A 59 year-old female, with unremarkable medical and surgical histories except an amigdalectomy in her childhood. She didn’t take any prescription or over-the-counter medications. 6 days before the admission presented a left-sided pleuritic-like pain with subsequent radiation to the left hemi-abdomen, presenting fever 48 hours later, and developing dyspnea with minimum effort 72 hours after the fever onset, being this the main reason for presenting to the Emergency Department.

On physical examination, the patient was conscious, oriented and receptive, she was of moderate build and appeared healthy and well hydrated, with a plum-colored malar flush. She was in sinus tachycardia, with a heart rate of 110 bpm, with no murmurs or added sounds, and her blood pressure was noted to be 140/80 mmHg. In the pulmonary auscultation, the vesicular murmur was diminished in 1/3 of the left lower hemithorax. A doubtful abdominal mass occupying left flank and left hypochondrium exceeding sagittal medial line and leaving the
left iliac fossa was palpated; rest of the abdomen without other findings; neither hernias nor adenopathies.

The initial work-up included laboratory investigations without any clinically significant results except for the urine analysis which developed the presence of 20-30 leukocytes/field. Basal arterial gasometry: pH: 7.47; PO$_2$: 56 mmHg; PCO$_2$: 29.4 mmHg; HCO$_3$: 21.3 meq/l; BE: -1; SaO$_2$: 91.6%. A chest x-ray was performed showing an elevation of the left hemidiaphragm with left costophrenic sinus obliteration, and an air-fluid level in the gastric chamber was observed (Figure 1). The abdominal x-ray showed a calcium-density image insinuating the iliopectineal ligament and dilated colonic haustra (Figure 2).

She was admitted to the internal medicine ward with the diagnosis of respiratory insufficiency and urinary tract infection, and a computer tomography scan was ordered, showing an elevation of both hemidiaphragms with a minimal pleural effusion (Figures 3 and 4) accompanied by a small atelectasis. In the abdominal study a large mass was easily observed, with maximum diameters of 17 cms (cephalocaudal), 16 cms (anterior-posterior) and 12.1 cms (transverse). This mass presented well formed partially calcified walls and contacted with the left hemidiaphragm, to which it displaced and compressed. An air-fluid level with plenty of gas bubbles and heterogeneous content with predominance of low density areas could be visualized inside. Hence, everything indicated the presence of an infected cyst, probably placed in the spleen, with the morphological appearance of a hydatidic cyst. With these findings, the patient was admitted to the Department of Surgery.

Two days later new laboratory results were received, emphasizing a high IgE (1190 U/ml) and PCR levels (356.7 mg/dl) and broad-spectrum antibiotic prophylaxis with Imipenem (1g/6hs) was implemented. Three days later the cyst was punctured and aspirated under CT scan control with a thick needle obtaining a foul-smelling fluid and in the same procedure a 12 F pig-tail drain was placed, evacuating between 50 and 500cc/day. Culture of the discharging material grew a *Streptococcus* sp. and *Echinococcus granulosus*.

In a control CT scan performed 4 days later the cyst had maximum diameters of 15 cms (cephalocaudal), 14 cms (anterior-posterior) and 10.5 cms (transverse).
3 days later a surgical programmed intervention was performed (10th day from admission). A left subcostal laparotomy revealed a 15 x 10 cms infected hydatidic cyst located in the lower pole of the spleen, containing foul-smelling pus, hydatidic vesicles and gas. This cyst presented numerous soft adhesions to epiploon, stomach and transverse colon. These adhesions were easily liberated and no splenectomy was needed, and a 2/3 cystpricystectomy was performed and a drainage was placed into the area. No postoperative incidents were reported and the patient was discharged with no further problems.

**DISCUSSION**

Hydatid disease caused by the genus *Echinococcus* is endemic in Eastern Europe, Middle East, South America, North Africa, Indian subcontinent, Australia and New Zealand. *Echinococcus granulosus* is the commonest organism involved, with dogs usually as the definitive host and human beings acting as an accidental intermediate host.

