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CLINICAL CASE

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Simultaneous pleural and peritoneal tuberculosis in adolescent: case report

Tuberculosis pleural y peritoneal simultánea en adolescentes: informe de caso

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ABSTRACT

The clinical manifestations are not specific on extrapulmonary tuberculosis and the infectious agent difficult detection do not able to diagnose this disease quickly, so the treatment is late due to a later diagnosis. This scientific article seeks for clinical and epidemiological aspects about peritoneal tuberculosis associated to pleura through this case report. The analytical features about social demographic, clinical and laboratorial variables was obtained from medical records. The diagnosis is suspected by unknown cause for pleural and peritoneal effusion dragged clinical history in young patient, and it is associated to present monocytic cells and high-level Adenosine Deaminase (ADA) in pleural and peritoneal liquid, beyond positive tuberculin skin test. The diagnostic confirmation was performed from gamma interferon (IFN-γ) serum positivity and caseous necrosis granulomatous inflammatory process pleural fragment biopsy.

Keywords: Tuberculosis, peritoneal tuberculosis, pleural tuberculosis.

RESUMEN

Las manifestaciones clínicas no son específicas de la tuberculosis extrapulmonar y la dificultad de detección del agente infeccioso no permite diagnosticar esta enfermedad con rapidez, por lo que el tratamiento es tardío debido a un diagnóstico retrasado. En este artículo científico, se buscan aspectos clínicos y epidemiológicos sobre la tuberculosis peritoneal asociada con la pleura, a través de este informe de caso. Los aspectos analíticos sobre las variables sociales, demográficas, clínicas y de laboratorio se obtuvieron de los registros médicos. Se sospecha que el diagnóstico se debe a una causa desconocida para el derrame pleural y peritoneal arrastrado por la historia clínica en un paciente joven, y se asocia con la presencia de células monocíticas y un alto nivel de adenosina desaminasa (ADA) en el líquido pleural y peritoneal, además de la prueba cutánea de tuberculina positiva. La confirmación diagnóstica se realizó a partir de la positividad del suero de interferón gamma (IFN-γ) y de la biopsia del fragmento pleural del proceso inflamatorio de necrosis granulomatosa.

Palabras clave: *Tuberculosis, tuberculosis peritoneal, tuberculosis pleural.*

INTRODUCTION

The tuberculosis is a contagious infectious disease that has been current in humanity since antiquity, despite of drug treatment for decades. This fact is due to disease control and people's life conditions in the transmission process.

The population impoverishment, uncontrolled urbanization, precarious and hard to reach health services are associated factors for increase of cases' number in the world. Even more the infection emergency for human immunodeficiency virus (HIV) caused tuberculosis worsening in places that were under control.[1,2]

This disease can be caused by one of seven *Mycobacterium* species complex: *M. bovis*, *M. africanum*, *M. canetti*, *M. microti*, *M. pinnipedi*, *M. caprae* e *M. tuberculosis*. The last specie is known as Koch bacillus, the most prevalent one. This agent transmission is by airway. The pulmonary or laryngeal tuberculosis is able to be expelled by coughing, speaking and wheezing, so the air droplets can be inhaled and infect host airway.

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One third of world's population would be infected by *M. tuberculosis* without symptoms, those are latent tuberculosis patients. The main infected people will control this latent infection throughout life and only 5 to 10% of cases could evolve to active disease form, mainly in the first two years after infection.[1,3]

World Health Organization's database reveals 10.4 million cases incidence in 2016, approximately 140 cases per 100 thousand inhabitants.[3]

Developing countries known as BRICS (Brazil, Russia, India, China, South Africa) have 53% of tuberculosis cases. This disease is among the 10 leading causes of death in the world, that caused 1,3 million deaths in 2016.[4]

According to WHO, in Brazil in 2017, 72,700 tuberculosis cases was notified within 10 to 74.7% cases per 100 thousand inhabitants according to each location.[1,5]

The extrapulmonary tuberculosis is the most frequent disease within symptoms as persistent dry or productive cough, afternoon fever, night sweats and weight loss.

The pulmonary tuberculosis patients within positive bacilloscopy maintains the transmission chain. A positive bacilloscopy patient could infect 10 to 15 people in social environments for a year.

After pulmonary infection, the bacillus will migrate to hilar and mediastinum ganglia through lymphatic system. After this first migration, the next one happens also through blood flow to reach other organs, that can be inactive for a long time period or eventually evolves to other forms of extrapulmonary involvement that depends on host immunity and pathogen balance.[6]

The most relevant extrapulmonary tuberculosis forms are pleural, ganglionic, meningoencephalic, pericardial, bone, intestinal and peritoneal.

