

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Surgery Quiz - Case 1

An 86-year-old woman presented at the emergency department of our hospital with diffuse abdominal pain, vomiting and low grade fever. Symptoms began 24 hours ago and gradually worsened. Abdominal pain was initially located in the middle line above the umbilicus and rapidly spread to the right upper quadrant (RUQ), reflecting to the ipsilateral scapula and lower thoracic spine. A few hours later the abdominal pain was characterized as diffuse. Vomiting first occurred after the onset of pain, was repeated several times and contained gastric fluid with no blood admixture. Low grade fever up to 38°C was also present and verified at the emergency department. No specific medical history or prior abdominal surgery was reported. The patient was on antihypertensive regimen for the last decade. Clinical examination revealed abdominal tenderness with marked guarding at the RUQ and a positive Murphy sign on palpation. Tachycardia was also present. CBC (complete blood count) and serum biochemical analysis revealed moderate leukocytosis (12,500/ μ L) with increased number of neutrophils (80%) and normal biochemical values. Plain abdominal radiograph was not diagnostic and an ultrasound examination of the upper abdomen was performed. Gallbladder wall thickening (5 mm) with subserosal edema and gallbladder distention, pericholecystic fluid and a positive sonographic Murphy sign were reported. No obvious cause of obstruction was observed. Based on clinical and radiologic findings, the diagnosis of acute cholecystitis was established. The patient was admitted in our clinic and received medical treatment with a double intravenous antibiotic regimen consisting of a second generation cephalosporin (cefoxitin) and an antianaerobic agent (metronidazole) for one week. Surgical intervention was excluded at this time because of the age and severity of inflammation and the absence of an obvious obstructive cause. The patient was discharged in good clinical condition with the prescription of an oral antibiotic regimen for one week.

Twenty days after hospital discharge, however, the patient presented once more at the emergency department with the same clinical presentation. Clinical examination and laboratory tests were consistent with the previous findings and she was readmitted in our clinic. At this time a computed tomography (CT) scan of the abdomen was performed which revealed the presence of a mass within the gallbladder, arising from its anterior wall and occupying half or more of the organ's lumen in transverse sections, and gallbladder distention with pericholecystic edema (fig. 1). CT findings were compatible with gallbladder malignancy. The patient underwent laparoscopic cholecystectomy which was laborious due to severe inflammation. Examination of the surgical specimen revealed purulent content (gallbladder empyema) and occult perforation of the gallbladder wall. Histological examination showed a benign adenomatous gallbladder polyp without evidence of malignant transformation. The patient received appropriate antibiotic treatment and was discharged two days postoperatively.

Comment

Gallbladder tumors are quite rare accounting for less than 3% (benign lesions) or 1% (malignant lesions) of the surgical specimens in patients undergoing laparoscopic cholecystectomy. Cholesterol polyps account for approximately 50% of all polypoid lesions of the gallbladder and are formed due to a defect in cholesterol metabolism. Their size is usually smaller than 10 mm in diameter and in most cases they are asymptomatic. Gallbladder adenomas can be sessile or polypoid and are considered as premalignant lesions. A size greater than 12 mm increases the risk of malignant transformation.

Early diagnosis of gallbladder tumors is rare due to the insidious nature of the disease. In most cases an incidental finding during cholecystectomy or clinical presentation of a complication raise suspicion of gallbladder pathology. The presence of a polypoid mass fixed to the

ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):508
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):508

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Figure 1. Abdominal CT scan section of an 86-year-old woman presenting with a neoplastic lesion of the gallbladder (arrow). Tumor dimensions on CT image were 3×3×4.5 cm.

gallbladder wall of normal thickness or wall thickening alone should raise suspicion of a neoplasm, although in many cases coexistence of benign gallbladder disease (e.g. cholelithiasis) is sufficient to explain the symptoms and, thus, the tumor is diagnosed only at exploration or pathologic examination of the resected gallbladder.

