

Congested

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Congested: A Clinical Presentation of the Inferior Caval Vein Syndrome

Inferior caval vein syndrome

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A 52-year old male patient presented to our emergency department with respiratory distress, clinically and biochemically in shock (lactate 11 mmol/L). On clinical examination, a blueish discoloration of the abdomen and lower extremities was observed (Figure 1A). On contrast-enhanced computed tomography, the inferior caval vein (IVC) was compressed at the level of the 4th lumbar vertebra by a massive collection of lymph nodes (Figure 1B,C), confirming diagnosis of IVC syndrome (IVCS). Emergency invasive contrast venography (Figure 2A,B) and subsequent thrombectomy was performed in our hybrid operating room with venous stent recanalization (Figure 2C). The patients' condition improved remarkably, after which he was discharged home on the sixth postoperative day on acenocoumarol. Diagnostic follow-up excluded malignancies, and laboratory findings were negative for tumor markers. Biopsy of the nodal collection was inconclusive. Two weeks postoperatively, magnetic resonance venography revealed a marked decrease of peri-caval vein thrombosis without evidence of pathological lymph nodes (Figure 3).

IVCS is less common than its superior caval vein counterpart and its true incidence has never been reported, potentially leading to under-recognition of this syndrome (1). The etiology of IVCS depends on the location of blood flow interruption and can roughly be divided into intraluminal obstructive, and external compressive causes. Intraluminal obstruction may be caused by a primary thrombotic event, while external IVC compression is usually the consequence of malignant growth of nearby organs (2). Another well-known risk factor is pregnancy, during which the distended uterus may progressively compress the IVC (3). In addition, prior abdominal surgery, obesity and presence of congenital malformations such as May-Thurner or Budd-Chiari syndrome might predispose to IVCS (2, 4). Treatment of ICVS depends on its etiology, and usually requires resolution of the cause of compression. In many patients, anticoagulant therapy might resolve or prevent future IVCS, while intravascular procedures and surgery are reserved for more critical cases (1). Although IVCS is obstructive of nature, it might fool the clinician as it mimics hypovolemic shock, while distension of jugular and upper torso veins is absent. Immediate recognition and treatment is imperative as it might lead to end-organ failure and eventual death.

FIGURE LEGENDS

Figure 1. Findings on clinical and computed tomography examination.

(A) blueish discoloration of the abdomen and lower extremities with clear evidence of venous congestion, (B, C) arterial contrast-enhanced coronal (B) and axial (C) computed tomography images revealing an inferior caval vein syndrome caused by a compressive collection of lymph nodes (arrows indicate the location of lymph node collection).

Figure 2. Pre- and postprocedural contrast venography.

(A, B) preprocedural imaging of the right (A) and left (B) femoral vein (asterix indicates occlusion), (C) postprocedural venography of the inferior caval vein (arrow indicates stent).

Figure 3. Follow-up magnetic resonance venography.

A remarkable spontaneous decrease of paracaval lymphadenopathy and caval vein compression without evidence of pathological lymph nodes (arrow indicates prior location of compressive nodes).

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338x190mm (300 x 300 DPI)



Figure 2. Pre- and postprocedural contrast venography. (A, B) preprocedural imaging of the right (A) and left (B) femoral vein (asterix indicates occlusion), (C) postprocedural venography of the inferior caval vein (arrow indicates stent).

1045x585mm (72 x 72 DPI)



Figure 3. Follow-up magnetic resonance venography. A remarkable spontaneous decrease of paracaval lymphadenopathy and caval vein compression without evidence of pathological lymph nodes (arrow indicates prior location of compressive nodes).

1057x793mm (72 x 72 DPI)