The extrapulmonary forms clinically manifested depend on system or organ involved, however nonspecific symptoms are reported in other diseases in many cases.

The unspecific symptomatology and difficulty of infectious agent detection in different organs become the extrapulmonary tuberculosis harder. Therefore, the late tuberculosis recognition delays the appropriate therapy.

This fact justifies the clinical and laboratorial knowledge as much peritoneal tuberculosis as pleural tuberculosis separated or together, helping in clinical diagnosis of these affections.

Objective

Knowing the epidemiological and clinical aspects of peritoneal tuberculosis associated with pleural tuberculosis.

METHOD

This is a clinical and observational study of case report about a adolescent diagnosed with simultaneous pleural and peritoneal tuberculosis. This adolescent was attended in children's hospital of the Brazilian Public Health System in São Paulo in 2019.

The analytical variables regarding sociodemographic, clinical and laboratory features were obtained from the medical record after research project approval of Santa Marcelina Medical School Ethics and Research Committee under reference 4'067,551.

CLINICAL CASE

15-year-old male adolescent, black, from São Paulo, young apprentice in metallurgical industry. The chief complaints are a diffuse abdominal pain, fever, chills, night sweats and weight loss for 45 days. In the last week of disease evolution, progressive abdominal volume began to grow and increase of respiratory distress.

On physical examination, the patient was in a regular general state, lucid and oriented, bleached 2+/4+, dyspneic 1+/4+, anicteric, acyanotic, febrile and lost weight.

Weight: 60 kg
Height: 168 cm
Temperature: 38 ºC
IMC: 21,3 kg/m²
Skin: BCG vaccination scar.
Respiratory system: low level dullness and vesicular murmur decrease on the left hemithorax base.
Respiratory rate: 28 breaths per minute.
Heart: Rhythmic normophonetic twice without murmurs heart sounds.
Heart rate: 107 beats per minute.
Abdomen: globose, normative positive hydroaerial noise, tense and painful at diffuse palpation, signs of ascites (positive shifting dullness), without hepatosplenomegaly, without collateral circulation.

During disease investigation, the following procedures were made:

Imaging tests

Chest X-ray: opacity in ⅔ of inferior hemithorax suggestive of pleural effusion.

Thoracic computerized tomography: pleural effusion on the left side; mosaic picture on the left lower lobe.

Abdominal computerized tomography: massive ascites, without mesenteric adenomegaly.

Pleural puncture and biopsy

Pleural liquid: citrus yellow; pH: 7; glucose: 73; protein: 5.7; lactate dehydrogenase (DHL): 862.

Bacilloscopy, culture and polymerase chain reaction (PCR) for *M. tuberculosis*: negative.

Adenosine-deaminase (ADA): 85.16 U/L.

Cytology: absence of neoplastic cells; leukocyte: 586 (93% mononuclear).

Histopathological evaluation of pleural fragments: granulomatous inflammatory process featured by epithelioid granuloma well-formed, not coalescent, lymphocytic infiltrate, Langerhans giant cells and caseous necrosis sources.

Ascitic fluid puncture: citrus yellow; pH: 8; glucose: 71; protein: 5.1 (albumin: 3 g/dL).

Serum-ascites albumin gradient (SAAG): 0.4 g/dL.

Lactate dehydrogenase (DHL): 764, amylase: 50 UI/L.

Bacilloscopy, culture and polymerase chain reaction (PCR) for *M. tuberculosis*: negative.

Adenosine-deaminase (ADA): 71.05 U/L.

Cytology: absence of neoplastic cells.

Blood test: glucose: 98; protein: 6.7 (albumin: 4.1 g/dL); lactate dehydrogenase (DHL): 538.

Skin test (PPD): 15 mm.

Interferon gamma (quantiferon TB gold-plus test): positive.

According to exams results, pleural and peritoneal presumptive diagnosis was performed, so the RIPE scheme (rifampicin, isoniazid, pyrazinamide, ethambutol) was prescribed. After 6 months of therapy, the patient got body weight gain, in addition to reducing pleural effusion, ascitis, fever and abdominal pain.