Enzymatic digestion of the shells of the eggs (shed in the faeces of the dogs) in the duodenum of the intermediate host releases the embryonic forms. These migrate through the intestinal wall to enter the portal circulation, from where they make their way towards the liver or lung, developing a triple cystic covering which grows at a rate of about 1 centimeter a year.²

Although hydatid disease is one of the oldest diseases known to man, the incidence of exclusive splenic location is very low, constituting 0.5 to 4% of all cases of hydatidosis,³ whereas the liver (70%) and the lung (15%), are the commonest settlements. The rare sites include heart,⁴ thyroid,⁵ breast,⁶ central nervous system,⁷ etc. Hydatid cyst is the only parasitic cyst of the spleen and it is said to be twice as common as the non-parasitic variety. Dieulafoy (1898) classification divides splenic hydatid cysts into ascending (immobile) and descending (mobile) types and into cortical, central and juxtasplenic types as per location of the cyst, being the abdominothoracic type added by Scherb (1904). Segond and Potherat divided hydatids into three groups: (a) those containing much fluid but few daughter cysts, (b) those packed with vesicles and (c) suppurating cysts. The largest cyst ever reported was in Australia and it contained 57 litres of fluid.⁸

A hydatid cyst consists of three layers. The outermost layer is the adventitia (pseudocyst), consisting of fibrous tissue as a result of the granulomatous reaction of the liver to the cyst, from which it is inseparable. The middle layer is the laminated membrane (ectocyst), which is formed of the parasite itself, and contains a crystal clear fluid if it is not superinfected. The hydatid fluid is antigenic and highly toxic, and can cause a potentially fatal anaphylactic reaction in humans. The innermost layer is the germinal epithelium (endocyst), being the only living part of the hydatid cyst and secreting the hydatid fluid internally and the laminated membrane externally, producing the new generations of parasites.

Although various hypothesis about the possible routes followed by the parasite in order to reach the spleen have been formulated, including vascular and lymphatic dissemination, reflux into spleen from portal vein, through haemorrhoidal veins, through small veins passing from the splenic flexure of the colon to the lower pole of the spleen, via arterial blood etc, the most logie route seems to be escaping of 10-15% of embryos from liver and lung filters into general circulation, a fraction of which settle down in the spleen.

The first clinical indication of the presence of splenic hydatid disease usually is an accidentally discovered mass in the abdomen mostly in left hypochondrium and less frequently in the epigastrium, pain which is usually a dull dragging ache, dyspepsia, constipation due to pressure on colon, hypertension due to left renal artery compression, and dyspnoea due to pushing up of the left diaphragm,⁹ however up to 30% are incidental findings in asymptomatic individuals.¹⁰ Other forms of clinical presentation include the rupture of splenic echinococcal cyst with anaphylactic shock¹¹ traumatic or not, acute abdomen,¹² rupture of longstanding splenic hydatid cyst into bronchial tree,¹³ or even a splenic hydatid cyst established fistula to the colon.¹⁴ Laboratory evaluation of patients with hydatid disease often yields non-specific data, being eosinophilia (above 3%) not significant in endemic areas. Elevated levels of IgE levels are a non-specific indicator of prior sensitization or active infection with parasitic organisms, while elevated levels of IgM classes specific to echinococcal organisms may be a sensitive indicator or recurrent disease.¹⁵ Serum immunoelectrophoresis is currently the
most reliable, with a sensitivity of approximately 90%, with 1 year positivity after the organism has been eradicated. The Casoni intradermal skin test, indirect hemagglutination and ELISA had also been used.

Image techniques have pushed biochemical into the background in the diagnosis of this disorder, and despite the fact that the diagnosis of splenic hydatidosis is difficult or impossible by conventional radiography alone, if the cyst is calcified hydatidosis may be suggested. Plain radiology of abdomen can reveal a soft tissue shadow with or without calcification, displacement of left diaphragm upwards, stomach to the right and transverse colon with splenic flexure downwards. Ultrasound and computed tomography (alone or in combination) established the definitive diagnosis of splenic hydatidosis in almost all the cases, being the echography cost effective and particularly valuable for follow up screening. CT is more accurate than ultrasound in localizing and delineating extent of the cyst.

Regarding to the medical treatment, Mebendazole (60 mg/kg/day for 6-24 months) or Albendazole (10 mg/kg/day for 6 months) can only complement surgery but cannot replace it, though in the last twenty years the need of systematic splenectomy has been critically reduced, avoiding overwhelming post splenectomy infection. Besides, though not in this case due to the apical location of the cyst, the laparoscopic surgery has an important and current role in this issue.16,17

The didactic interest of this case is based on the final diagnosis of a large splenic hydatid cyst in a patient who was admitted to the Emergency Department presenting unusual and unspecific clinical signs but showing suspicious findings in the first chest radiography performed being confirmed by spectacular abdominal CT scan images. In the beginning these images induced confusion among experimented clinicians due to their great similarity with pathologic abdominal processes like the retention stomach or the empty viscera covered perforation. Subsequently, the diagnosis of a large infected hydatidic splenic cyst was confirmed, being this issue considered common in our environment but very unfrequent when the affected organ is spleen.

It is important to underline that it is relevant to maintain hydatid disease and its possible complications as one of the differential diagnosis to consider in endemic areas when the clinical condition justify it, being compulsory the prevention against this parasitation by Public Health.

REFERENCES