

peer reviewed **CASE REPORT****Case report of superior vena cava syndrome with collateral circulation**

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Cancer and its therapy may lead to urgent conditions of which superior vena cava syndrome is one. In view of the range of cancers some patients may require assistance in an emergency care facility. This case report describes a known lymphoma patient who presented with severe SVC syndrome symptoms in the emergency department. He then underwent emergency CT imaging for urgent treatment planning. The syndrome's pathophysiology, imaging and possible treatment plan are presented.

**Keywords**

Computerised tomography, radiation oncologist, interventional radiologist, lymphoma.

**Case report**

A 21 year old male who was awaiting a treatment plan for a recently diagnosed lymphoma presented to the emergency department as he was experiencing shortness of breath, generalised fatigue and a swollen neck.

A computed tomography (CT) chest examination was requested. As per departmental protocol for CT chest angiography only the post contrast enhanced series was performed. His urea and creatine levels were confirmed to be within normal limits. Informed consent was obtained and contrast media was administered through a cannula on the left antecubital vein at a rate of 3mls a second. However, due to an anticipated possibility of collateral flow manual bolus tracking was used. The scan commenced when contrast started

filling the superior vena cava (SVC). Axial CT images demonstrated a large mass in the mediastinum compressing the trachea and obliterating the SVC above the azygos vein entry point into it (Figure 1). As a sequestration to the obstruction collateral circulation from the paraspinal plexus, azygos, hemiazygos and costal vessels is demonstrated in Figures 1 to 4. SVC obstruction on CT images includes: lack of opacification of the SVC (Figures 1,2 & 3), an intramural filling defect or severe narrowing of the superior vena cava (Figure 2), and visualisation of collateral vascular channels (Figures 1,2 & 3)<sup>[4]</sup>. The CT findings confirmed SVC obstruction syndrome caused by a mediastinal mass. Collateral circulation was also demonstrated.

**Discussion**

SVC syndrome results from partial or com-

plete obstruction of blood flow through the superior cava vein to the right atrium, causing severe reduction in venous return from the head, neck and upper extremities<sup>[1]</sup>. This syndrome is listed amongst oncology emergencies. The syndrome encompasses a constellation of symptoms and signs resulting from obstruction of the SVC. The increased pressure in the upper body results in oedema of the head, neck, arms, often cyanosis, plethora and distended subcutaneous vessels. The decreased venous return may also result in hemodynamic compromise<sup>[2]</sup>. Radiological imaging is therefore useful to confirm the diagnosis and identify the site and cause of SVC obstruction<sup>[3]</sup>.

The SVC carries blood from the head, arms, and upper torso to the heart. It carries approximately one third of the venous return to the heart. Compression of the



Figure 1: Axial CT. White arrow shows mass in the mediastinum compressing on the SVC and trachea. Fat, short white arrow shows collateral circulation from the spinal venous plexus. Black arrow shows the azygos vein emptying into the SVC upon being supplied by the collateral circulation network.

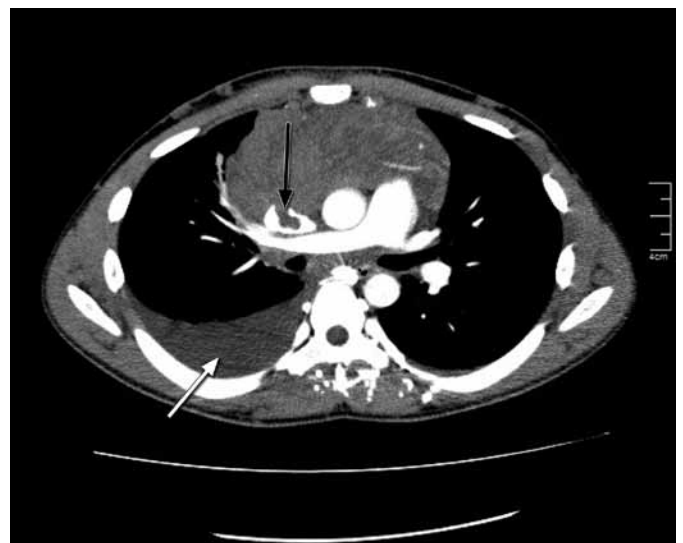


Figure 2: Black arrow shows SVC with a thrombus. White arrow shows pleural effusion in the right lung.