DISCUSSION

The pleural tuberculosis is the most common extrapulmonary manifestation and it represents the main reason of development of chronic pleural effusion that impacts young people. This manifestation is approximately 8% of tuberculosis new cases.[7,8]

The pleural involvement can happen by hematogenous or lymphatic way, so that is a primary pleural involvement. That can also happen by contiguity, secondary to a pulmonary source.[1,9]

The *Mycobacterium tuberculosis* identification on pleural fluid or tissue sample is gold standard diagnosis method. However, this diagnosis method has low sensitivity, less than 30% on pleural liquid and approximately 50% on pleural tissue, that is a clinical practice challenge because the diagnosis is hard to be definitive.[10]

In facing these challenges, in most cases, the diagnosis can be reached from as much patient signs and symptoms as imaging exams results and pleural liquid laboratory findings under cytological, biochemical and physical aspects. Besides pleural biopsy, the measurement of adenosine-deaminase (ADA), gamma interferon (IFN-γ) and polymerase chain reaction (PCR) and skin test is important to consider in diagnosis establishment.[11]

The clinical symptomatology is poorly specific. Generally, the present symptoms are pleuritic thoracic pain, respiratory distress and the triad: asthenia, weight loss and anorexia in until 70% of cases. Fever and dry cough happen in until 60% of patients.[1]

Frequently, the pleural effusion is unilateral, mild to moderate volume, that is 25 to 75% of patient hemithorax filling.[10]

The appearance of the fluid is citrus yellow, but eventually it can be cloudy.

The pleural effusion by tuberculosis whose biochemical features include elevation of protein levels over 4.5 g/dL and slight DHL increase.[10]

Through cytological studies, increase of cellularity is verified, from 1,000 to 6,000 cells/mm^3 through macrophages and lymphocytes, but in the disease initial phase there may be neutrophilic predominance. The number of mesothelial cells resulting from pleural peeling do not exceed 5%.[9,10]

In anatomopathological study, the demonstration of granulomas within or without caseous necrosis in pleural fragments is a great indicator for pleural tuberculosis primarily in disease high incidences locations, while other diseases as sarcoidosis, rheumatoid arthritis and fungal infection have the same histological standard. It is noteworthy that human immunodeficiency virus (HIV) and diabetes

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immunosuppression could not reveal granuloma in patients. The association between pleural biopsy and pleural fragment culture permits until 87% diagnosed patients.[9,12]

Surely, the pleural biopsy technique is more difficult to be performed in not specialized services.

The ADA quantification is an enzyme produced by macrophages and activated T lymphocytes. This quantification is usually elevated, that is higher level than 40 U/L, so it is necessary to consider differential diagnosis with other pathologies as rheumatoid arthritis, systemic lupus erythematosus, lymphoma, some adenocarcinomas and empyemas. The method sensitivity varies from 90 to 100% and the method specificity varies from 89 to 100%. This diagnosis method is more sensitive for pleural tuberculosis than pleural histopathological exam and bacteriological tests.[9,10,12,13]

The gamma interferon (IFN-γ) is a useful marker for pleural tuberculosis diagnosis. Using 140 pg/mL cut-off, sensitivity varies from 86 to 97% and specificity varies from 95 to 100%. Income from this diagnosis method is similar to ADA dosage in pleural liquid. Despite of high specificity as much in pleural fragment as pleural liquid, with the advantage of not be altered on other pathologies; however, its high cost limits its routine use.[9,12]

Polymerase chain reaction (PCR), technique for *M. tuberculosis* nucleic acid amplifying, has high sensitivity (95%) and high specificity (98%) in sputum samples, but it has low sensitivity of pleural affection diagnosis. Despite of high specificity as much in pleural fragment as pleural liquid, the pleural tissue sensitivity is very variable, that is from 20 to 90% and in the pleural liquid is 17%.[9,12]

Pleural tuberculosis differential diagnosis is through pulmonary neoplasia. In the last situation, the affection impact individuals of a more advanced age that are in state of cachexia. Pleural effusion is high volume, sometimes bilateral, in cloudy appearance (lymph) or serohemorrhagic, within protein slight increase and sharp DHL increase. The cytology reveals neoplastic cells, when it is negative the diagnosis is confirmed by pleural fragment anatomopathological exam.[10]

The patient in this clinical case, besides presenting pleural involvement, one pulmonary parenchymal lesion detected only on thoracic computerized tomography. Some pulmonary tissue amendments are not noticeable on thorax radiography, such as lobular center nodules with segmental distribution, bronchial wall thickening, mosaic aspect, translated by higher and lower density areas coexistence on pulmonary parenchyma, the latter being air trapping resulting from scarring constrictive bronchiolitis, and the «tree-in-bud» image, due to bronchiolar dilatation by exudative material presence. These findings are detected on 30 to 60% of pleural tuberculosis patients submitted to thoracic computerized tomography.[9,14]

Pleural affection by contiguity could reach peritoneum, so it causes peritoneal tuberculosis. Pleural effusion is observed in 22 to 32% of peritoneal tuberculosis patients and pulmonary source in 15 to 20% of cases.[15,16]

