma.

He walked around the hospital ward on postoperative day (POD) 1, and body weight loss was not observed. On POD 2, after walking, he developed oxygen desaturation and required 10 L/min of oxygen. Contrast-enhanced CT revealed bilateral pulmonary embolism (PE). The epidural catheter was immediately removed, and after 2 hours, anticoagulant therapy with fondaparinux and warfarin was initiated according to the recommendation of the cardiologist on POD 2. The treatment for PE and the clinical course were summarized in **Fig. 1**. Warfarin therapy was started at only 1 mg/day because it was the early postoperative period and the chest drainage tube remained in place. Fondaparinux (7.5 mg) was subcutaneously injected once a day. We decided not to perform tissue plasminogen activator treatment because hypotension had not developed and it was still the early postoperative period. The oxygen demands were gradually decreased to 7 L/min

Fig. 2 Axial contrast-enhanced computed tomography showing the epidural mass located in the right-posterior aspect of the spinal canal. This finding was not detected timely but was retrospectively revealed.

on POD 3, 3 L/min on POD 4, 1 L/min on POD 5, and no supplementary oxygen was needed after POD 9. The chest tube was removed without bleeding on POD 3. The target level of prothrombin time-international normalized ratio (PT-INR) was defined at 1.5, and the amount of warfarin was gradually increased to 2 mg on POD 6 and 3 mg on POD 9 per the suggestion of the cardiologist. On POD 13, the PT-INR reached 1.47 and fondaparinux administration was stopped.

On the morning of POD 14, he complained of back pain, but it was initially thought to be related to surgical wound. PT-INR was 1.51. He developed lower extremity numbness at 1 p.m.. Back pain subsequently increased, and paraplegia developed at 2 p.m.. Contrast-enhanced CT was performed on suspicion of recurrent PE, aortic dissection, or SEH, but a radiologist detected no abnormalities (**Fig. 2**). As paraplegia developed and sensory deficit ascended to the umbilicus and nipple, he was taken to emergency hospital to receive neurologist's examination.

Chest magnetic resonance imaging was immediately performed and it revealed a small amount of SEH extending between Th4 and Th7 compressing the spinal cord (**Fig. 3**). He underwent emergency laminectomy and hematoma evacuation. Intraoperative findings revealed both fresh blood and old hematoma at the level of Th5–Th6 where the epidural catheter was inserted. After 2.5 months' rehabilitation, he regained all sensation and was able to walk and was discharged.

Fig. 3 Sagittal magnetic resonance image revealed a spinal epidural hematoma extending between Th4 and Th7 compressing the spinal cord (A: T1-weighted; B: T2-weighted).

Discussion and Conclusion

In this case, SEH occurred on POD 14, even though appropriate anticoagulant therapy for PE had been initiated after epidural catheter removal. Thoracic surgeons should keep SEH in mind for rapid diagnosis and early treatment even late after epidural anesthesia.

Both epidural anesthesia and anticoagulant therapy are etiological factors for SEH.¹⁾ In our institution epidural anesthesia is generally not employed for patients who need anticoagulant therapy in the perioperative period. However, this patient needed anticoagulant therapy because of postoperative PE and accordingly both of them coexisted. Intraoperative findings of both old hematoma and fresh blood meant that epidural catheter insertion caused some amount of hematoma, and anticoagulant therapy induced additional fresh hematoma. The information regarding possible initial damage is limited. There are no data on the number of times the anesthesiologist tried to insert the epidural catheter. According to the anesthesia record, it took only 11 minutes to perform epidural anesthesia, and continuous bleeding was not observed.

The thoracic vertebral canal is narrower than the lumbar one, and the epidural space in the midthoracic region is especially narrow.²⁾ The thoracic extradural space contained less fatty and fibrous connective tissues and became widely patent after injecting a given amount of air compared with the lumbar extradural space.³⁾ Based on these data, it is important to recognize that even a small amount of hematoma could compress the spinal cord especially

in the midthoracic region where the epidural catheter is placed for lung surgery.

Approximately 70% cases of SEH related to lumbar or epidural anesthesia presented within a day, and almost all cases occurred within a week.¹⁾ Our case is extremely rare because SEH occurred on POD 14 despite appropriate anticoagulant therapy for PE; the maximum PT-INR was 1.51 and fondaparinux treatment did not require monitoring.

The Japanese Circulation Society and 6 other societies jointly developed guidelines for DVT prevention and they classified patients into 4 categories: low, intermediate, high, and highest depending on their risk of DVT.⁴⁾ This classification is based on that of American College of Chest Physicians (ACCP) 6th edition, but the recommended prophylaxes for each rank in the former is determined to be equal to the original prophylaxis for one risk level lower. These treatment regimens are conservative because of a lack of data for Japanese patients and the fact that Japanese patients develop PE less frequently. This case met the definition of high risk because it was a major cancer surgery in a patient ≥ 40 years old, and recommended prophylaxis is IPC or anticoagulant therapy according to Japanese guidelines, whereas anticoagulant therapy is recommended by the ACCP and other societies including the American Society of Clinical Oncology and the European Society for Medical Oncology.^{5,6)} Anticoagulant therapy seemed necessary for preventing this patient from PE.

Low-molecular-weight heparin and fondaparinux cause a lower risk of bleeding than unfractionated heparin but are not officially indicated for patients with lung surgery as a prophylaxis of DVT in Japan. Considering the risk of bleeding associated with unfractionated heparin, it is important to strictly select appropriate candidates. Niimi, et al. reported that soluble fibrin, one of fibrin-related markers, reflected hypercoagulable state before clot formation and was more useful than D-dimer to diagnose DVT on POD 1.⁷⁾ Soluble fibrin may be useful to select adequate candidates for prophylactic anticoagulant therapy.

There are several reports that paravertebral block is as effective as epidural anesthesia during thoracic surgery.⁸⁾ We now perform paravertebral block instead of epidural anesthesia. We should carefully consider the indication for epidural anesthesia and select alternative techniques when possible.

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Disclosure Statement

Y. Kobayashi and the other co-authors have no conflict of interest.

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