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RESEARCH ARTICLE

ACUTE SURGICAL ABDOMEN: UNEXPECTED MEDICAL CAUSE

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Abstract

Acute surgical abdomen is a diagnostic and therapeutic emergency, manifested by a generalized contracture of the abdominal muscles following a peritoneal irritation. In the majority of cases, acute peritonitis is the most common etiology requiring urgent surgical intervention. Nevertheless, some medical pathologies, notably acute adrenal deficiency and inflammatory colitis, can mimic this surgical situation, leading to an aberrant surgical intervention. Through a clinical case, we report the diagnostic and therapeutic modalities of a multifocal tuberculosis initially admitted for an acute surgical abdomen.

Introduction:-

Multifocal tuberculosis is a rare entity especially on immunocompetent person. All organs can be affected and it is defined by the involvement of at least two extra-pulmonary sites associated or not with a pulmonary invasion. [1-2]

In front of the clinical polymorphism, the insidious manifestations, the similarity to several pathologies as well as the difficulty to have a microbiological certainty, its diagnosis presents real problems delaying the setting up of an effective treatment. [3]

We report the case of a young immunocompetent patient admitted for an acute surgical abdomen and whose radiological investigations, especially computed tomography CT, had revealed a tubercular pulmonary miliary, leading to the suspicion of multifocal tuberculosis with intestinal involvement, this was confirmed by an ileocoloscopy with biopsy and anatomopathological study, and kidney involvement was strongly suspected on the basis of a search for Koch's Bacillus in the urine.

Patient Information:
A 23 years old student, without a history of tuberculosis contagion or previous surgery, presented with an abdominal pain and asthenia, associated to diarrhea and constipation with episodic vomiting, along with a fever history, cough, night sweats and weight loss over two months.

Clinical findings:
Physical examination revealed a cachectic patient with extracellular dehydration. On the hemodynamic level, the patient was stable with a systolic blood pressure of 122 mmhg and diastolic blood pressure of 69 mmhg, tachycardia

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at 134 beats per minute, afebrile at 37.2 C, with a blood glucose postprandial level of 2.04 g/l. Pulmonary examination showed a polypnea with a respiratory rate of 35 round per minute. Regarding to abdominal examination, it revealed a general abdominal contracture with a high level of awareness and pain. On rectal examination, the fingertips only brought back material.

No signs of focalization, neither stiffness of the neck was found.

Diagnostic assessment:
In front of this surgical abdomen, a peritonitis was initially evoked, which pushed us to complete the diagnostic approach while ensuring the initial conditioning of the patient including: dorsal recumbency, peripheral venous catheter, cardio-respiratory monitoring, putting on oxygen and a nasogastric probe. Laboratory tests revealed hypochromic microcytic anemia with haemoglobin at 0.8 g/l, lymphopenia at 870/mm3, with a leukocyte value at the norm. Fluid and electrolyte imbalance were revealed including hyponatremia, hypokalemia, hypochloremia and metabolic acidosis with an increased anion gap that complicated the acute renal failure with urea and creatinine values of 2.68 g/l and 65.71 mg/l respectively. The hepatic assessment was without any abnormality of cytolysis or cholestasis. But the patient presented a significant biological inflammatory syndrome with a CRP of 320 mg/l and procalcitonin > 100. For the phospho-calcium balance, we noted a hypocalcemia with normal phosphoremia.

An ultrasonography was performed immediately which allowed us to eliminate a peritonitis as it is the first diagnosis to be evoked in front of a surgical abdomen, although it shows a bilateral homogeneous hypertrophy of the kidneys without dilatation of part or all the calyceal system with normal size of kidneys (139 x 62 mm) for the right one and (146 x 73) for the left one.

The Scan view showed a scissuritis on the right lung. (figure 1)

Abdominal and chest computed tomography clearly showed a collection of tiny discrete pulmonary millimetric opacities in the both lungs, as far as bilateral kidneys hypertrophy, several abdominal adenopathies, ascitis and low-grade pleurisy. (figure 2)

Acute adrenal deficiency was suspected and ruled out by cortisolemia.