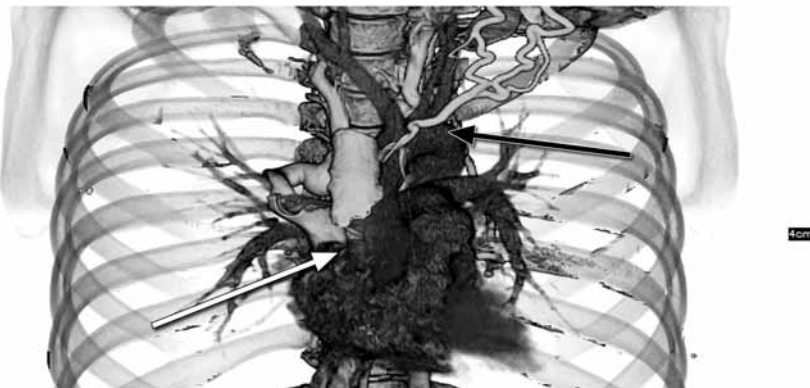


Figure 3: 3D showing SVC obstructed (white arrow) and being fed by the collateral circulation (black arrow).

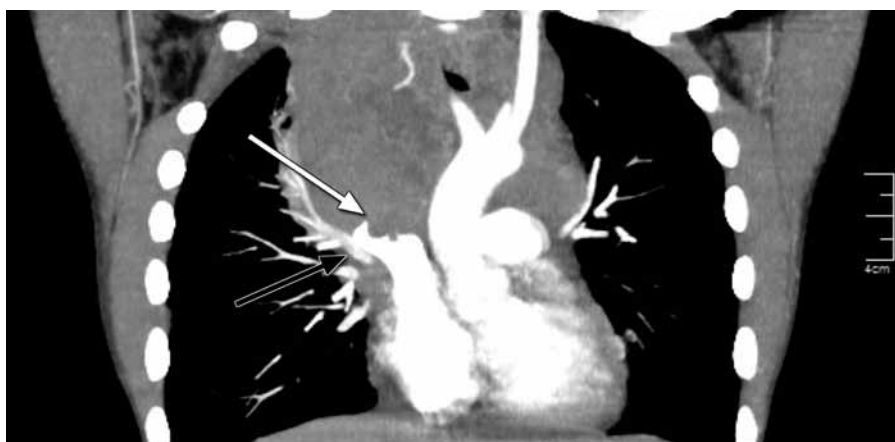


Figure 4: Coronal image. White arrow shows mediastinal mass obstructing the SVC and a thrombus in the vena cava. Black arrow shows the point of entry of the collateral circulation into the SVC.

SVC may result from the presence of a mass in the middle or anterior mediastinum, consisting of enlarged right paratracheal lymph nodes, lymphoma, thymoma, an inflammatory process, or an aortic aneurysm<sup>[5]</sup>. Lymphomas are the most likely cause of anterior mediastinal masses, often with SVC obstruction and pleural and pericardial effusions<sup>[6]</sup>. SVC syndrome, first described in 1757 by William Hunter, refers to constellation of clinical symptoms caused by obstruction of the SVC. The obstruction is nearly always (>85%) attributable to advanced malignancy, most commonly lung cancer<sup>[2]</sup>. Less frequent causes are lymphoma, metastatic cancer, germ cell tumours, thymoma and mesotheliomas<sup>[3]</sup>. The SVC obstruction by malignant diseases is either by direct invasion and compression or by tumour thrombus of the SVC<sup>[7]</sup>. Most SVC syndromes are associated with advanced malignant diseases that cause invasion of the venous intima or an extrinsic mass effect. This syndrome is caused by gradual compression of the SVC leading to oedema and retrograde venous flow; it can also be caused more abruptly in thrombotic cases<sup>[8]</sup>. Obstruction of the SVC causes elevated pressure

in the veins draining into it and increased or reversed blood flow through collateral vessels<sup>[7]</sup>.

When there is an obstruction blood flows through a collateral venous network to the lower body, often via the azygos vein<sup>[3]</sup>. In the event of chronic occlusion, collateral pathways seen with SVC obstruction include the azygos-hemiazygos, internal and external mammary, lateral thoracic and vertebral pathways<sup>[9]</sup>. In addition several unusual collateral pathways may be seen with superior or inferior vena cava obstruction<sup>[9]</sup>. The azygos-hemiazygos pathway includes the azygos, hemiazygos, intercostals, and lumbar veins<sup>[9]</sup>. The azygos and hemiazygos veins can divert blood away from the SVC if the level obstruction is above the azygos arch. Antegrade flow from the azygos to the right atrium is evident in Figure 4<sup>[4]</sup>.

The hemiazygos is the common trunk of the last three to five left intercostals veins. The various collateral pathways seen in superior vena cava obstruction can result in early enhancement of the IVC during imaging. Collateral vessels following the azygos-hemiazygos and vertebral venous

plexus route drain directly into the inferior vena cava<sup>[10]</sup>.

Occluded lymphatic flow from increased hydrostatic pressure in the SVC and left brachiocephalic vein probably contributes to the development of chylous pleural fluid. Chylous and exudates pleural effusions occur in most patients with SVC syndrome; the effusions are usually small and resolve upon correction of the underlying obstruction<sup>[8]</sup>.

