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Catheter-Directed Thrombolysis for Acute SVC Obstruction in a Patient with Metastatic Breast Cancer

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ABSTRACT

Background: External compression, thrombosis, or stenosis of the superior vena cava can lead to superior vena cava syndrome, a diagnosis that should be considered swiftly in patients presenting with classic symptoms such as facial and neck swelling, plethora, and distended neck veins.

Case Presentation: We report a case of acute port-a-cath associated superior vena cava thrombosis in a longstanding, previously uncomplicated vascular access device in a patient with stable ER+/PR+/HER2+ metastatic breast cancer. After initial treatment, there was limited clinical improvement with subcutaneous low molecular weight heparin (LMWH). Following multidisciplinary team discussion, catheter-directed thrombolysis was performed, which resulted in complete symptom resolution.

Conclusion: The recognition of signs and symptoms is crucial in diagnosing acute superior vena cava syndrome, particularly in patients with a malignancy history or a central venous access device in situ. The thrombotic complications of port-a-cath symptoms can occur at any time and management should be guided by multidisciplinary discussion. In appropriately selected patients, catheter-directed thrombolysis can be successful and can lead to rapid symptom resolution.

INTRODUCTION

Superior vena cava syndrome (SVCS) is a clinical condition due to obstruction of the superior vena cava (SVC) characterized by facial and upper extremity oedema and engorgement of neck veins, resulting in dyspnoea cyanosis collateralization of chest wall vessels, and is generally confirmed by cross-sectional imaging.[1] Superior vena cava obstruction (SVCO) is caused by external compression of the SVC by intrathoracic malignant disease, most commonly bronchogenic carcinoma or lymphoma.[2]

Intrinsic stenosis or thrombosis of the SVC caused by central lines or medical devices can also result in SVC syndrome.[3] The increasing use of central venous access devices, such as port-a-cath systems, which are placed to facilitate long-term chemotherapy administration, has led to an increase in the number of patients diagnosed with superior vena cava syndrome.[4-6] Thrombotic complications of port-a-cath systems occur most commonly due to stenosis or occlusion of the host vein due to thrombus formation at the catheter

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tip, which more commonly occurs when the catheter tip is incorrectly positioned.[7,8]

CASE PRESENTATION

A 39-year-old female with a history of de novo bone-limited oligometastatic triple-positive breast cancer, on maintenance trastuzumab, pertuzumab and tamoxifen since the completion of her initial chemotherapy in 2016, presented to the emergency department with a two-hour history of malaise with facial swelling and plethora. She had a right-sided port-a-cath in situ, which was inserted at the time of her diagnosis to facilitate chemotherapy administration; the port-a-cath had been accessed without any problem two days ealier.

She was placed on bed rest in an upright position, and a urinary catheter was inserted to ensure minimal exertion. On examination, she appeared unwell, with significant facial plethora, periorbital and facial oedema, and distended neck veins. Based on her presentation and the severity of her symptoms, a clinical diagnosis of SVCO was made, and she was commenced on therapeutic tinzaparin at a dose of 175u/kg.[9]

Her condition worsened with facial engorgement with increasing facial plethora (Figure 1A).

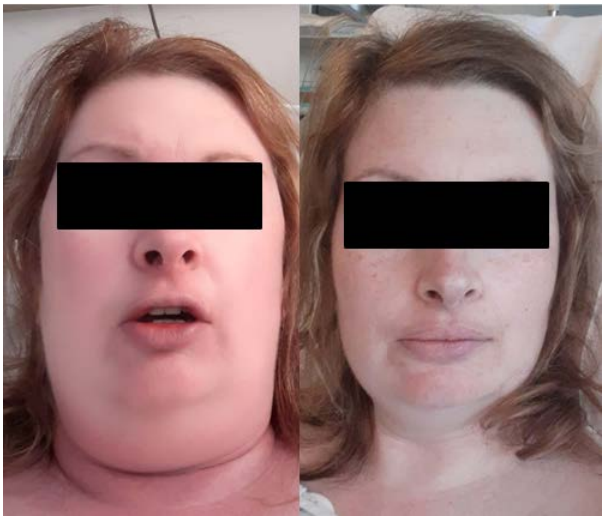

Figure 1A (Left). Facial oedema and plethora in keeping with clinical signs of SVCO
Figure 1B (Right). Resolution of signs 24 hours after catheter-directed thrombolysis