Because of the patient's origin, the clinical findings and also the diagnosis assessment a miliary tuberculosis with intestinal and urogenital involvement was obviously evoked.

To complete the diagnosis approach, a Tuberculin skin test was negative, as the Koch's bacillus in the sputum was negative too but the urine culture was definitely positive.

Ileocoloscopy with anatomopathological study of the biopsies confirmed the diagnosis of intestinal involvement by showing the presence of epithelia gigantocellular granuloma with caseous necrosis.

Meningeal involvement was eliminated by performing a lumbar puncture which came back without any abnormality. HIV serology was negative.

Therapeutic intervention:
Initial treatment consists of fluids and electrolyte resuscitation by vascular filling with saline and bicarbonate serum, a probabilistic antibiotherapy with appropriate dose to the kidney function, antituberculosis chemotherapy with four drugs and eventually corticosteroid drugs at rate of 40 mg three times a day.

Follow-up and outcome of interventions:
Following the initiation of these therapeutic fluids and drugs, his pyrexia resolved, his respiratory state improved, also we have noted the sanitation of his heart frequency, but most importantly the regression of his pain. Moreover, the resuscitation of the patient allowed to correct his hyponatremia, acidosis, in addition to the potassium recharge which normalized his hypokalemia, all that allowed the clear improvement of his kidney function which was in progressive decline and he was finally discharged from the hospital 1 week after.

The patient's check-up that we scheduled one month after noticed a good weight gain, the regression of the anemia and the improvement of the intestinal absorption.

Discussion:-

Tuberculosis is a leading cause of preventable morbidity and mortality due to an infectious agent worldwide. Primarily, the disease involves the lungs and, at times, distant blood-borne spread results in the development of extrapulmonary tuberculosis. Infrequently, intense systemic dissemination from the rupture of a Mycobacterium tuberculosis-laden focus into a vascular channel result in a morphologically characteristic form of disease known as miliary tuberculosis. [4]

Intestinal involvement is often unrecognized, this location must be systematically sought especially if there is abdominal pain with signs of tuberculosis impregnation. It represents the 6th extra-pulmonary localization of tuberculosis [8] while kidney tuberculosis represents the second localization after lymph node involvement.[7] The intestinal form, which is less well described in the literature, is associated with pulmonary tuberculosis in 20-30% of cases, a location that guides and facilitates the diagnosis. [9-10]

The pathophysiology of this intestinal involvement has been attributed to several mechanisms at first to hematogenous dissemination of BAAR from an active pulmonary site, or ingestion of infected sputum and milk or Contamination by contiguity from adjacent affected organs. [5] Concerning the kidney tuberculosis most often results from hematogenous dissemination by glomerular passage of the Koch's bacillus in 25 to 65%.[6] It affects men preferentially with a clinical latency that can lead to significant destruction of the urinary tract. [12]The diagnosis of miliary tuberculosis can be difficult as the clinical manifestations can be atypical and non-specific. The onset is generally insidious with a variable latency period according to the series, that was 82 days in the series of Collado. [11]

Clinical signs are dominated by abdominal pain, observed in 82 to 88% of cases in the literature. Acute pseudo surgical forms have also been described, also appendicular and sub occlusive syndromes are noted in 14-18% of cases, respectively. [14]Much more rarely, the disease can be revealed by an intestinal perforation which would be secondary to the caseous necrosis of intra-parietal tubercular nodules. [5]

In our case, in addition to intestinal tuberculosis, our patient had also kidney failure, the mains symptoms are functional urinary signs such as dysuria and hematuria. It should be realized that a large proportion of patients who are found to have renal tuberculosis do not, in fact, present with characteristic urinary tract symptoms. General signs such as fever, chills, sweats, asthenia are sometimes encountered, but some patients may be asymptomatic which makes the diagnosis all the more difficult. [6]

