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Tubercular bowel perforation: what to do?

Bağırsak tüberkülozu perforasyonu: Ne yapmalı?

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BACKGROUND
The incidence of abdominal tuberculosis (TB) is increasing in western and developed countries. This pathology has several complications, including free intestinal perforation. The aim of this study was to analytically summarize all the pertinent literature discussing the various treatments for TB-related perforations.

METHODS
We reviewed the patient database of the Emergency Surgery Department of the Bologna University Hospital, checking the last 13 years. A retrospective review was conducted of all reported cases of intestinal perforation due to intestinal TB published through 3 March 2009.

RESULTS
119 cases of abdominal TB presenting with intestinal perforation were published. There are no standardized guidelines regarding the surgical treatment. Of the 119 reported cases, 40 (33.6%) were treated with resection and anastomosis, 17 (14.2%) with direct sutures, 4 (3.3%) with a simple drain, and in 57, the treatment was not reported.

CONCLUSION
No clinical evidence has been available for analysis to discern the optimal surgical strategy for treating intestinal perforations induced by TB. The direct closure of the perforation typically correlates with poor morbidity and mortality results. The better treatment seems to be the surgical resection of the perforated part with anastomosis. However, pharmacological therapy remains the essential pillar of treatment.

Key Words: Perforation; surgery; treatment; tuberculosis.

AMAÇ
Abdominal tüberküloz (TB) insidansı, Batı ülkeleri ile gelişmiş ülkelerde artmaktadır. Bu patoloji, serbest intestinal perforasyonu da içeren birkaç komplikasyona sahiptir. Bu çalışmanın amacı, tüberkülozla ilişkili perforasyonlara yönelik çeşitli tedavileri tartışan ilgili tüm literatürü analitik olarak özetlemektir.

GEREÇ VE YÖNTEM
Bologna Üniversite Hastanesi Acil Servis Departmanı'nın son 13 yıldaki kayıtlarını sorgulayan hasta veritabanı gözden geçirildi. 3 Mart 2009 tarihine kadar bildirilmiş olan intestinal TB'ye bağlı raporlanan bütün intestinal perforasyonu olguları geriye dönük olarak değerlendirildi.

BULGULAR
İntestinal perforasyon ile başvuran 119 abdominal tüberküloz olgusu yayınlanmıştır. Cerrahi tedavi ile ilgili standardize kılavuzlar bulunmamaktadır. Bildirilen 119 olgudan, 40'ı (%33,6) rezeksiyon ve anastomozla, 17'si (%14,2) doğrudan dikişlerle, 4'ü (%3,3) basit bir drenle tedavi edilmiş ve 57 olgunun tedavisi de rapor edilmemiştir.

SONUÇ
TB'ye bağlı bağırsak delinmelerinin cerrahi tedavisi için en uygun stratejiyi analizle ayırt edilecek hiçbir klinik kanıt saptanmadı. Perforasyonun doğrudan kapatılması, tipik olarak yüksek morbidite ve mortaliteyle korelasyon göstermektedir. Perfore kısmın rezeksiyonu ile birlikte anastomoz gerçekleştirilmesi daha iyi tedavi gibi görünmektedir. Yine de, farmakolojik tedavi, tedavinin esas ayağı olarak kalmaya devam etmektedir.

Anahtar Sözcükler: Perforasyon; cerrahi; tedavi; tüberküloz.

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Tubercular infection can involve the chest as well as many parts of the human body, namely the abdomen, peritoneum and bowel. Although the entire gut can be involved, the ileocecal area is most commonly affected.[1-3] Abdominal tuberculosis (TB) is the sixth most frequent extra-pulmonary location[3,4] and it is relatively rare in the industrialized world. On the other hand, the incidence of abdominal TB is increasing in western and developed countries due to immigration from developing countries, aging populations, increasing incidence of human immunodeficiency virus (HIV) infection, and misdiagnosis with ineffectual treatment.[4,5] For these reasons, abdominal TB represents an interesting challenge for surgeons in developed countries as well.