Computed tomography of the thorax demonstrated thrombus at the port tip with near-total occlusion of the superior vena cava and extension into the left brachiocephalic vein (Figure 2), with extensive collateralization in the left supraclavicular region. The tip of the port-a-cath was in a cranial position, with its tip in the mid-SVC. The optimal position for the tip of a port-a-cath is at the cavo-atrial junction or upper right atrium.[10]

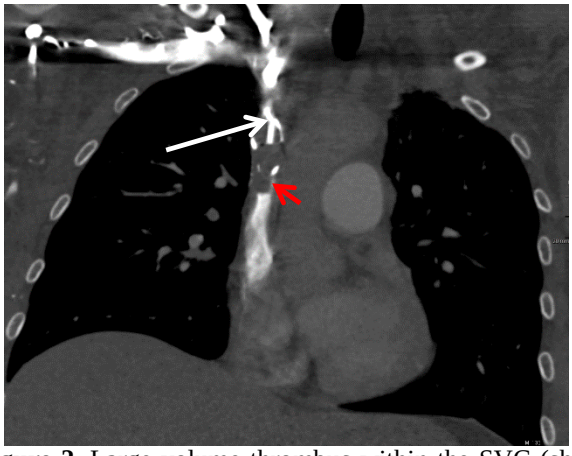

Figure 2. Large volume thrombus within the SVC (short red arrow) with the port-a-cath tubing seen centrally within this thrombus (long white arrow)

Due to her escalating symptoms, treatment was intensified, with a change of anticoagulation to twice daily enoxaparin at a 110% dose by weight, with a view to escalating care to a higher dependency ward to administer unfractionated heparin, in case of any clinical deterioration. Tamoxifen was stopped due to its pro-thrombotic properties.

There was no clinical improvement over the following 24 hours. Following multidisciplinary discussion, a decision was made to proceed with catheter-directed thrombolysis. Given the history of malignancy, a CT brain was performed to exclude intracranial metastases, which would contraindicate alteplase administration. Venography via the left brachial vein confirmed a large v olume of near occlusive thrombus within the left brachiocephalic vein and SVC (Figure 3). A thrombolysis catheter was positioned across the thrombus before catheter-directed thrombolysis with alteplase initiated at a rate of 1mg/hr.

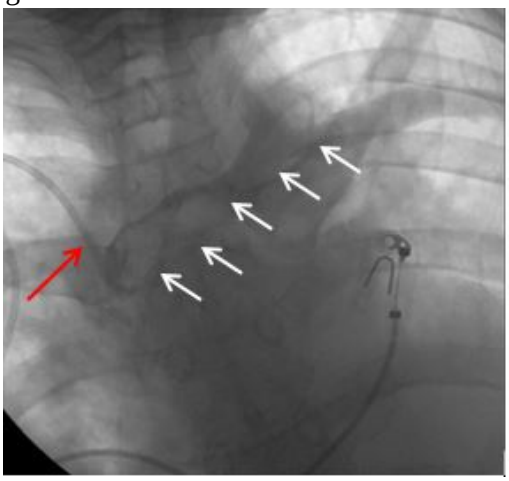

Figure 3. Venography demonstrating thrombus within the left brachiocephalic vein extending into the SVC (white arrows). The tip of the port-a-cath (red arrow) is displaced cranially by the thrombus, sub-optimally located at the junction between the right brachiocephalic vein and the SVC.

The following morning, there was a marked clinical and symptomatic improvement (Figure 1B). A repeat venogram was performed, demonstrating complete resolution of the thrombus. Venography also revealed a narrowing at the left brachio-cephalic/SVC junction, near the tip of the port-a-cath which responded to venoplasty (Figure 4). The remaining clinical course was uneventful, and the patient was discharged 72 hours later on therapeutic tinzaparin.

