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Varices of inferior epigastric veins caused by chronic inferior vena cava obstruction: mimicking normal venous flow pattern on radionuclide venography

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A 21-year-old patient with long-standing inferior vena cava obstruction secondary to idiopathic thrombosis extending from the external iliac veins underwent a radionuclide venography with Tc-99m pertechnetate labeled erythrocytes. The blood pool phase of the study revealed bilaterally distorted inferior epigastric veins mimicking normal venous flow pattern. The authors present this case to discuss the possible alternative routes and the underlying physiopathologic mechanism of this unusual flow pattern in chronic inferior vena cava obstruction.

Key words: Tc-99m RBC, inferior vena cava, thrombosis

INTRODUCTION

THE INFERIOR VENA CAVA (IVC), the largest vein in the body, drains blood from the abdomen and lower extremities. It originates behind the right common iliac artery, ascends in the retroperitoneum, and penetrates the diaphragm to enter the right atrium. Although it has a host of tributaries, the main ones are the common iliac veins, lumbar veins, right ovarian or testicular vein, renal veins, right suprarenal vein, phrenic vein, and hepatic veins. Different etiologic factors such as congenital, traumatic, inflammatory or neoplastic diseases can cause IVC obstruction. Despite this major function, complete occlusion of the IVC is compatible with life because of the presence of several secondary systems that can compensate for its function when required.

CASE REPORT

A 21-year-old male suffering from venous engorgement in the abdominal wall and swelling of both legs for more than one year underwent bilateral lower extremity radionuclide venography with Tc-99m RBC to exclude possible venous obstruction. Radionuclide venography was performed after the injection of 20 mg pyrophosphate for *in vivo* labeling of RBCs at the same session. Dynamic images were taken immediately after the injection of 15 mCi (555 MBq) of Tc-99m pertechnetate into both dorsal pedal veins. Early filling and varicose changes in the inferior epigastric veins were noted in the dynamic phase of the scintigraphic study (Fig. 1). Delayed blood pool images showed distorted and kinked inferior epigastric veins mimicking patent external-common iliac veins and IVC at first sight (Fig. 2). The IVC obstruction was confirmed by contrast venography subsequently (Fig. 3). No underlying cause was identified and the diagnosis was made as an idiopathic thrombosis of the IVC extending from the iliac veins. Anticoagulant therapy to resolve the thrombus was initiated. Though radionuclide venography is a valuable technique to evaluate the collateral pathways in the presence of deep venous obstruction, it should be remembered that sometimes, prominent superficial collaterals can superpose on the original venous routes and mimic normal flow patterns.

DISCUSSION

Although congenital, traumatic, inflammatory or

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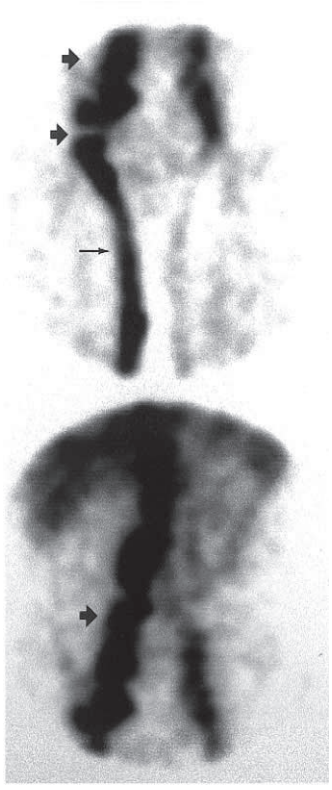

Fig. 1 Dynamic images revealed early filling of the radiotracer in the bilateral great saphenous (*thin arrow*), femoral and inferior epigastric veins (*thick arrows*). Varicose inferior epigastric veins were noted in the early phase of the study, especially on the right side.

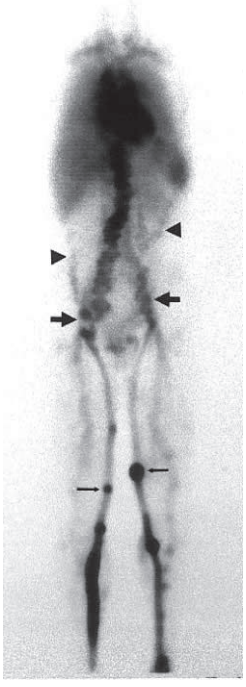

Fig. 2 Whole-body images taken during the equilibrium phase of Tc-99m pertechnetate labeled RBC showed distorted and kinked bilateral inferior epigastric veins mimicking patent iliac external-common iliac veins and IVC (*thick arrows*). In addition, other collateral veins on the abdominal wall were noted (*arrowheads*). Multiple hot spots in the bilateral great saphenous veins are representative of marked dilatations of varicose veins (*thin arrows*).

neoplastic diseases may also cause IVC obstruction, the major cause is thrombosis.[1,2] Often because of superior extension of idiopathic thrombophlebitis in the lower extremities or pelvis, thrombus can also be secondary to more generalized conditions such as dehydration, sepsis, localized inflammation, pelvic inflammatory disease, coagulopathy, congestive heart failure, immobility, trauma, or severe exertion.[1,3] After IVC obstruction, the low pressure collaterals develop easily within a short period of time to allow retrograde flow.[4] The collateral channels might be classified as practical routes and potential routes on the basis of *in vivo* venography and injection and dissection techniques, respectively.[5] In fact, all vessels that parallel the axis of an obstructed vessel are potential collateral pathways if they are directly or indirectly linked to the trunk above and below the level of obstruction, but the practical routes are more important in diagnostic imaging and in clinical practice. Because the collateral circulation is further modified by the level of obstruction, alternative routes of venous flow of IVC can be subdivided and calssified as infrarenal obstruction, middle IVC obstruction and upper IVC obstruction.[6] The findings in our case were consistent with infrarenal obstruction since the IVC and iliac veins were not visualized during the contrast and radionuclide venographies. This level of the