Abdominal TB typically presents itself in three major forms as well as with several less common symptoms: (I) the ascitic type, (II) the plastic type, which causes intestinal obstruction, and (III) the glandular type, which involves the mesenteric nodules. It is less common to observe tuberculous strictures, nodules, fistulae, or an interconnected association of these manifestations.[3,6]

There are several complications involving intestinal TB, including bowel obstruction (31.7%), intestinal perforation (4.9%), enterocutaneous fistula (2.4%), and small bowel volvulus resulting from mesenteric lymphadenitis (2.4%).[7] Different studies typically denote different percentages for these complications.[8] Free intestinal perforation is an uncommon complication of intestinal TB due to a reactive thickening of the peritoneum and subsequent adhesion formations with surrounding tissues.[9] It accounts for 1-10% of abdominal TB cases and has a poor prognosis, with a mortality rate higher than 30%.[10-12]

To date, there has been no clinical evidence available to assess the best surgical means to treat intestinal perforations due to TB. The main cause of such unavailability is the extremely rare nature of this complication.

MATERIALS AND METHODS

We reviewed the patient database of the Emergency Surgery Department of the Bologna University Hospital, checking the last 13 years (1995-2008).

Then, a retrospective review was conducted of all reported cases of intestinal perforation due to intestinal TB published through 3 March 2009. On PubMed (1966-2009), using the key words "tuberculosis and intestinal perforation", we found 216 articles. For purposes of practicality, we excluded from the compilation process 49 different articles that were written in languages other than English, Italian, Spanish, or French. Of the remaining articles, 103 were excluded because they did not address the topic of interest. In the end, we definitively considered 54 articles (25%), including case reports, case series and reviews. In particular, we searched for the methods used to diagnose and treat tuberculous intestinal perforation.

RESULTS

Case Report

In the analysis of the patient database of our Unit, we found 5 cases of abdominal TB, only one (20%) of which presented with perforation.

A 53-year-old woman from Eritrea was admitted to the Department of Internal Medicine four months prior with a one-month history of minor epigastric pain, progressive abdominal distension, and hyperpyrexia. On her physical examination, the patient looked ill and febrile (temperature 38.8°C). Abdominal examination showed no evidence of peritonitis, and her blood profile showed hemoglobin 11.5 g/dl, leukocyte count 9.0 x 10^3/uL (neutrophils 85.5%), ESR (erythrocyte sedimentation rate) 83 mm/hr, Ca-125 585 u/ml, and albumin 2.5 g/dl. All hepatitis markers were negative, and all liver and renal function tests and electrolytes appeared normal. Chest radiography assessments were normal and esophagogastroduodenoscopy (EGDS) showed only a sliding hiatal hernia. Colonoscopy was negative up to the hepatic flexure. An abdominal ultrasonography (US) scan detected ascites without hepatopathies or portal hypertension, and an evacuative paracentesis of about 4 liters (L) was consequently performed. A cytologic examination of the ascitic fluid showed normal mesothelial cells and rare granulocytes, neutrophils, lymphocytes, and histiocytes, followed by a negative microbiological exam for bacteria and mycobacteria. The computed tomography (CT) scan showed pleural bilateral effusion, ascites and enlarged mesenteric lymph nodes (Fig. 1). The Mantoux intradermic reaction was negative. Treatment was thereby commenced with an empirical antibiotic therapy using third-generation cephalosporin after hemoculture tested positive for *Staphylococcus haemolyticus*, and a slight improvement in the pa-

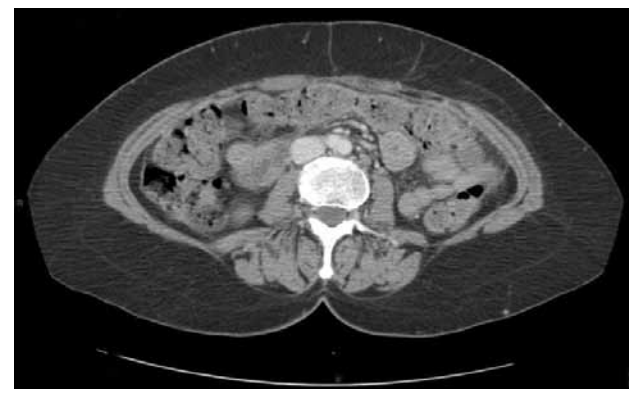

Fig. 1. CT scan shows mesenteric lymph node enlargement.

