Superior vena cava syndrome arising from subclavian vein port catheter implantation and paraneoplastic syndrome

Subklavyan ven port kateteri komplikasyonu ve paraneoplastik sendrom sonucu gelişen süperiyor vena kava sendromu

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Central venous thrombosis is an important complication of venous catheterization. We presented a 49-year-old male patient who developed massive central venous thrombosis causing superior vena cava (SVC) syndrome after placement of a right subclavian vein port catheter. The patient had inoperable gastric cancer for which he had been receiving chemotherapy for two years. He had a six-month history of fixed port catheter placement into the right subclavian vein. Contrast-enhanced computed tomography (CT) of the chest showed complete obstruction of the SVC and CT angiography showed extensive thrombosis from the subclavian vein to the end of the SVC. Extensive lung and mediastinal metastases were also observed. Surgical intervention was not considered. Fibrinolytic therapy was instituted with 75 mg tissue plasminogen activator (tPA) infusion for 18 hours. The patient's symptoms and the signs of SVC syndrome disappeared and clinical parameters returned to normal within several hours. The day after completion of fibrinolytic therapy, repeat contrast CT angiography showed total resolution of SVC thrombosis. Slow infusion of tPA may be effective in the treatment of SVC syndrome caused by acute thrombosis.

Key words: Catheterization, central venous/adverse effects; fibrinolytic agents/therapeutic use; paraneoplastic syndromes; superior vena cava syndrome; thrombosis/etiology.

Santral venöz tromboz venöz kateterizasyonun önemli bir komplikasyonudur. Bu yazıda, sağ subklavyan vene port kateteri yerleştirilmesinden sonra süperiyor vena kava (SVK) sendromuna yol açan yoğun santral venöz tromboz gelişen 49 yaşında bir erkek hasta sunuldu. Hasta inoperabl gastrik kanser nedeniyle iki yıldır kemoterapi görmekteydi ve sağ subklavyan vene altı ay önce port kateteri yerleştirilmişti. Kontrastlı göğüs bilgisayarlı tomografisinde (BT) SVK'yi tamamen tıkayan trombüs ve BT anjiyografide subklavyan venden SVK'ye kadar uzanan yaygın tromboz görüldü. Ayrıca, akciğer ve mediyastende yaygın metaztatik lezyonlar gözlendi. Hasta için cerrahi girişim düşünülmedi ve 75 mg doku plazminojen aktivatörü (tPA) ile 18 saatlik infüzyon uygulandı. Fibrinolitik tedavinin birkaç saati içinde hastanın semptomları ve SVK sendromu bulguları kayboldu ve klinik tablosu normale döndü. Fibrinolitik tedavinin bitiminden bir gün sonra tekrarlanan kontrastlı BT anjiyografide SVK trombozunun tamamen çözüldüğü görüldü. Yavaş ve uzun süreli tPA infüzyonu akut trombozun yol açtığı SVK sendromu tedavisinde etkili olabilir.

Anahtar sözcükler: Kateterizasyon, santral venöz/yan etki; fibrinolitik ajan/terapötik kullanım; paraneoplastik sendrom; süperiyor vena kava sendromu; tromboz/etyoloji.

Central venous catheterization and port placement are essential practices in the modern management strategy for chemotherapy applications. Thrombosis is one of the most frequent and severe complications of vascular ports.[1] Venous thrombosis may also occur as a paraneoplastic manifestation of some internal cancers.[2] We describe a case of thrombotic occlusion of the entire superior vena cava (SVC) after placement of a subclavian vein port. Although surgical removal of the thrombosis is a well-known strategy for the treatment of SVC occlusions, fibrinolytic therapy may be an alternative strategy for port-associated thrombotic SVC syndrome.

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CASE REPORT

A 49-year-old male patient presented to the emergency room with dyspnea, shortness of breath, weakness, and unconsciousness. Swelling of the face, neck and both extremities, purple-colored face, sweating, and forced speaking were observed on inspection. Physical examination showed tachycardia, tachypnea, weak pulse, and hypotension. Laboratory findings including complete blood cell count, serum electrolytes, clotting profile, and cardiac enzymes were in normal limits. Arterial blood gas analysis showed severely decreased oxygen saturation, high level of CO_2, and acidosis. He was admitted to the coronary care unit and intubated for mechanical ventilation.

The patient had inoperable gastric cancer for which he had been receiving only chemotherapy for two years. He had a six-month history of fixed port catheter placement into the right subclavian vein, resulting in left subclavian vein thrombosis which was suboptimally treated by subcutaneous enoxaparin. It was learned that he had not received enoxaparin within the past month.