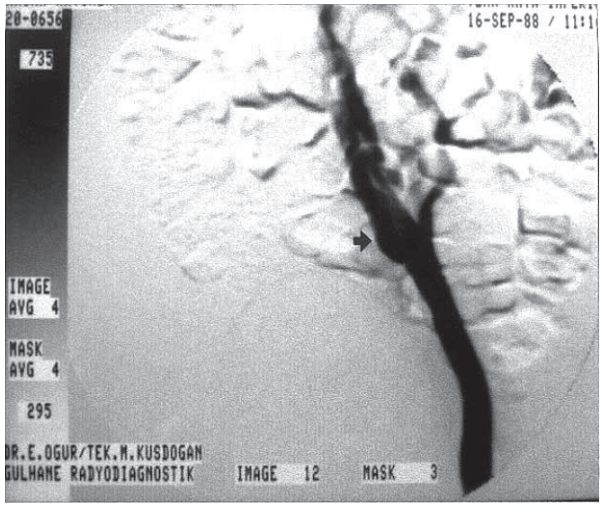

Fig. 3 Venography after the injection of contrast material into the left femoral vein revealed venous return dependent mainly on the collaterals especially to the left inferior epigastric vein (*arrow*) that drains superiorly into the internal mammary vein. The IVC and iliac vein could not be visualized during the procedure.

IVC is more commonly involved with occlusive disease than are the remaining segments.[7]

In the presence of infrarenal IVC obstruction, there are four alternative practical pathways[4]: a) The central route (the ascending lumbar veins, internal and external vertebral venous plexuses, azygos-hemiazygos complex and the inferior vena cava itself above the obstruction level), b) Intermediate route (the gonadal veins, the ureteric veins, and the left renal-azygos venous system), c) Portal route (filling via the superior hemorrhoidal anastomosis with the middle and inferior hemorrhoidal plexuses of the internal iliac venous system. When abdominal wall veins are transporting large volumes of blood in a collateral fashion, there may be drainage into a patent umbilical vein), d) Superficial route (inferior epigastric veins, circumflex iliac and superficial epigastric veins). The principal venous collaterals are the azygos-hemiazygos veins, since they connect the superior and inferior vena cava and anastomose with the common iliacs through the ascending lumbar veins and other tributaries.[8] In most instances it is these bilateral collaterals that account for an important, characteristic, and early feature of vena caval obstruction on radionuclide venography.[9] These are also the most frequently demonstrated pathways on contrast-enhanced vena cavograms.[10]

If a vein is valved against the necessary direction of the collateral flow as in inferior epigastric veins, it cannot act as a collateral channel immediately. Since it takes several weeks to overcome the valve function in the superficial routes, during this time, other routes (e.g. intermediate, central and portal) may accommodate the increase in flow and therefore diminish the need for further collateral transport from the superficial system. Therefore superficial routes are numerous but are not always prominent in infrarenal obstruction of the IVC except in those cases in which there is occlusion of one or both of the common iliac veins.[4] Since the iliac veins of our case were also occluded by thrombus, the femoral venous blood flow was blocked from reaching the other paracaval collaterals by this low level obstruction, and then, superficial venous system in the abdominal wall remained the only alternative route to transport the blood.

A single large channel is more effective in collateral circulation than multiple small channels. This principle is in conformity with Poiseulle's law: The volume of flow is proportional to the fourth power of the diameter of the vessel[11] and it may explain the prominent appearance of bilateral inferior epigastric veins as a major conduit vessel in the abdominal wall of our case.

In healthy veins, one-way valves usually direct the flow of venous blood upward. Prolonged and increased workload leads to increased hydrostatic pressures that can cause chronic venous distention and dilatation. Once the vein undergoes dilatation to approximately twice its diameter, the valves become incomplete and flow can occur in either direction.[11] Reflux through incompetent valves in the veins causes elevation of the venous pressure, and veins continue to dilate and become tortuous in response to continued high pressure. Wali et al. have proposed that dilatation and distensibility of the vein wall under normal and increased venous pressure are due to a deficiency in smooth muscle cells and elastic fibers and disproportionate increase in fibrous tissue.[12] Since the inferior epigastric veins have worked as a main conduit vessel, chronic increased workload and intraluminal pressure have caused its dilatation. Hence the distortion and tortuosity increased in time and the unusual varicose appearance of the inferior epigastric veins evolved in our case.

In IVC obstruction, a hot spot suggesting systemic-portal shunt via the paraumbilical vein has been reported to be seen in the quadrate lobe in some cases.[13,14] Although abdominal wall veins were transporting large volumes of blood in a collateral fashion, we did not notice the quadrate lobe in our case. For this hot spot to develop, it appears necessary to have systemic-portal venous blood flow through the paraumbilical vein.

As a conclusion, although radionuclide venography with Tc-99m RBC blood pool imaging is helpful to assess venous flow abnormalities and to detect unusual collateral formation in the presence of the IVC obstruction, it should be remembered that prominent superficial collaterals could superpose on original venous routes and mimic normal flow patterns.

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