Disappearance of a major thrombus in the brachiocephalic vein without anticoagulant therapy in a patient with seminoma: A case report

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Abstract

This is the first case report describing brachiocephalic vein thrombosis without compression by a metastatic tumour during chemotherapy for testicular cancer. According to previous reports of testicular cancer patients with a major thrombus, anticoagulant therapy was required to resolve all cases. However, in the present case, a major thrombus in the brachiocephalic vein disappeared without anticoagulant therapy. This 42-year-old man was diagnosed with testicular seminoma and multiple metastases to the para-aortic lymph nodes. After 3 cycles of cisplatin, etoposide and bleomycin (PEB) therapy, a major thrombus in the right brachiocephalic vein was recognized on a computed tomography (CT) scan. Although no anticoagulant therapy was undertaken, the thrombus in the right brachiocephalic vein was no longer visible on CT after the fourth cycle of PEB therapy.

Introduction

Cisplatin-based chemotherapy is the main treatment for metastatic testicular cancer, and is generally well-tolerated. However, some non-hematological toxicity is infrequently found in patients undergoing cisplatin-based chemotherapy. According to previous reports of testicular cancer patients receiving chemotherapy, thrombosis is rare. Several reports have described venous thrombosis in testicular cancer patients during chemotherapy.¹⁻³

We report a case of thrombosis in the right brachiocephalic vein that occurred during cisplatin, etoposide and bleomycin (PEB) therapy for testicular seminoma. The thrombosis in the present case disappeared even though no anticoagulant therapy was undertaken.

Case report

A 42-year-old man visited the emergency department of our hospital with a complaint of abdominal pain in March 2009. He was of a medium build and had no smoking history. He had no history of thrombosis. Enhanced abdominal computed tomography (CT) images showed enlargement of the para-aortic lymph nodes. The patient was admitted to the department of surgery with suspected malignant lymphoma or metastatic tumour of the stomach. A lymph node biopsy was performed via laparotomy and the pathological findings of the lymph node indicated seminoma. The patient was referred to our department for a further workup.

A right high-inguinal orchiectomy was performed and the patient was diagnosed with seminoma (T1 N3 M0 S3, AJCC Stage IIIB) by a pathological examination. We started PEB therapy (20 mg/mm² cisplatin, 100 mg/mm² etoposide, and 30 mg/body bleomycin). However, he had severe pancy-topenia after the first and second PEB therapies. Therefore, we reduced the dosage of the each drug to 80% at the third cycle.

An enhanced CT scan was obtained after the third cycle. The images showed a partial regression of the metastases and, unexpectedly, a non-enhanced deposit. This deposit seemed to be a major thrombus in the right brachiocephalic vein, although the patient had no symptoms of thrombosis and had never experienced thrombotic events (Fig. 1a). At that time, the platelet count was at its highest $(532 \times 10^9/L)$.

Subsequently, we consulted with our hospital cardiologist. The cardiologist recommended further PEB therapy without anticoagulant therapy; it was thought that the platelet count would drop to about 50×10^{9} /L based on the fact that the patient had experienced severe thrombocytopenia after receiving previous PEB therapy. Therefore, we performed the fourth cycle of PEB therapy and observed a decrease in the platelet count to about 50×10^{9} /L on day 21.



Fig. 1a. Enhanced chest computed tomography scan after the third cycle of cisplatin, etoposide, and bleomycin (PEB) therapy showed a thrombus in the right brachiocephalic vein (arrow).

CT images after the fourth cycle showed that the thrombus in the right brachiocephalic vein had disappeared (Fig. 1b) with remission of the tumour. There has been no sign of a thrombus on CT scans taken over a 1-year period following the disappearance of the first thrombus.

Discussion

In this case, we detected a major thrombosis of the brachiocephalic vein during PEB therapy for testicular cancer. It subsequently disappeared without anticoagulant therapy.

There have been several reports of venous thrombosis due to compression by metastatic tumours.²⁻⁴ However, there are only a few reports of venous thrombosis without such vascular compression.

According to previous reports of testicular cancer patients with a thrombus detectable by CT scanning, all cases were treated by anticoagulant therapy, such as warfarin, low-molecular heparin and acetylsalicylic acid.^{3,5} However, in the present case, the major thrombus had completely resolved itself without the need for anticoagulant therapy.

The common types of thrombosis during chemotherapy for testicular cancer are deep vein thrombosis (DVT) of the legs, venous access port-related DVT and pulmonary embolism.^{2,6} Central venous thrombosis is a quite exceptional complication of testicular cancer. In our case, the brachiocephalic vein thrombosis may have been caused by some chemical factors because the tumour did not compress the vein and agents were administered into a peripheral vein, without using an access port.

Although the complete mechanism of the coagulation abnormality associated with cancer has not yet been clarified, some mechanisms which may contribute to it have been elucidated in previous studies. These mechanisms include: tissue factors produced in cancer cells, endothelial impairment by cytokines secreted from activated leukocytes, and vascular toxicity associated with antineoplastic agents.^{7,8}



Fig. 1b. Enhanced chest computed tomography scan after the fourth cycle of cisplatin, etoposide, and bleomycin (PEB) therapy, the thrombus disappeared.

Some factors that cause thrombosis in cancer patients during chemotherapy have been reported. These factors include the progression of cancer, tumour location (genital), anemia, a high platelet count, a high leukocyte count $(450 \times 10^9/L)$, obesity, corticosteroids for antiemetic agents, and the administration of agents with vascular toxicity (cisplatin, etoposide).⁹⁻¹¹ The present case had 3 risk factors: a genital tumour, high platelet count (532 × 10⁹/L, when the third PEB therapy was initiated), and the administration of agents with vascular toxicity.

The platelet count when the third cycle of PEB therapy was started was at its highest (532×10^{9} /L), and the thrombus was subsequently observed on a CT scan. Although no anticoagulant therapy was undertaken, the thrombus disappeared when the platelet count dropped to about 50×10^{9} /L after the fourth cycle. Therefore, in our case, the high platelet count might have strongly contributed to the appearance of the thrombus.

Conclusion

We encountered a case of testicular cancer with thrombosis in a brachiocephalic vein, without compression of the vein by a metastatic tumour during PEB therapy. The thrombus was successfully eradicated without any anticoagulant chemotherapy. In this patient, a high platelet count may have strongly contributed to the appearance of the thrombus.

Competing interests: None declared.

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