

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Internal Medicine Quiz – Case 9

A 60-year-old woman was admitted for progressive pitting edema of the lower part of the body and dyspnea on exertion, which started 3 months ago.

Sixteen months ago, she had received chemotherapy and radiation therapy for cancer of the uterine cervix that was locally advanced, encroaching the left ureter. She was a former smoker with 5 pack years. Her medical history was unremarkable.

On physical examination her vitals were BP 120/70 mmHg, HR 100/min, RR 26/min and her temperature was normal. Symmetrical edema of the lower half of the body up to the umbilicus and the lumbar area was noted. The edema was pitting, soft and non-tender, and worsened on assumption of upright posture. No lymph nodes were palpable. Auscultation of the heart was normal and the jugular veins were not distended. Lung auscultation revealed reduced breath sounds at the right base, accompanied by dullness on percussion. The abdomen was non-tender, distended symmetrically due to ascites, and distention of the veins of the lateral abdominal wall with cephalad blood flow was noted (fig. 1).

Laboratory testing showed elevated serum urea at 103 mg/dL (range 17–50 mg/dL) and creatinine at 2 mg/dL (range 0.6–1.2 mg/dL), hyponatremia (126 mmol/L, range 136–145 mmol/L), and elevated alanine aminotransferase (349 U/L, range 5–40 U/L), aspartate aminotransferase (141 U/L, range 5–40 U/L), alkaline phosphatase (684 U/L, range 64–280 U/L), γ -GT (396 U/L, range 7–32 U/L), LDH (1,015 U/L, range 200–460 U/L) and CPK (303 U/L, range 20–190 U/L) and a normal serum protein electrophoretic pattern.

A chest X-ray was obtained that showed normal lung parenchyma and cardiothoracic index, but a mild elevation of the right hemidiaphragm. On echocardiography of the heart the left ventricular ejection fraction was shown to be normal (>65%), along with the cardiac valves and the pericardium, ruling out cardiac failure.

Paracentesis of the ascitic fluid proved it to be portal hypertensive ascites with a SAAG (serum-ascites albumin gradient) of 2, negative cultures and negative cytologic examination. An ultrasound examination of the liver and spleen was ordered, which apart from the ascites revealed no abnormalities, whereas a lower abdomen ultrasound showed a normal right kidney, but

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C.S. Floudas,¹
T. Ntalageorgos,¹
C. Kanta,¹
M. Moustardas,²
K. Revenas,³
G. Tzortzis,³
G. Vaiopoulos,¹
H. Gogas¹

¹First Department of Internal Medicine,

²2nd Department of Propedeutic Surgery,

³Department of Radiology, National and Kapodistrian University of Athens, Medical School, "Laiko" General Hospital, Athens, Greece



Figure 1

a shrunken left kidney with dilated renal pelvis. An abdominal CT scan confirmed the presence of ascites and indicated the presence of some small para-aortic lymph nodes, but no mass. The presence of progressive portal hypertensive ascites with distention of the abdominal wall veins in the face of a non-cirrhotic liver indicated the need for imaging of the intra-abdominal veins, in search for evidence of obstruction. Therefore, a triplex ultrasonography of the portal and splenic veins and of the inferior vena cava was obtained, with no abnormal findings. Doppler ultrasonography of the veins of the lower limbs displayed delayed flow with enhancement after external pressure however, color duplex ultrasonography of the lower limbs was normal.

An MRI venography of the inferior vena cava (IVC) showed a stenotic area (fig. 2) and a transfemoral contrast IVC venography (cavography) was ordered. The cavography (fig. 3) revealed a relatively abrupt but smooth cut-off of the venous flow at the level of the diaphragm as well as collateral circulation via the azygos and vertebral veins. Endovascular management with balloon angioplasty and placement of an intraluminal stent (fig. 4) resulted in restoration of the venous flow and relief of the venous and portal hypertension. After 3 days, marked improvement of the edema and ascites had occurred, as well as a reduction of the serum creatinine to 1.3 mg/dL. A repeat cytological examination of the ascites revealed malignant cells. After one week the patient succumbed to sepsis from rupture of the small intestine due to metastatic disease.

Comment

The inferior vena cava syndrome (IVCS) is an obstruction of

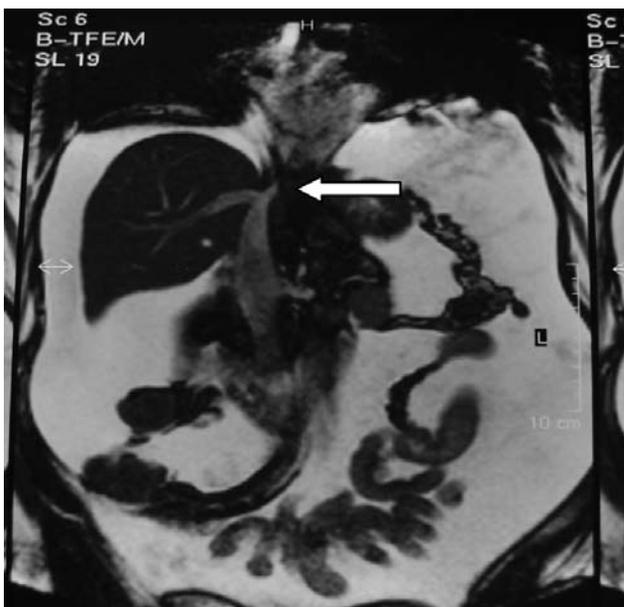


Figure 2



Figure 3



Figure 4

the IVC that can be the result of direct invasion (from a tumor), compression (as can happen in pregnancy, from the enlarged uterus, or from enlarged lymph nodes) and thrombosis. Thrombosis at the suprahepatic-diaphragmatic portion of the IVC, that leads to hepatic venous outflow obstruction, is part of the Budd-Chiari syndrome (BCS). BCS can also be the outcome of thrombosis of the hepatic veins or of obliterative hepatocavopathy, a condition that is relatively more common in Asia; the latter is caused by mem-

branous obstruction of the IVC at the level of the diaphragm, and can lead to development of hepatocellular carcinoma. Malignant obstruction of the IVC is more often seen in patients with advanced cancer, which often have extensive metastatic disease of the liver or lymph nodes.

The clinical manifestations of the IVCS are secondary to venous congestion and vary according to the severity, the duration and the level of the obstruction, as well as the richness of collateral blood flow. Ascites and edema of the abdominal wall, scrotum and lower limbs may be the presenting symptoms. Tachycardia may also be present, as well as respiratory distress due to pulmonary restriction from increased pressure on the diaphragm. Impairment of the renal and hepatic function can also be observed.

Patients that are diagnosed with malignant IVCS are usually

terminal or at an advanced stage, so most treatment measures are aimed to symptomatic improvement and palliation. Conservative measures (diuretics etc.) provide minimal relief from the edema and ascites. Endovenous treatment can be helpful: for thrombosed segments catheter-directed thrombolysis may be attempted, whereas for stenotic segments balloon angioplasty and dilation followed by stenting for improved patency is an option. Prognosis is grave.

Corresponding author:

C.S. Floudas, First Department of Internal Medicine, National and Kapodistrian University of Athens, Medical School, "Laiko" General Hospital, 17 Agiou Thoma street, GR-115 27 Athens, Greece, tel.: +30 210 74 56 312
e-mail: chfloudas@gmail.com