Computed tomography (CT) is more sensitive than ultrasound in identifying gallbladder neoplasm and the management of benign lesions depends on size and the presence or absence of symptoms. The risk of malignant transformation of polypoid lesions increases when the lesion is greater than 1 cm. The recommendations for lesions less than 1 cm include follow-up and reevaluation of the lesion with repeated imaging studies. Patients with symptomatic lesions, i.e. biliary colic or acute cholecystitis, are eligible for cholecystectomy. The indications for cholecystectomy in asymptomatic cases include coexistent biliary disease, such as cholelithiasis, a polyp greater than 1 cm in patients older than 50 years with a wide-based lesion and a single polyp lesion or an enlarging lesion. Laparoscopic cholecystectomy is usually the surgical procedure of choice in most cases.

Prognosis of benign lesions of the gallbladder is obviously excellent provided that prompt surgical treatment is administered.

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Diagnosis: Polypoid lesion of the gallbladder.

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Clinical Immunology Quiz - Case 2

A 45-year-old male was admitted to the hospital complaining of increasing breathlessness and productive cough. His medical problems commenced in early childhood with recurrent episodes of pneumonia and he reported frequent episodes of sinusitis and respiratory infections as an adult. Upon physical examination, diffuse rhonchi and inspiratory crackles were heard at both the middle and lower respiratory areas, while no hepatosplenomegaly or adenopathy was observed. Laboratory studies revealed a white blood count of $5.9 \times 10^9/L$ (normal values: $4-10 \times 10^9/L$), hemoglobin 13.8 g/dL (normal values: 14–16 g/dL), platelet count $197 \times 10^9/L$ (normal values: $140-400 \times 10^9/L$). The white count differential was as follows: neutrophils 77.7%, lymphocytes 19.3%, monocytes 3%, without the presence of blasts. The erythrocyte sedimentation rate was 65 mm for the first hour (normal <20 mm) and C-reactive protein levels were 6.1 mg/dL (normal <0.8 mg/dL). Renal and liver function, electrolytes, and uric acid were all normal. High-resolution computed tomography of the chest revealed multiple, extensive, variably sized cystic bronchiectasis in both lungs. Peripheral blood of the patient was referred to the Immunology Lab for immunophenotyping that revealed reduced number of B-cells (fig. 1, Tab. 1).

Which is the next laboratory step?

Serum protein electrophoresis showed hypogammaglobulinemia and the quantitative evaluation of serum immunoglobulins revealed a marked decrease of IgG and IgA levels (tab. 1). The above findings (B-cell reduction, hypogammaglobulinemia and a medical history of recurrent respiratory infections from early childhood) were consistent with the diagnosis of common variable immunodeficiency (CVID).

Comment

CVID is the most prevalent primary immunodeficiency presenting in childhood or adult life. Peaks of onset occur in children aged 1–5 years and in persons aged 16–20 years. Interestingly, more than two-thirds of patients are aged 21 years or older when CVID is diagnosed. Most patients have reduced serum levels of IgG and IgA, normal or slightly reduced levels of IgM and normal or low numbers of B-cells. About one-third of the patients have some degree of abnormality of cell-mediated immunity. Affected individuals experience recurrent respiratory bacterial infections and a delayed diagnosis (as in our patient) can lead to permanent damage to the bronchi, resulting in bronchiectasis and pulmonary arterial hypertension. Autoimmune phenomena and a higher prevalence of malignancies (especially of lymphomas) are other common complications of the disease. The mainstay of treatment for CVID

ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):509
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):509

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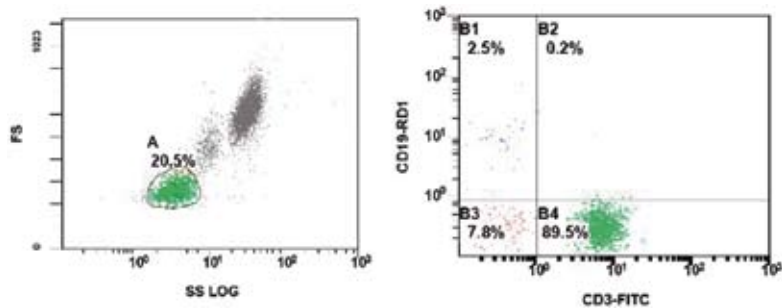


Figure 1. Flow cytometry analysis showing reduced number of B-cells.