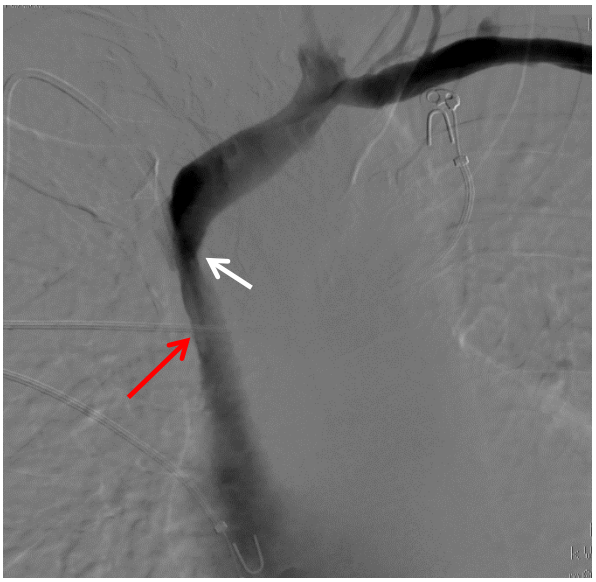

Figure 4. Venography post 22 hours of thrombolysis demonstrating complete resolution of the thrombus. The tip of the port-a-cath (red arrow) is now positioned more caudally within the mid SVC. There is a stenosis in the cranial SVC (white arrow) which responded to venoplasty.

The inciting factor for thrombosis was felt to be secondary to the positioning of the port-a-cath tip. Therefore, six weeks later, the chest wall pocket was opened, and the old port tubing was removed and a new longer tubing was inserted with the tip positioned at the cavo-atrial junction as she had an ongoing need for central access. Following this intervention, the patient was transitioned from tinzaparin to rivaroxaban and tamoxifen was resumed. She remains well on rivaroxaban and maintenance subcutaneous Herceptin and tamoxifen.

DISCUSSION

Patients with underlying malignancy are at higher risk of developing thrombotic complications. Here we discuss a patient with SVC obstruction secondary to port-a-cath related thrombosis, despite the uncomplicated use of port-a-cath for more than five years. Other contributing risk factors for our patient included obesity and tamoxifen use, although tamoxifen-associated VTE is more common in the first two years of therapy.[11,12]

Patients presenting with SVCS generally have characteristic clinical findings on examination, which can vary in severity and duration. On examination, patients will most commonly have oedema of the face, neck, and/or upper extremities and collateralization of the veins in the neck or on the chest wall. Less common signs include plethora, cyanosis, dyspnoea, dysphagia, stridor, or neurological symptoms.[13] In acute SVC obstructions, patients present with a short history and more classic symptoms, such as in our patient's case. If the SVC obstruction has occurred over time, patients may have less severe signs and symptoms due to compensation from collateral vessels. In either scenario, signs and symptoms of cerebral oedema, laryngeal oedema, and haemodynamic compromise suggest a life-threatening or severe SVCS and should prompt immediate management.[14,15]

While there are no consensus guidelines on managing venous device-associated thrombus and subsequent SVC syndrome, this case demonstrates catheter-directed thrombolysis's safety and clinical utility for port-a-cath related thrombosis in the appropriately selected patient. Multidisciplinary collaboration and discussion to balance bleeding and thrombotic risks led to the successful use of catheter-directed thrombolysis. Early detection of thrombosis is essential as thrombolysis produces higher success rates if carried out in the first five days following clot formation.[7]

CONCLUSION

Venous thrombosis is a rare but well-recognized adverse event in patients with venous access devices and can lead to an acute or chronic presentation of superior vena cava syndrome. This case demonstrates that thrombotic complications of central venous access devices can develop at any time following insertion. Recognition of classic SVCO symptoms allows immediate and appropriate management, which is essential to limit morbidity. Catheter-directed thrombolysis can be safe and effective in appropriately selected patients with significant clinical symptoms. This case demonstrates that timely recognition and subsequent prompt intervention are vital to successful patient outcomes.

ETHICAL CONSIDERATION

Informed consent for publication was obtained from the patient.

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None.

CONFLICT OF INTEREST

None.

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