Fig. 2. Jejunum-colic fistula (frontal view).

tient's general subjective condition was observed. The patient was then discharged one month later with the provisional diagnosis of "unknown-origin ascites".

Three months later, the woman was readmitted to the hospital with hyperpyrexia, asthenia, weight loss, ascites, and night sweating. Her blood profile showed hemoglobin 8 g/dl, leukocyte count 16.98 x 10^3/uL (neutrophils 83.3%), ESR 24 mm/hr, and albumin 2.3 g/dl. A chest X-ray revealed right pleural effusion while the US abdominal scan showed ascites. A barium meal revealed a jejunum-colic fistula when a portion of the contrast meal seemed to precociously enter the ascending part of the colon (Fig. 2). A complete colonoscopy, performed to verify the hypothesis of an inflammatory bowel disease (IBD), yielded three nodules covered by normal mucosa. A new Mantoux intradermic reaction was performed, and it showed negative results. The day after the contrast meal, the patient exhibited an acute abdomen with signs of peritonitis. A CT scan was then performed which showed barium in the abdominal cavity.

The patient was then transferred to the emergency surgery unit where a laparotomy was performed. During the laparotomy, about 1 L of fecaloid fluid was drawn from the abdominal cavity. The entire visceral and parietal surfaces of the peritoneum were full of yellow-white nodules (ranging from 1 to 5 mm in size) (Fig. 3). A biopsy was taken. The entire cavity was affected by the infection; the small bowel was firmly attached to the omentum and adhesiolysis was consequently performed to free the stomach, right and left colon, rectum, and uterus, including Fallopian tubes and ovaries. Although all these structures were affected by the inflammatory process, no perforation was evident. As checking for a perforation could have been difficult and dangerous, the dissection was stopped without probing for evidence of perforation. A thorough washing of the entire abdominal cavity was performed, five drains were placed, and the abdomen was closed. A few days later, the histopathological examination of the nodules revealed a chronic flogistic granulomatous process with a giant-cellular reaction (Fig. 4). Although an official diagnosis of tubercular peritonitis had not yet been confirmed, treatment commenced with an ex-juvantibus anti-tubercular therapy (pyrazinamide 5 mg po three times a day, isoniazid 300 mg IV once a day, rifampin 600 mg IV once a day, and ethambutol 500 mg IV twice a day) alongside a total parenteral nutrition (TPN) regimen.

One week after beginning the treatment therapy, a new Mantoux intradermic reaction yielded a positive result. Three weeks after the surgical intervention, an

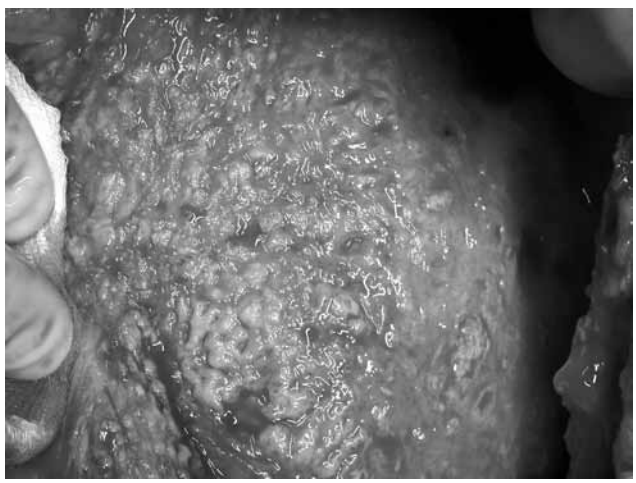

Fig. 3. Yellow-white nodules covering the entire peritoneal (parietal and visceral) surface.