Table 1. Lymphocyte subsets and quantitative serum immunoglobulins of the patient.

Blood lymphocyte subpopulations ($\times 10^9/L$)		
Total lymphocyte count	1.215	[1.0–3.5]
T lymphocytes		
CD3	1.167	[0.9–2.5]
CD4	0.554	[0.5–1.5]
CD8	0.583	[0.25–1.0]
B lymphocytes		
CD19	0.031	[0.1–0.5]

Quantitative serum immunoglobulins (mg/dL)		
IgG	331	[874–1690]
IgA	28	[99–300]
IgM	78	[64–249]

is Ig replacement therapy (400–600 mg/kg every 3–4 weeks) that can stop the cycle of recurrent infections.

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Diagnosis: Common variable immunodeficiency (CVID)

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Gastroenterology-Endoscopy Quiz - Case 2

A 82 years old female patient was admitted to our hospital with melenas. She had not any past history of aspirin, NSAIDs, or anti-platelet usage and no complaint for any gastrointestinal (GI) symptom. Digital rectal examination was positive for melena, and there were no other abnormal findings in physical examination. Her vital signs were steady and laboratory results revealed a severe microcytic anemia (Hct 20.3% Hg 6.5 g/dL), WBC 7,360/μL, PLT 195,000/μL, urea 134 mg/dL, and creatinine 1.07 mg/dL. Prothrombin time and INR were within normal range.

During the last three years she was hospitalized several times due to upper GI bleeding or severe iron deficiency anemia. She had multiple gastroscopies, two colonoscopies and one capsule endoscopy of the small bowel. All endoscopic examinations were not diagnostic for the source of bleeding.

This time, an upper GI endoscopy revealed "fresh" blood in the second part of the duodenum (fig. 1). After water lavage a red thrombus became evident, which was subsequently removed (fig. 2). Neither ulceration, nor angiodysplasia was present in the underlying mucosa, treated successfully with heater probe, three pulses of 20 Joules in total.



Figure 1



Figure 2

Comment

The Dieulafoy lesion is a rare but an ever-present potential cause of life-threatening hemorrhage, and in the hemodynamically unstable patient with otherwise normal endoscopic findings, this diagnosis has to be considered.

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Diagnosis: Vascular Dieulafoy lesion of the duodenum

ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):510
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):510

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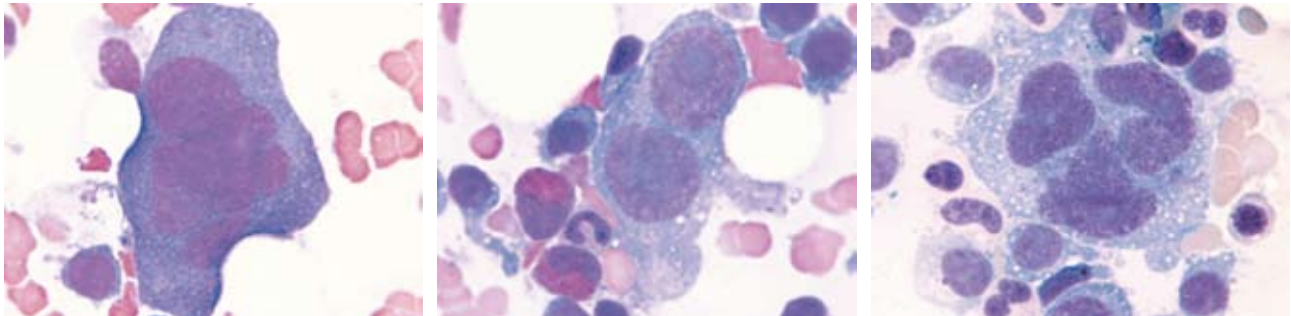
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CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Hematology Quiz - Case 2