Multidetector CT has the advantage of combining cross sectional imaging for diagnosis of the cause of the SVC obstruction with multiplanar reformation that best delineates the level and extent of venous obstruction. Such information is invaluable to the surgeon, radiation oncologist, or interventional radiologist for planning further management<sup>[4]</sup>.

## Conclusion

SVC syndrome gradually develops over several weeks and is unlikely to be a life threatening condition unless the patient presents with airway obstruction or increased intracranial pressure<sup>[3]</sup>. Treatment is both supportive and definitive therapy<sup>[8]</sup>. Multiple collateral venous pathways can form to bypass an obstruction of the SVC. The patient in this case report had approximately 50% tracheal stenosis and good collateral circulation. In view of this he was scheduled for chemotherapy treatment. Other symptoms of the syndrome will be managed as they present.

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## peer reviewed CASE REPORT

# Case report: An open book fracture of the pubic symphysis demonstrated by multi-slice computed tomography (MSCT)

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### Abstract

This case report describes an open book fracture of the pubic symphysis with an accompanying large pelvic haematoma, diagnosed in a young male following a motorcycle accident. His clinical history, radiological findings as well as treatment are discussed.

### Keywords

Haematoma, embolisation, active haemorrhage.

### Case report

A young male involved in a motorcycle accident was admitted to a private hospital for trauma management. He had no medical history of note. He required ventilation, inotropic support and blood transfusions. He was then referred for a full body scan.

A CT scan of his brain, cervical spine, chest, abdomen and pelvis was performed. The chest, abdomen and pelvis protocol allowed for his chest to be scanned in an arterial phase utilizing bolus tracking and the abdomen and pelvis in the venous phase. The slice thickness for the primary data was acquired using 5mm slice widths and the secondary data were reconstructed at 1.25mm. Due to patient movement the acquired images were suboptimal but the scan revealed that there were no definite liver or pancreatic parenchymal injuries with both kidneys enhancing uniformly.

The CT findings (Figures 1-4) demonstrated an un-displaced base of skull fracture of the left temporal bone and left middle cranial fossa; a subarachnoid haemorrhage with blood in the gyri and sulci was present. There was excessive fluid in the nasal cavity and oropharynx. No cervical spine injuries were noted and there was normal alignment of the vertebrae. The 2<sup>nd</sup>, 3<sup>rd</sup> and 4<sup>th</sup> ribs were frac-

tured with extensive air space opacification in the upper lobes of the chest. An apical pneumothorax was present on the right side.

An open book fracture was seen at the pubic symphysis (Figure 1 and 4). Active haemorrhage was present in the right adnexal area with a large haematoma displacing the bladder in a superolateral direction (Figure 2 and 3). Some haemorrhage was noted in the mesenteric root. Oedema in the anterior abdominal wall was also identified (Figure 3).

The patient underwent bilateral iliac artery embolisation where occlusion of the medial sacral branches was achieved. He then had a voiding cystourethrograde (VCU) procedure to demonstrate his micturating abilities. An anterior plate was surgically inserted to stabilize the open book fracture.

### Discussion

An open book fracture of the pubis symphysis can be described as an anterior force that causes a disruption of the pubic symphysis<sup>[1]</sup>. Separation of the pubic symphysis greater than 1cm is considered abnormal. The force causes each hemi pelvis to externally rotate<sup>[1]</sup>. Fixation of the open book fracture depends on the stability of the patient and displacement of the fracture. Internal fixation with anterior plat-

ing and screws is often performed when there is an open book fracture however the patient should be haemodynamically stable<sup>[2]</sup>.

Pelvic fractures can lead to death. However, the fractures are not the primary cause as related injuries can cause further complications<sup>[3]</sup>. Pelvic trauma has a high mortality rate and with the use of multi slice computed tomography (MSCT), fractures, vascular and organ injuries can be identified with ease. Compared to plain-film radiography MSCT has a higher sensitivity to detect abnormalities. Pelvic fractures are often accompanied by haemorrhaging. With open book fractures the sacroiliac joint is supported by posterior ligaments and opens like a hinge. Open book fractures require the highest number of blood transfusions. Pelvic fractures lead to extensive bleeding; the retroperitoneum is capable of holding 4 litres of blood<sup>[4]</sup>.

Embolisation of arterial haemorrhaging caused by pelvic fractures is advisable even in unstable patients as death and organ failure can be avoided<sup>[3]</sup>. Transcatheter arterial embolisation (TAE) in relation to haemorrhage control and reduced transfusions has proven successful in 85%-100% of the patients it is performed on<sup>[5]</sup> and should be performed as soon as possible to prevent morbidity or mortality<sup>[6]</sup>.