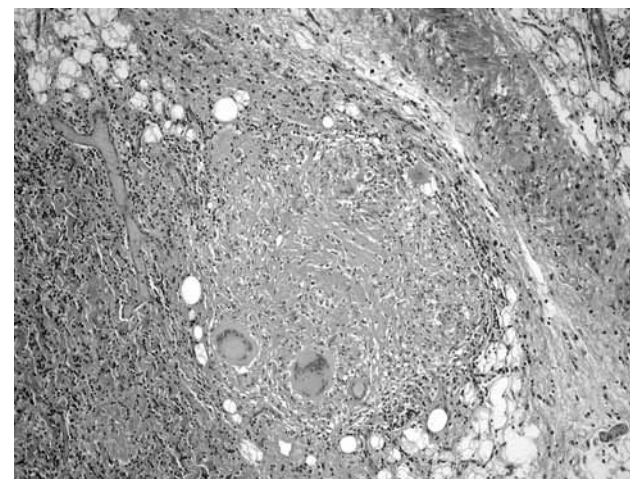

Fig. 4. Histology: chronic flogistic granulomatous process with a giant-cellular reaction.

Table 1. Summary of all published papers

Authors	No. of patients with abdominal TB	No. of patients with intestinal perforation	Site of perforation	Diagnosis	Treatment	Outcome
Sweetman WR (1958) [50]	70	20	NR*	Laparotomy - histology	12 simple suture 2 drain 6 NR*	15 survived 5 died (suture group)
Gleason T (1979) [51]	49	1	1 duodenum	Laparotomy - histology	1 simple suture	1 survived
Ecgleston FC (1983) [52]	137	21	15 ileum 3 jejunum 3 multiple	Laparotomy	16 resection + anastomosis 5 simple suture	NR*
Gilinsky NH (1986) [53]	52	7	4 ileum 2 colon 1 appendix 1 rectum	Laparotomy - histology	3 simple suture 4 resection + anastomosis	6 survived 1 died (suture group)
Kapoor VK (1986) [43]	45	6	4 ileum 2 not found	Laparotomy - histology	4 resection + anastomosis 2 simple drain	6 survived
Dorè P (1990) [54]	2	2	1 ileum 1 colon	Laparotomy - histology	1 resection + anastomosis 1 colonic stoma	2 died
Uygur-B. O (2003) [18]	31	3	NR*	Laparotomy - histology (60.8%) Ex-juvantibus therapy (28.8%)	NR*	NR*
Rahman A (2003) [55]	3	3	3 ileum	Laparotomy - histology	3 resection + anastomosis	1 died
Tanrikulu CA (2005) [4]	39	3	3 ileum	Ascitic fluid culture (8%) Histology (20%)	NR*	3 survived
Cengiz A (2005) [56]	12	12	10 ileum 2 jejunum	Laparotomy - histology	7 simple suture 5 resection + anastomosis	9 survived 3 died
Jhobta RS (2006) [41]	20	20	20 small bowel		NR*	NR*
Leung VKS (2006) [57]	22	2	2 ileum	Laparotomy - histology	2 resection + anastomosis	2 survived
Clarke DL (2007) [12]	67	5	4 jejunum 1 stomach	Laparotomy - histology	NR*	3 died 2 survived
Leone V (2007) [58]	6	1	1 ileum	Laparotomy - histology	1 resection + anastomosis	1 survived
Tan KK (2008) [44]	57	6	3 ileum 2 colon 1 duodenum	Laparotomy - histology (71.9%) Clinical (28.1%)	2 resection + anastomosis 1 appendectomy 1 drainage 2 NR*	4 survived 2 died (resection + anastomosis group)
Et al. [22,59-62]	5	5	4 ileum 1 colon	Laparotomy - histology	4 resection + anastomosis 1 simple suture	4 survived 1 died (suture group)
Present study	5	1	1 not found	Laparotomy - histology	1 simple drain	1 survived
Total	622	119			54 survived - 18 died	

NR: Not reported.