A 56-year-old man was admitted to our hospital because of back pain, headache, visual disturbances, and gum bleeding during the last 5 days. On physical examination the patient was pale and sleepy, while several ecchymoses were seen in his arms. His heart, lungs, abdomen and lymph nodes were normal. The full blood count showed a normocytic anemia (Hb 9.9 g/dL, MCV 91 fL), thrombocytopenia (platelet count of $82 \times 10^9/L$) and normal white blood cell ($8.2 \times 10^9/L$) and differential counts. Blood biochemistry revealed an elevated serum creatinine (3.2 mg/dL), severe hypercalcemia (16 mg/dL) and hypoalbuminemia (2.9 g/L). Protein electrophoresis and immunoelectrophoresis revealed a monoclonal IgA κ peak of 68 g/L. Beta2-microglobulin was also elevated (12.7 mg/L). The chest radiograph, computed tomography of the brain that was performed in the emergency department and radiography of the skull were normal. MRI of the lumbar spine showed diffuse infiltration of the bone marrow by malignant cells and multiple osteolytic lesions.

A few abnormal cells of plasmacytic morphology were found in the peripheral blood. In the bone marrow aspiration smears, numerous large atypical plasma cells with multiple nucleoli, at a proportion of 60% of the total nucleated cell population, were observed (figures 1–3). A trephine marrow biopsy displayed highly pleomorphic cells with coarse clockface-like chromatin and eccentric cytoplasm. Immunophenotypic study showed a moderate expression of CD56, CD45 and CD10 antigen and a dim positivity of CD138 and cytoplasmic lambda



light chain. Karyotyping showed hyperploidy (92, XY) with several numerical and structural abnormalities.

The diagnosis was established and the patient was given a combination of chemotherapy plus high dose dexamethasone. The symptoms improved and the full blood counts were reversed to normal. Patient achieved a partial response and was planned for high dose therapy with autologous stem cell support. Unfortunately, during the 4th course of his treatment the patient died due to septicemia.

Comment

In some cases of multiple myeloma bone marrow contains plasmablasts with a more immature appearance usually containing a central nucleus, fine chromatin network and well visible nucleoli, less cytoplasm (hyperbasophilic) as compared to preplasmacytes and less well visible perinuclear halo. The preplasmacytes as well

as the plasmablasts present a cytoplasmic/nuclear asynchrony and numerous abnormalities; patients may often present a uniformity of a cellular subject while frequently they present an adequate heterogeneity of plasmacytic population. At times some large sized immature plasmacytes are present with multiple and numerous nuclei, abnormality of chromatin appearance, polyploidy and cytoplasmic abnormalities, with a very malignant appearance assuming the appearance of anaplastic myeloma (particular value when the plasmacyte percentage is low).

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Diagnosis: Anaplastic myeloma

ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):511
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):511

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CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Histopathology Quiz - Case 2