Concerning the laboratory testing, a number of hematological and biochemical laboratory abnormalities have been described in miliary tuberculosis but their significance is controversial.[15]Standard biological testing has an important role in the diagnosis of intestinal tuberculosis. The presence of lymphopenia associated with a biological inflammatory syndrome (increased SV and CRP) is suggestive. It is necessary to look for deficiency anemia, hypoalbuminemia, hypocalcemia, decreased prothrombin level, deficiency of lipo and water-soluble vitamins. [8]A renal function failure may also be complicated by metabolic acidosis with increased anion gap in addition to hypocalcemia and hyperphosphatemia. [6]A positive tuberculin skin test or interferon-gamma release assays result only indicates infection with mycobacterium tuberculosis and does not indicate active disease [15]But its negativity does not eliminate the diagnosis. The microbiological diagnosis consists of a search for BAAR by Ziehl-Neelsen or auramine staining, or by culture in Lowenstein medium or MGIT liquid medium on fresh morning urine. Due to low sensitivity, the sample must be repeated 3 to 6 days in a row. It may take up to 6 weeks for the culture result to arrive. The use of PCR detection of M.tuberculosis DNA in urine increases sensitivity and diagnosis. [6-13]

Imaging modalities, such as, chest radiograph, ultrasonography, computed tomography, magnetic resonance imaging have been used to define the extent of organ involvement and evaluating response to treatment.
Miliary pattern on chest radiograph, the hall mark of miliary tuberculosis, is seen in majority of patients. Sometimes it can be normal initially and the typical miliary pattern may evolve over the course of disease evolution. Thus, periodic chest radiographic examination should be done in patients with suspected miliary tuberculosis during the

course of their diagnostic evaluation. [15]Ultrasonography can facilitate partially detection of focal lesions, in particular, renal tuberculosis should be suspected in the presence of images of bilateral renal hypertrophy with calcific irregularity of papillary necrosis, infundibular stenosis, scarring or calcification of the renal parenchyma, ureteral stenosis/dilatation, or in the presence of thickening of the bladder mucosa.[6-7-13]Ileocoloscopy remains the best examination for the diagnosis of intestinal tuberculosis.[5]As Tuberculosis can affect the entire gastrointestinal tract with a predilection for the ileocaecal region. This predominant location could be explained by physiological stasis, the high power of digestive absorption and the abundance of lymphoid tissue in this region. The lack of specificity of clinical signs and endoscopic lesions explains the difficulty of diagnosis which may lead to confusion with other diseases such as Crohn's disease (CD), cancer, amebiasis, yersiniosis and periappendicular abscess. Thus, it is imperative to perform multiple ileocolic biopsies (8 to 10), on the edges of the ulcers, in search of epithelioid and gigantocellular granulomas associated with caseous necrosis which represent the key to the diagnosis. [5]

According to WHO recommendations, the treatment of urogenital tuberculosis consists of four-drug therapy, sometimes combined with surgical management. [6]The treatment regimen is the same as that for pulmonary tuberculosis as well as for intestinal one. The recommended protocol in our country is based on a daily four-way therapy of 2 months made of rifampicin, isoniazid, ethambutol, pyrazinamide for 2 months, followed by a dual therapy of rifampicin and isoniazid for 4 months. In case of renal insufficiency, the doses of ethambutol and pyrazinamide must be adapted. However, the total duration of treatment is not agreed upon, as in practice a prolonged treatment is frequently observed in case of urinary tuberculosis, as it will decrease the relapses. [6]

Conclusion:-

Before any acute surgical abdomen, and after elimination of acute peritonitis, it is necessary to consider evoking the frequent and unknown medical causes, particularly intestinal and peritoneal tuberculosis, by looking for the insidious onset of symptoms and the existence of general signs of tuberculosis, which will guide the radiological, endoscopic and microbiological investigations.

Take home message:

1. Before proceeding to a surgical intervention, think about the medical causes.
2. Never refer to the first obvious diagnosis.