abdominal X-ray with Gastrografin® meal showed neither fistulas nor perforations and no abdominal effusion was detected by a CT contrast scan. The patient's condition continued to improve during the admission period; no obstructive episodes occurred and the peritonitis appeared to heal satisfactorily. Intestinal transit was resumed 16 days after the intervention, and the first drain was removed 22 days after the surgery. Twenty-five days after the operation, the patient began a combined nutritional regimen, thereby transitioning away from the parenteral nutrition (PN) treatment. On the 30th day after the surgical procedure, the PN was fully discontinued and an abdominal X-ray with Gastrografin® was performed, yielding negative results. Fifty days after the surgical intervention, the last drain was removed and the patient was discharged. After 7 months of anti-TB therapy, the follow-up CT was negative and monitored parameters including Ca-125 (8 U/ml) resulted in the improvement of the patient's condition. The patient was checked one and a half years after the surgery during a routine examination, and appeared to be in good health with no recurrences or complications.

Literature Review

The aim of this study was to analytically summarize all pertinent literature discussing the various treatments for TB-related pathology. We also attempted to stress the difficulty of performing a differential diagnosis among a confounding plethora of non-specific and unclear signs and symptoms.

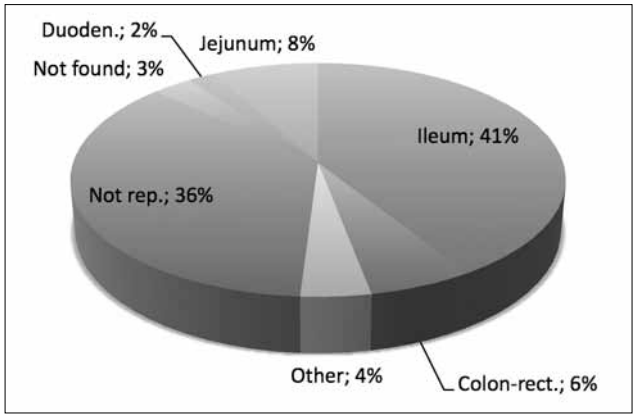

Fig. 5. Site of tubercular perforation.

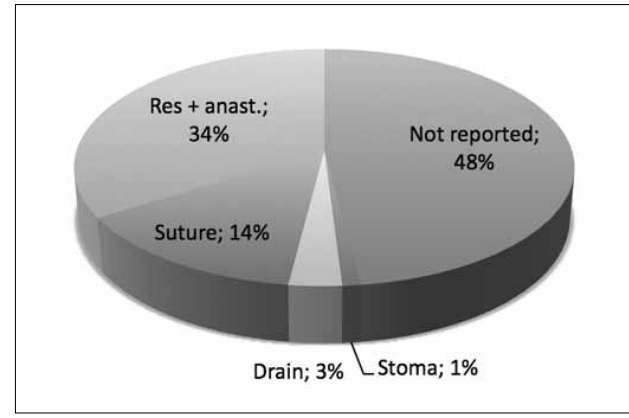

Fig. 6. Type of treatment for tubercular perforation.

In the reviewed literature, we found 54 relevant articles including case reports, case series, and reviews. A total of 622 patients with abdominal TB were considered, among them 119 presenting with intestinal perforation (Table 1). Of the 119 cases reported, in 92 (77.3%), the diagnosis was reached via laparotomy and histology; the remaining 27 (22.7%) were diagnosed by different methods such as ex-juvantibus therapy, clinical findings and ascitic fluid culture.

Analyzing the sites of perforation, we observed 2 perforations of the duodenum, 9 of the jejunum, 50 of the ileum, and 7 of the colon-rectum. The others were localized as: 1 in the stomach and 1 in the appendix, and 3 were multiple. In 3 cases, the surgeons were not able to identify the exact site of perforation, and in 43, the authors failed to report these sites (Fig. 5). We can conclude, in accordance with the reviewed literature, that the most frequent site of perforation is the ileocecal area.