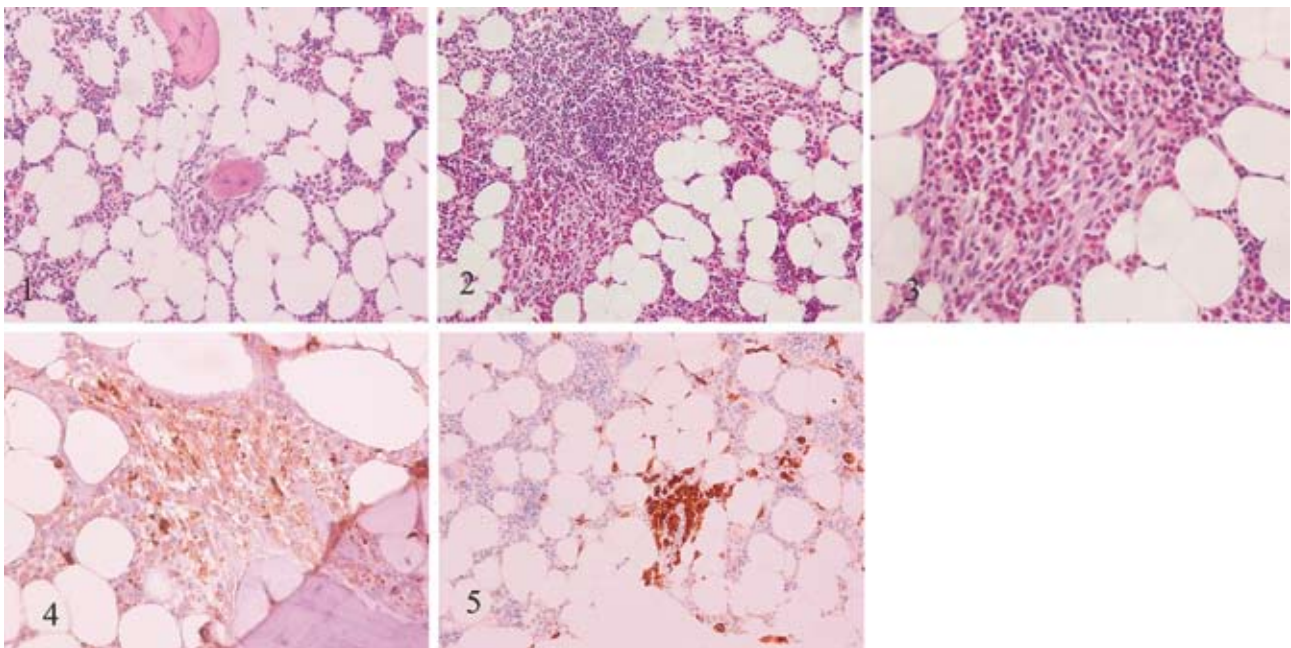
A 50 year-old male was recently diagnosed with a classical Hodgkin's lymphoma (mixed cellularity) on the grounds of a histological evaluation of a cervical lymph node biopsy. Before treatment application a bone marrow trephine biopsy was performed on the purpose of the determination of the stage of the patient's disease.

Histological examination of the bone marrow trephine biopsy showed normal cellularity (40% adipocytes). The erythroid lineage showed mild hyperplasia whereas the myeloid to erythroid ration was 3:2. The number of megakaryocytes

ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):512
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):512

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Figures 1, 2, 3, 4, 5

was in normal levels. A characteristic finding was the presence of multiple nodular, perivascular or paratrabecular lesions with significant fibrosis composed of pale, frequently spindle-shaped cells with oval or reniform nuclei (figures 1, 2). These cells were admixed with a variable number of eosinophils, lymphocytes and see-green histiocytes (fig. 3).

On Giemsa staining these spindle-shaped cells contained apparent positive granules. Moreover, on immunohistochemical grounds they were positive for CD68 (PGM1, fig. 4) and c-KIT (CD117, fig. 5) antibodies. Immunostaining for CD30 and CD15 antibodies was negative.

Comment

The diagnosis of SM-AHNMD is established when the criteria of systemic mastocytosis (defined by WHO classification 2001) are met and coexists with a clonal hematological non-mast cell lineage disorder (myelodysplastic syndrome, chronic myeloproliferative disease, acute myelogenous leukemia or lymphoma).