Peritoneal tuberculosis is the least frequent disease involvement form, though the peritoneum is the most common involved abdominal structure by abdominal tuberculosis. It represents 4 to 7% of extrapulmonary form cases and 0.1 to 0.7% of every clinical manifestation kind for this disease. It is more common for age group between 35 and 45 years old, chronic liver disease, HIV, diabetes mellitus, under anti-TNF treatment, on peritoneal dialysis and on alcoholic people.[17-21]

Peritoneal tuberculosis is a peritoneum *Mycobacterium tuberculosis* growth resulting. It happens through pre-existing latent sources in this structure, due to previous hematogenous dissemination from a primary pulmonar source; from active pulmonary tuberculosis hematogenous dissemination, and rarely by contiguity from a abdominal organs or adjacent pelvic organs, vertebral column and pleura involvement, as described by this scientific article.[17,22]

Clinical manifestation can be acute or chronic. and symptoms as: fever, paleness, pain and abdominal distention, swelling, anorexia, weight loss, weakness, sweats and bowel habit disorder is reported. Abdominal pain and abdominal volume increase are associated to ascites, that happens late in the development of symptomatology, so these symptoms are most common patient's chief complaint.[17,18,20,21]

Peritoneal tuberculosis has three described forms: wet, within free or loculated ascite; the dried, within caseous nodules and peritoneal fibrosis; and fibrotic, within grouping straps, omental masses and adherences. The wet one is most frequent clinical form of this disease, in which ascite is developed secondarily to peritoneum protein liquid exudate, similar to mechanism that develops ascites in peritoneal carcinomatosis patients. More than 90% of peritoneal tuberculosis has ascite at time of diagnosis,

while the rest of patients present the wet and/or fibrotic one in advanced stages.[17,21,22]

This pathology differential diagnosis is very broad, including other granulomatous diseases, such as sarcoidosis and systemic amyloidosis, besides other infectious processes and malignant affections as lymphoma, mesothelioma and peritoneal carcinomatosis.[17,18,20]

This affection diagnosis methods are not always accessible and some demand a lot of time for results obtaining, including ascitic fluid analysis under cellularity features and biochemistry when present; biomarkers dosage such as ADA and IGRA in addition to bacteriological exam; besides peritoneal fragment anatomopathology and skin test.

Peritoneal tuberculosis must be suspected when facing inexplicable lymphocytic ascite, cellularity varying from 500 to 1500 cells, within serum-ascites albumin gradient (SAAG) is less than 1.1 g/dL in liver cirrhosis absence and high-level DHL.[17,21]

Gamma interferon (IFN-γ) IGRA is a biomarker that plays a potential role in peritoneal tuberculosis diagnosis, whose sensitivity is 93% and specificity is 99%.[23]

The gold standard diagnosis method is infectious agent isolation in ascitic fluid culture or evidence to granulomatous process within central caseous necrosis in peritoneal biopsy. Nevertheless, the bacillus identification possibility in bacilloscopy (3%) and in culture (35%) is too low, due to the small amount of bacillus in this fluid. That's why laparoscopy with guided-biopsy is the best way for a fast and specific diagnosis, revealing caseous granulomas in 100% of cases and positivity in bacillus culture in 74%.[17,18,21,24]

The validity of using polymerase chain reaction in ascitic fluid regarding peritoneal tuberculosis research is controversial yet, due to low bacillus amount in ascitic fluid and also prime kind used for performing this exam. Uzunku & Harma found PCR positivity for *M. tuberculosis* in 100% of 11 investigated cases; while Thoreau et col. obtained positivity in only 1 case of 3 investigated patients.[15,25]

Finally, it is worth noting that in investigation process as much pleural tuberculosis as peritoneal tuberculosis, the skin test is useful, demonstrating positive outcome of most cases. In Brazil, the tuberculin used is PPD-RT23 administered intradermally in the middle third of the left forearm anterior face, within reading after 48-72 hours. Positive outcome is considered by more or equal 5 mm induration, indicating disease or infection presence, depending on patient's vacational status assessment and clinical assessment. In the early stage of pleural disease, the skin test could be negative, due to lymphocytes being recruited in pleural or peritoneal space, with possible tuberculinique turning after a second skin test substance administration.[1,9,16,26]

CONCLUSION

Pleural and peritoneal tuberculosis should be remembered as differential diagnosis in young patient, within dragged pleural effusion and inexplicable ascite. High-levels of ADA and monocyte cells presence as much pleural fluid as ascitic fluid, besides positive skin test and gamma interferon (IFN-γ) serum presence, the tuberculosis diagnosis is confirmed, since infectious agent isolation in pleural and peritoneal effusion is not always possible.