A clinical diagnosis of SVC syndrome was made. Contrast-enhanced computed tomography (CT) of the chest showed complete obstruction of the SVC (Fig. 1a) and CT angiography showed extensive thrombosis from the right subclavian vein to the end of the SVC, and collateral circulation along the thoracic wall into the inferior vena cava. On the chest CT scan, extensive lung and mediastinal metastases were also observed. Considering rapid deterioration of symptoms, extensive involvement by metastatic gastric cancer, and high surgical risk, surgical intervention was not considered. Fibrinolytic therapy was the only choice for the patient, which was instituted with 75 mg tissue plasminogen activator (tPA) infusion for 18 hours, combined with enoxaparin 0.6 ml twice daily and aspirin 300 mg daily. Complete blood count, blood gases, and clotting profile were monitored. The patient's symptoms and the signs of SVC syndrome disappeared and clinical parameters returned to normal within several hours. The patient was then extubated. Slight gingival and nose bleeding continued. At the end of tPA infusion, we observed hematoma in the leg muscles and subcutaneous bleeding in both arms. Hematocrit decreased to 27%, and one unit of erythrocyte suspension was infused to the patient. The day after completion of fibrinolytic therapy, contrast CT angiography was repeated, which showed total resolution of SVC thrombosis (Fig. 1b). There was no residual thrombosis in the subclavian vein, SVC, and right atrium. After two days of hospital care, the patient was discharged without major bleeding and thrombosis-related complications.

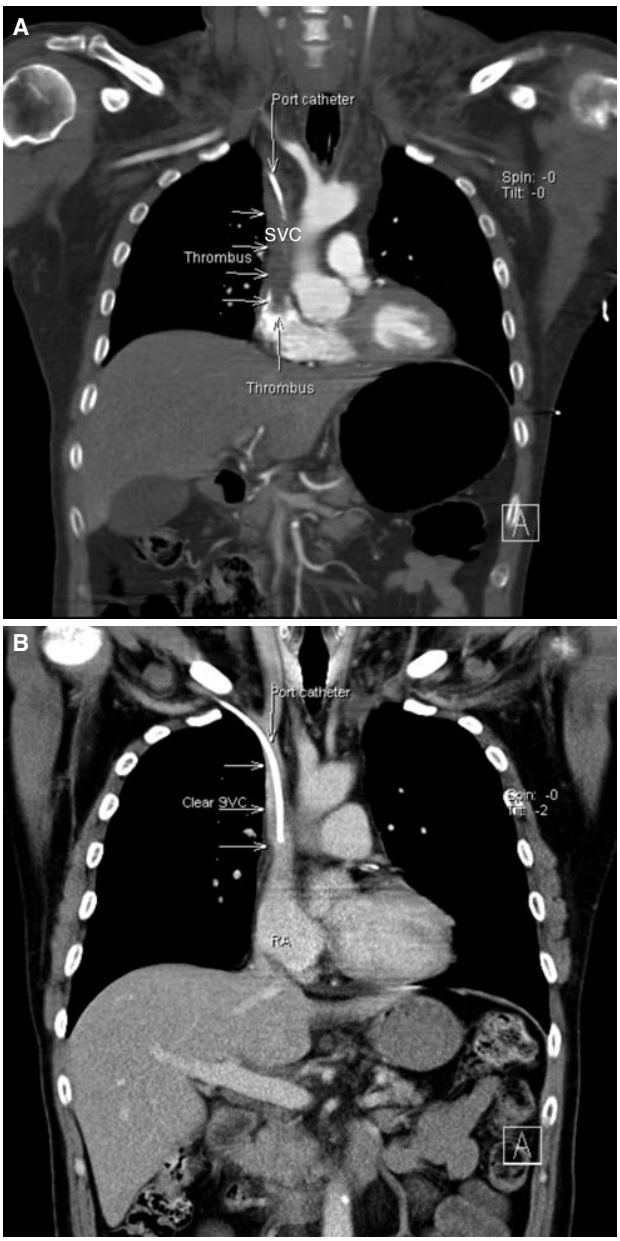

Figure 1. (B) Contrast computed tomography angiography showing massive thrombotic occlusion of the superior vena cava (SVC). **(B)** Complete resolution of massive thrombosis following fibrinolytic therapy. SVC: Superior vena cava; RA: Right atrium.