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Diagnosis: Systemic mast cell disease with an associated non mast-cell hematological disorder (SM-AHNMD)

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Internal Medicine Quiz - Case 2

A 62-year-old woman presented to the Emergency Department with a feeling of burn and bloat in upper abdomen, which expanded in chest and anterior neck, accompanied with dysphagia after meals, during the last month. She also states that the last 15 days she has a gorge-rise, spitting out clean saliva, which appears early in the morning, when she wakes up, for which she was prescribed initially with omeprazole, with no result, and then metoclopramide-ranitidine that reduced discomfort frequency. A generalized weakness, lack of appetite and a 5 kg loss of weight is reported during the last month. She reported no any fever or chills since symptoms appeared. The patient has not noticed any remarkable change in her defecations considering the presence of blood, the colour, the frequency or the texture. The patient's medical history is significant for osteoporosis, diagnosed before 4 years, treated by disphosphonates.

On physical examination the patient was awake and uptight. Heart rate was 98 bpm, blood pressure 150/80 mmHg, respiratory rate 18 breaths per minute, temperature 36.4 °C. The patient's heart rhythm was irregular (tachycardia, arrhythmia), no murmurs or gallops. The lungs were clear to auscultation and abdomen was soft, nondistended and painless to pressure, normal bowel sounds. Liver and spleen were normal, no tangible nodes. Skin examination was clear.

Laboratory examinations revealed normal complete blood account (CBC) and normal basic chemistry panel, except from borderline low urea, 16 mg/dL (normal laboratory's range: 17–50 mg/dL). The ECG show supraventricular systoles and LAH, but no evidence of ischemia. A laryngoscopy was normal, without remarkable findings. A radiography (X-ray) of the chest was ordered (figures 1 and 2).

An ultrasonography of the abdomen was performed (liver-spleen-pancreas-gall bladder-biliary ducts), which was normal, except from a lesion in the right hepatic lobe compatible with hemangioma (32,7×24,7 mm). The Mantoux test was negative.

CT scan of chest-abdomen showed a large, orbicular mass (8×8 cm) with definite-smooth borders and heterogeneous structure, in the anterior mediastinum, followed by an atelectasia of the left upper pulmonary lobe (figures 3 and 4). The histological examination revealed diagnosis.

Comment

Most radiologists divide the mediastinum into 3 compartments, as proposed by Felson. Anterior mediastinum is bounded anteriorly by the sternum and posteriorly by a line drawn from the anterior aspect of the trachea and along the posterior heart border. Posterior mediastinum is defined by a line that is 1 cm posterior to the anterior edge of the vertebral bodies. Middle compartment lies between anterior and posterior mediastinum. The lesions which may found in posterial mediastinal compartement are shown in figure 5.

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ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):513
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):513

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Figure 1



Figure 2



Figure 3



Figure 4

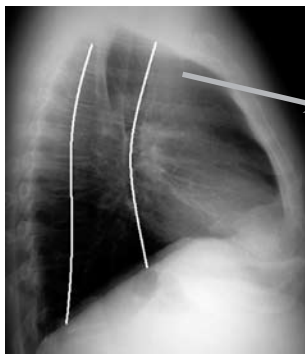


Figure 5

- Anterior mediastinum
1. Thymoma
 2. Aneurysm
 3. Angiomatous tumor
 4. Esophageal tumor
 5. Goiter
 6. Lipoma
 7. Lymphoma
 8. Morgani hernia
 9. Parathyroid tumor
 10. Pericardial cyst
 11. Teratoma
 12. Thyroid tumor

Diagnosis: Thymoma

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Internal Medicine Quiz - Case 3

An 80-year-old man was complaining of pain in the left shoulder and arm for the last 2 months. He did not report any recent traumatism or repeated mechanical strain of the shoulder joint, while he did not have any similar complaints in the past. His past medical history included mild arterial hypertension, diabetes mellitus treated with insulin, coronary artery disease and chronic obstructive pulmonary disease, both of them adequately controlled with medication. He was a former smoker, with 50 pack/years. He did not have any fever or arthralgias in any other joints and apart from weight loss of 5 kg in the last 4 months he did not report any other systemic complaints. After seeing an orthopedic surgeon NSAIDs were prescribed, which led to improvement of the pain. For the next 2 weeks he continued taking the NSAIDs according to the intensity of the pain. Eventually his left arm and hand developed weakness and his pain grew stronger and refractory to the NSAIDs, necessitating the use of codeine based analgesics. At that time the patient developed a persistent cough as well, which led him to the outpatient department of our hospital. Initial physical examination revealed tenderness on palpation and a palpable mass of the left supraclavicular fossa as well as ptosis of the left eyelid, and radiologic investigations were ordered. A chest X-ray was obtained (figures 1A and 1B). After this, a CT scan of the thorax (figures 2A and 2B) was obtained.