Many of the published papers do not include the description of the methods used to treat the perforation and many of the treatment outcomes are omitted. Forty cases (33.6%) were treated with resection and anastomosis, 17 (14.2%) with simple suture of the perforation, and 4 (3.3%) with the simple positioning of a drain; in 57 cases, the method of treatment was not reported (Fig. 6). Mortality was only reported in a few cases, as seen in Table 1. This lack in reporting the outcomes of the various surgical treatments does not help to establish the best way to treat this rare form of perforation. In fact, the outcome was reported in 72 cases (60.5%), and the total mortality was 25%. Dividing results into the different treatment groups, it is clear how the direct suture gained poorer results, with a mortality of 41.1%, against the resection with anastomosis group, in which mortality reached 21%.

DISCUSSION

Although TB remains relatively rare in developed countries, especially the type involving the gastrointestinal tract, an increase in the number of cases in Europe and the United States has become evident since the mid 1980s.[13] The resurgence of this pathology is directly related to the increasing number of individuals infected with HIV.[2] It has been estimated that a person infected with both HIV and TB has a 7% to 10% chance per year of developing active TB, as opposed to the 10% lifetime chance of someone who is infected with only TB.[14] Over the last four decades, a number of audits on abdominal TB from many parts of the world have been conducted.[8,11,12,15-19] However, many of these audits were conducted during the pre-HIV era. With the global diffusion of HIV, the clinical manifestation of abdominal TB differs greatly from its description in these early reports.

Abdominal TB is a disease that predominantly affects young adults.[3,4,20] Two-thirds of all cases involve patients between 21 and 40 years of age. There is no difference in the incidence rate between male and female subjects, although some studies suggest a slightly increased female predisposition.[3,12,21]

Clinical presentation can be acute, chronic, or both acute and chronic. In the majority of cases, constitutional symptoms are present, including fever (40-70%), pain (80-95%), diarrhea (11-20%), constipation, alternating constipation and diarrhea, weight loss (40-90%), anorexia, malaise, ascites, abdominal distension, night sweating, and hematochezia.[3,4,12] Despite a popular misconception that intestinal complications are often linked to pulmonary TB, only 15-20% of the patients with gastrointestinal tubercular complications have concomitantly active pulmonary TB. Thirty to 50% of patients with abdominal TB have a normal chest film[12] and the tuberculin skin test is positive in only 42% of patients. Given these statistics, such assessments cannot be used as reliable predictors of disease. It should be mentioned that, in areas where TB is endemic, this test has been known to have high false-positive rates, and typically has a lower specificity for abdominal disease than pulmonary TB.[20] For these reasons, they could not be used

to establish the diagnosis in our case as well. Furthermore, tuberculin skin tests cannot accurately differentiate between active disease and previous sensitization by contact or vaccination.[21] Many papers published on abdominal TB emphasize the difficulty of diagnosing this enigmatic pathology. Unfortunately, the tests that clinicians and surgeons rely on are often not sufficiently conclusive to guarantee a reasonably definitive diagnosis. The primary problem is not diagnosing the intestinal perforation, as this is often straightforward enough. Instead, the problem lies in properly identifying the cause, in this case intestinal TB. In any case, intestinal perforation caused by primary abdominal TB is relatively rare.[22]

The instrumental modalities used during investigation include: chest X-rays, small bowel barium meal, barium enema, US, CT, and colonoscopy. Laboratory, immunological, microbiological, and clinical modalities include: ascitic fluid examination, ELISA, peritoneal biopsies, acid-fast bacilli research in the sputum, hematic tests, and tuberculin skin test.[3,4,12,23-25] All of these diagnostic instruments demonstrate a different grade of reliability and confidence, but none of them yields a conclusive diagnosis with any definitive certainty.

We now will try to discuss our case, considering the review of the literature, trying to underline eventual mistakes or misunderstandings in the diagnostic-therapeutic circuit.