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Competing interests:

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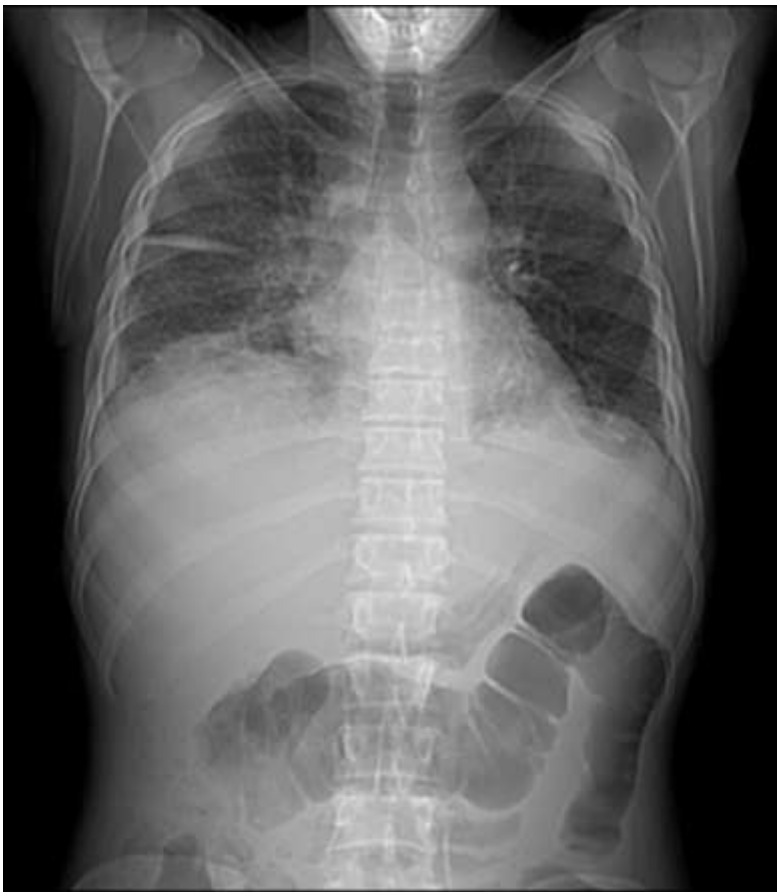

Figure 1:-

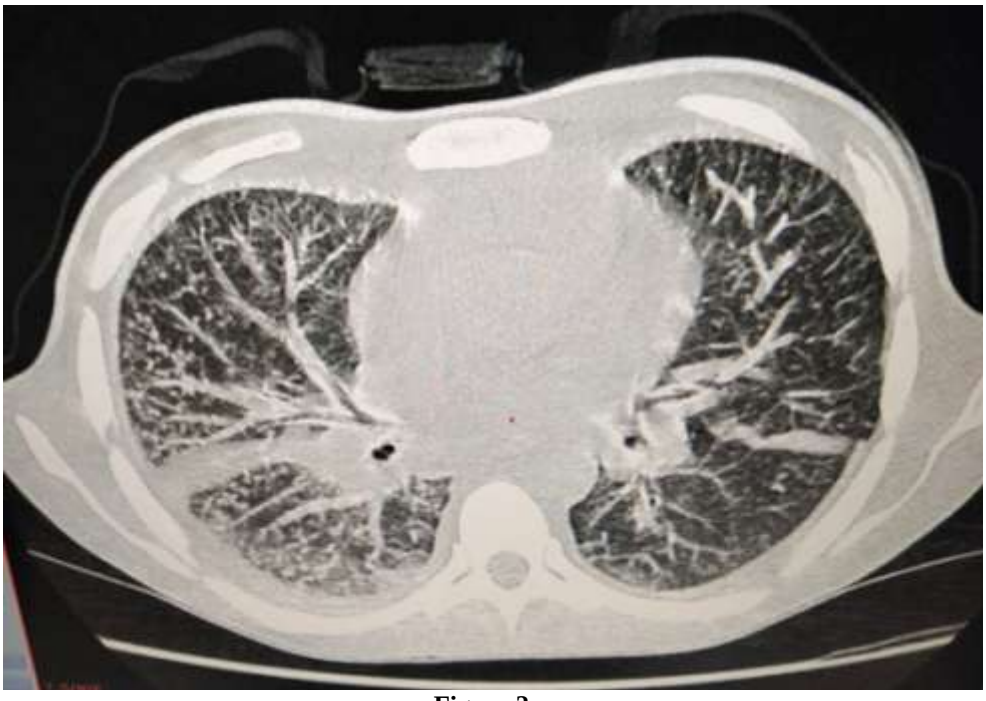

Figure 2:-

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