Comment

The chest X-ray (fig. 1A) revealed a large mass at the apex of the left lung as well as a pathologic fracture of the posterior part of the 2nd rib (fig. 1B). The CT scan of the thorax (figures 2A and 2B) showed that the mass originated posteriorly and extended to the intervertebral foramina, while its expansion through the chest cage caused the fracture of the 2nd rib.

Tumor of the superior pulmonary sulcus, with involvement of the left brachial plexus and the stellate ganglion (classic Pancoast tumor) of the left lung. Although this clinical diagnosis is usually compatible with a non-small-cell lung cancer, alternative diagnoses, such as lymphoma, primary chest wall tumors and tuberculosis must

be ruled out before treatment efforts are undertaken (induction chemoradiotherapy followed by surgical resection). A transthoracic fine needle aspiration was performed.

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ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):514
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):514

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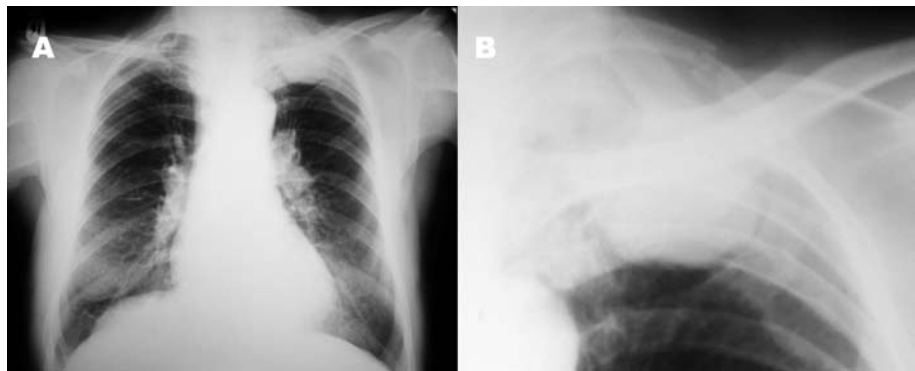


Figure 1

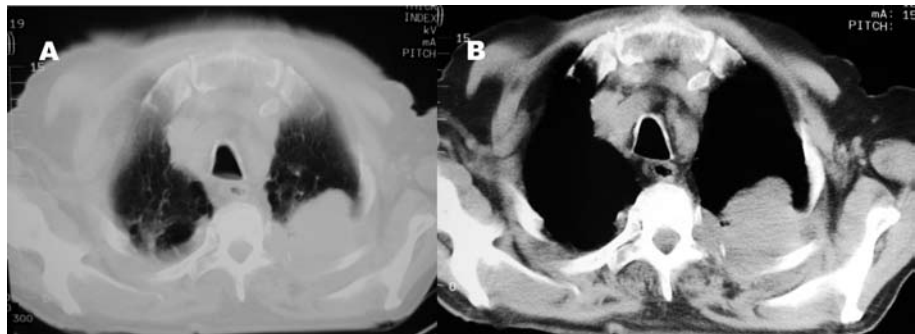


Figure 2

Diagnosis: Non-small-cell carcinoma of the left lung

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Vascular diseases Quiz - Case 2

A 56-year-old male presented to our Emergency Department with nausea and anuria of acute onset. On physical examination flank pain, more intense on the left side was found. Laboratory investigation revealed a hemoglobin (Hb) of 13 g/dL, white blood cell (WBC) count of $9 \times 10^9/L$, platelet (PLTs) count of $252 \times 10^9/L$ and serum creatinine (Cr) of 6.1 mg/dL. His past medical history included a type III thoracoabdominal aortic aneurysm (TAAA) extending from the mid-portion of the descending thoracic aorta to the level of the left renal artery, an infrarenal abdominal aortic aneurysm (AAA) and chronic renal insufficiency (baseline serum creatinine: 2.1 mg/dL) secondary to right renal artery chronic occlusion and 95% left renal artery stenosis (fig. 1).