Paracentesis has a low diagnostic yield. Direct smears for the Ziehl-Neelsen stain are typically ineffectual, with reported sensitivity ranging from 0% [18,26,27] to 6%.[2,28-30] Cultures of ascitic fluid take a considerable amount of time before results are available, with most tuberculous ascites cases culminating in negative results.[17,24,30] However, it should be noted that the rate of positive culture results could be improved by obtaining 1 L of ascitic fluid concentrated via centrifugation.[24,28] Our hospital microbiologist did not concentrate the ascitic fluid via centrifugation. Several authors suggest that C-reactive protein (CRP) of ascitic fluid obtained by US-guided fine needle aspiration is the diagnostic method of choice for patients with a strong suspicion of intestinal TB. They also asserted that it should always be performed prior to any surgical intervention.[23] In our case, before the urgent surgical intervention, there was not a strong enough suspicion of abdominal TB to perform CRP analysis in the ascitic fluid. If a concentration via centrifugation had been performed, a mycobacteria may have been found, thus leading to a modification in the therapeutic circuit.

Other authors describe the evaluation of adenosine deaminase (ADA) in ascitic fluid[31] as a useful diagnostic tool. This enzyme level is proportional to the level of T-lymphocyte activity. T-cells are stimulated by microbacterial antigens. The level of ADA in tuberculous ascites is significantly higher than it is in cirrhotic or malignant ascites.[3,20] In the event of co-infection with HIV, ADA values could be either normal or slightly below average.[3] However, even if the patient was not HIV-positive, the possibility of using ADA had not been considered.

Many reports have shown strong correlations between tuberculous peritonitis and high Ca-125 serum levels. An elevated serum level of Ca-125 is often indicative of the peritoneal diffusion of ovarian carcinomas. The elevation of this marker in the serum, especially in unclear or convoluted cases, should be examined considering the possibility of peritoneal TB.[32-35] There also seems to be a direct association between serum Ca-125 levels and the resulting efficacy of antituberculous therapy.[33] In our patients, the dosing of Ca-125 was performed about 4 months before the admission in our Operative Unit. It has not been considered as a strong indicator of peritoneal involvement or suggestive for tubercular peritonitis in the absence of signs of other malignancies.

Radiology is very helpful with respect to modern imaging, even without a high grade of specificity. CT scanning is often unable to differentiate this pathology from other intra-abdominal disorders. However, CT scanning is adept at detecting various intra-abdominal features typical of abdominal TB, including thickening of the intestinal mucosa, stranding and thickening of the small bowel mesentery, and the retroperitoneal lymphadenopathy.

Endoscopic techniques like colonoscopy are extremely useful, principally for detecting lesions of the terminal ileum and colon.[3] This kind of examination helps to differentiate between other colon-related pathologies that could present with unspecified bowel symptoms. In the case we explain, a colonoscopy was performed to verify the suspicion of an IBD, and the only findings reported were three nodules covered by apparently normal mucosa, of which the endoscopist decided not to perform a biopsy.

Although both clinicians and surgeons of modern medicine have access to and experience with the aforementioned facilities and techniques, the diagnosis of abdominal TB remains principally a surgical endeavor. Many authors have suggested a laparotomy as the best way to obtain an adequate amount of tissue for a definitive diagnosis of abdominal TB.[8,12,15,16]

Four out of five reviews published between 1972 and 2007[12,15,16,18,19] argue that a precise diagnosis can only be reached intraoperatively or by observing the effects of drug treatment. These reviews primarily dis-

cussed cases from 1962. Although technological advancement since 1962 has had a profound effect on the medical field, the methods of diagnosing intestinal TB remain relatively unchanged. Beginning roughly 10 years ago, laparoscopies began being utilized to investigate the peritoneal cavity for signs of abdominal TB. Even if this technique enabled inspection of the peritoneal cavity without overly invasive maneuvers, it did not reduce dependence on open surgery. Clarke et al.[12] explained how they were only able to establish a conclusive diagnosis and definitive therapeutic intervention in one out of five patients that underwent laparoscopy. Laparoscopies have been hailed as being particularly useful for obtaining quality samples for microbiological analysis. However, laparoscopic investigation of tuberculous peritonitis has failed in 1% to 16% of cases,[29,36,37] and is not without risk. The major complication is intestinal perforation,[38,39] which was originally thought to be more common in the fibro-adhesive form.[29] Five case series investigated the use of laparoscopy in 257 cases of tubercular peritonitis.[29,37-40] They investigated morbidity and complications linked to the use of this technique. Four cases of major complications (intestinal perforation) and three minor complications (ascites oozing through laparoscopic wound and omental hemorrhage) were reported. Obviously, the use of diagnostic laparoscopy is not feasible in urgent settings.