DISCUSSION

Central venous port catheterization is frequently used in cancer patients receiving chemotherapy. Gastric cancer may give rise to central venous thrombosis as a paraneoplastic manifestation. Development of a

fibrin sleeve around a catheter or thrombus adherent to a vessel wall is not uncommon after central venous catheterization. The incidence of fibrin sleeve formation ranges from 42% to 100%.[3,4] The formation, growth, and dissolution of venous thrombosis are governed by several thrombogenic (duration of catheterization, physical properties of the catheter, nature of infused fluid) and protective mechanisms. Despite the high incidence of, and the presence of many factors predisposing to, local thrombosis around catheter tips, symptomatic massive thrombosis causing SVC syndrome, as seen in our case, is not common. Although central venous port catheter was the main cause of catheter-related thrombosis in our patient, it can be speculated that metastatic gastric cancer must have contributed to the extent of thrombosis.

Venography and CT angiography are the most useful diagnostic methods for the diagnosis of SVC syndrome. We found CT angiography easy and rapid not only for the diagnosis of our patient but also for comparison of pre-and post-treatment findings.

Anticoagulation therapy and surgical repair consisting of thrombectomy have been the mainstay of treatment to prevent clot propagation and relieve symptoms in patients with catheter-related thrombosis.[5,6] Doty et al.[7] reported that a spiral vein bypass graft for the treatment of obstructed SVC relieved SVC syndrome and provided excellent long-term patency in 16 patients.

In our patient, massive thrombosis was completely resolved after 18 hours of tPA infusion. Guijarro Escribano et al.[8] presented a case of SVC syndrome associated with catheter use for chemotherapy, which was successfully treated with catheter-directed (intraclot) infusion of urokinase. In contrast, Suzuki at al.[9] reported that thrombolytic therapy was not effective in a patient with massive organizing thrombi in the superior vena cava and bilateral internal jugular and subclavian veins. Endovascular stenting may be another option in patients with SVC thrombosis. Boza at al.[10] treated a patient successfully with thrombolysis, angioplasty, and stent placement.

In conclusion, central venous port catheterization may result in severe thrombosis which may be further complicated by internal cancers, emphasizing the need for close evaluation of patients bearing a central venous catheter. Slow infusion of fibrinolytic therapy may achieve complete resolution of thrombosis especially when diagnosed early.

REFERENCES

1. Schuster W, Vennebusch H, Doetsch N, Taube HD. Vena cava superior thrombosis following placement of internal jugular vein catheter. Anaesthesist 1978;27:546-7. [Abstract]
2. May M, Seehafer M, Helke C, Uberrück T, Gunia S, Hoschke B. Superior vena cava syndrome with bilateral jugular and subclavian vein thrombosis. Paraneoplastic manifestion of renal cell carcinoma. Urologe A 2003;42: 1374-7. [Abstract]
3. Brismar B, Hårdstedt C, Jacobson S. Diagnosis of thrombosis by catheter phlebography after prolonged central venous catheterization. Ann Surg 1981;194: 779-83.
4. Raad II, Luna M, Khalil SA, Costerton JW, Lam C, Bodey GP. The relationship between the thrombotic and infectious complications of central venous catheters. JAMA 1994;271:1014-6.
5. Ko SH, Kang SK, Lee SK, Song HS. Massive thrombosis after central venous catheterization in a patient with previously undiagnosed Behcet's disease. J Korean Med Sci 2001;16:814-6.
6. Calderon MC, Lozano VM, Jaquez A, Villaseñor C. Surgical repair of superior vena cava syndrome. Ann Thorac Surg 2001;71:1351-3.
7. Doty JR, Flores JH, Doty DB. Superior vena cava obstruction: bypass using spiral vein graft. Ann Thorac Surg 1999;67:1111-6.
8. Guijarro Escribano JF, Antón RF, Colmenarejo Rubio A, Sáenz Cascos L, Sainz González F, Alguacil Rodríguez R. Superior vena cava syndrome with central venous catheter for chemotherapy treated successfully with fibrinolysis. Clin Transl Oncol 2007;9:198-200.
9. Suzuki J, Kanbayashi T, Koizumi T, Honda T, Yamada H, Sekiguchi M. A case of superior vena cava syndrome due to extensive venous thrombosis of an idiopathic nature. Nihon Kyobu Shikkan Gakkai Zasshi 1992;30:1761-5. [Abstract]
10. Boza C, Mertens R, Valdés F, Kramer A, Mariné L, Vergara J. Endovascular therapy of superior vena cava syndrome: clinical case. Rev Med Chil 2001; 129:421-6. [Abstract]