Comment

Based on patient's medical history and presentation, either dissection or rupture of the TAAA was at first suspected. Therefore, the patient was subjected immediately to thoracic and abdominal CT scan, which however did not reveal any signs of aortic dissection or rupture.

Subsequently, total thrombotic occlusion of the 95% stenosed solitary left renal artery was assumed that was confirmed by Duplex ultrasonography. The patient was subsequently transferred to the endovascular suite, where endovascular recanalization and stenting of the occluded renal artery was performed successfully (fig. 2). Urine output was restored immediately after the procedure, and serum creatinine level was reduced to 1.6 mg/dL, 10 days postoperatively.

Thrombosis of a stenotic renal artery leading to total occlusion and acute renal failure should always be suspected in a patient with a solitary functioning kidney that presents with anuria of acute onset. After confirmation of the diagnosis with ultrasonography, attempts for renal blood flow restoration should be performed immediately. However, recanalization of a totally occluded renal artery may not be always successful and especially in patients with a solitary functioning kidney the occlusion can lead to irreversible renal failure. To avoid such a devastating result, solitary renal artery stenosis should be managed more aggressively in comparison with unilateral renal artery disease. Indeed, to date, percutaneous revascularization is strongly recommended for patients with renal artery stenosis in a solitary functioning kidney (Class IIa, Level of Evidence: B, ACC/AHA 2005 Practice Guidelines).

It should be highlighted that anti-hypertensive treatment with angiotensin converting enzyme (ACE) inhibitors could have also been the cause of acute renal failure and anuria in our patient. However, he denied being on any ACE inhibitor, despite hypertension. Angiotensin II receptor blockade (ARB) could have also caused acute renal failure to our patient, despite the fact that these agents are considered generally safer than ACEs in this setting. Although acute renal failure induced by ACEs or ARBs is commonly reversible, both these antihypertensive agents should be avoided in patients with renal artery stenosis of a solitary functioning kidney. Additionally, every patient with a solitary functioning kidney that presents with acute anuria

ARCHIVES OF HELLENIC MEDICINE 2007, 24(5):515
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2007, 24(5):515

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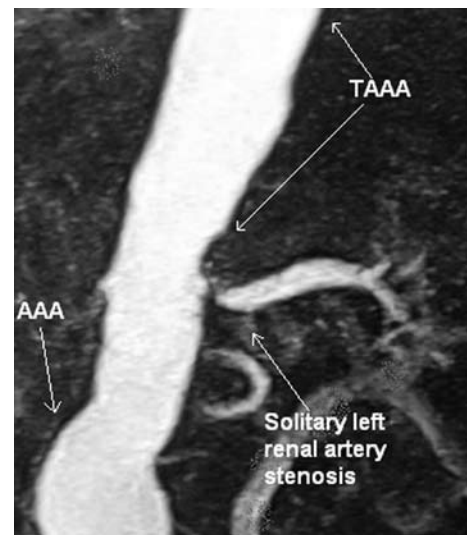


Figure 1. Computed tomography angiogram demonstrating the TAAA, AAA and solitary left renal artery stenosis.



Figure 2. Digital subtraction angiography demonstrating solitary left renal artery thrombosis (A) and recanalization after angioplasty and stenting (B).

should be investigated for ACE inhibitor/ARB intake.

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Diagnosis: Acute anuria due to solitary renal artery occlusion