Even if a patient presents with all the appropriate signs and symptoms, there is little conclusive evidence that alludes to intestinal TB perforation. The percentage of intestinal perforation due to abdominal TB is very low, even in countries with high TB incidence. In 2006, Jhobta et al.[41] reported that 3.9% of peritonitis cases in a series of 504 consecutive intestinal perforations resulted from TB-induced intestinal perforation. Another article, considering a series of 204 consecutive patients, reported only 4 cases (1.9%) of intestinal perforation resulting from TB.[42] It should be pointed out that incidence rates have decreased significantly in the past 20 years. In 1986, Kapoor et al.[43] reported a series of 6 cases with an incidence of TB-induced perforation of 13.3%. In those years, the percentages of TB-induced intestinal perforations ranged from 7.5% to 12.2%.[9,10,43]

The clinical presentation of these cases is rather characteristic of the pathology. The diagnosis of intestinal perforation is not difficult in most cases. For the surgeon, the difficulty arises when trying to reach an intra-operative diagnosis and when subsequently planning appropriate treatment for the patient.

Surgical treatment of tuberculous perforations is rather controversial. Although pharmacological treatment remains the central pillar of abdominal TB, emergency surgery is often required for its acute complications,[44] particularly for perforations. There is very little scientific evidence with which to base an argument for the best way to treat these complications of abdominal TB, and the noticeable lack of literature in this field certainly does not help. The bulk of this topic's background is derived from a series of case reports and case series published in a 40-year period. In all, 119 cases have been described, with various means of treatment. In many of the reported cases, the surgical treatment and the outcomes were not adequately described (Table 1). However, direct closure of the perforation with or without bypass is generally associated with poorer results.[45] Resection and anastomosis is therefore recommended,[46] especially if combined with postoperative anti-tubercular therapy.[47-49] However, regardless of the surgical procedure, the mortality rate is relatively high, ranging from 30%[45,46] to 60%.[12] This high rate of mortality is principally due to the poor clinical perioperative conditions of patients undergoing surgery. In addition to already being a highly debilitating pathology, the peritonitis resulting from the perforation overwhelms the patient's capacity to bear surgical stress. In all the reviewed literature, not a single report was found that resembled our case. We believe that our course of action in this case was a safe and responsible way to approach the problem. In the presence of extensive adhesions, no attempt should be made to locate the perforation, as injury to the adherent intestinal loops is likely and focal fistula may result in the postoperative period.[43] In this case, it is possible to place drains and immediately begin an ex-juvantibus anti-tubercular therapy once the surgical intervention has been completed. In our experience, this approach resulted in a complete restoration of health within a reasonable recovery period, even in a very weak patient.

In conclusion, intestinal TB should always be considered when deliberating the possible cause of intestinal perforation. There are no standardized guidelines regarding the surgical treatment of this complication, as it is relatively rare in developed countries, and the chance of making a correct preoperative diagnosis is often unlikely. Many clinical, radiological or laboratory methods can often be employed. Although laparoscopy is a useful technique, the only way of reaching a conclusive diagnosis involves a laparotomy with subsequent histological examination.

The mortality rates seem to be significantly higher when intestinal perforations are treated with simple sutures.[45] Intestinal resection and primary anastomosis seems to be the better way to treat TB-induced intestinal perforation. However, anti-tubercular therapy undisputedly remains the essential pillar of treatment for abdominal TB. Lastly, when searching for the perforation becomes invasive and dangerous or when

the exact site simply cannot be conclusively located, placement of drains and immediate ex-juvantibus anti-tubercular therapy is probably the best solution.

Concluding, the lack of proper research on this topic as demonstrated by incomplete and often inconclusive literature requires further studies to comprehensively investigate ideal treatment for TB perforations. More complete studies performed by centers experienced with this kind of pathology are necessary in order to define standardized guidelines regarding the treatment of this complication.

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