Anatomopathological exam must be performed whenever possible, as much pleural fragment as peritoneal fragment, besides it permits granulomatous process within central caseous necrosis, that is so suggestive for mycobacterial affection, allowing this agent identification in specific culturing media.

REFERENCES

1. Brasil. Ministério da Saúde. Secretaria de Vigilância em Saúde. Departamento de Vigilância das Doenças Transmissíveis. Coordenação Geral do Programa Nacional de Controle da Tuberculose. Manual de Recomendações para o Controle da Tuberculose no Brasil. Brasília, 2019. [acesso em 22 set 2019]. Disponível em: http://bvsms.saude.gov.br/bvs/publicacoes/manual_recomendacoes_controle_tuberculose_brasil_2_ed.pdf
2. San Pedro A, Oliveira RM. Tuberculose e indicadores socioeconômicos: revisão sistemática da literatura. Rev Panm Salud Publica. 2013; 33 (4): 294-301.
3. World Health Organization (WHO). Global tuberculosis report. 2017. [Acesso em 22 set 2019]. Disponível em: https://www.who.int/tb/publications/global_report/gtbr2017_main_text.pdf
4. Mello FCQ, Silva DR, Dalcomo MP. Tuberculose: onde estamos? J Bras Pneumol. 2018; 44 (2): 82-82.
5. Brasil. Ministério da Saúde. Secretaria de Vigilância em Saúde. Secretaria de Vigilância em Saúde. Brasil Livre da Tuberculose: evolução dos cenários epidemiológicos e operacionais da doença. Boletim Epidemiológico. 2019; 50 (9). Acesso em 16 de jan 2020. Disponível em: http://portalarquivos2.saude.gov.br/images/pdf/2019/marco/22/2019-009.pdf
6. Carvalho ACC, Cardoso CAA, Martire TM, Migliori GB, Sant'Anna CC. Aspectos epidemiológicos, manifestações clínicas e prevenção da tuberculose pediátrica sob a perspectiva da Estratégia End TB. J Bras Pneumol. 2018; 44 (2): 134-144.

7. Rodríguez R. tuberculosis pleural em paciente pediátrico: reporte de un caso y revisión de la literatura. Neumol Pediatr. 2018; 13 (1): 29-31.
8. Shaw JA, Diacon AH, Koegelenberg CFN. Tuberculous pleural effusion. Respiratory. 2019; 24: 962-971.
9. Seiscento M, Conde MB, Dalcolmo MMP. Tuberculose pleural. J Bras Pneumol [Internet]. 2006 [cited 2019 Oct 16]; 32 (Suppl 4): S174-S181. Disponível em: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S1806-37132006000900003&lng=en.
10. Antonangelo A, Vatgas FS, Seiscento M, Bombarda S, Teixeira L, Sales RKB. Clinical and laboratory parameters in the diferential diagnosis of pleural effusion secondary to tuberculosis of cancer. Clinics. 2007; 52 (5): 585-590.
11. Gopi A, Madhavan SM, Sharma SK, Sahn AS. Diagnosis and treatment of tuberculous pleural effusion in 2006. Chest. 2007; 131 (3): 880-889. doi: 10.1378/chest.06-2063.
12. Silva CT Jr. Abordagem diagnostica da tuberculose pleural, ganglionar, renal e de sistema nervosos central. Pulmão RJ. 2012; 21 (1): 32-35.
13. Kaisemann MC, Kritski AL, Pereira MFC, Trajman A. Dosagem da atividade da adenosina deaminae no liquido pleural para o diagnóstico da tuberculose pleural. J Bras Pneumol. 2004; 30 (6): 549-556.
14. Rocha EL, Pedrassa BC, Bormann RL, Kierszenbaum, Torres LR, d'Ippolito G. Tuberculose abdominal: uma revisão radiológica com ênfase em achados de tomografia computadorizada e ressonância magnética. Radiol Bras. 2015; 48 (3): 181-191. Disponível em: http://dx.doi.org/10.1590/0100-3984.2013.1801
15. Thoreau N, Fain O, Babinet P et al. Tuberculose péritonéale: 27 cas dans la banlieue nord-est de Paris N. Int J Tuberc Lung Dis. 2002; 6 (3): 253-258.
16. Vaid U, Kane GC. Tuberculous peritonitis. Microbiol Spectrum. 2017; 5 (1):1-6. doi: 10.1128/microbiolspec.TNMI7-0006-2016
17. Wanderley BR, Maquiné GA, Vieira GN, Tallo FS, Lopes RD, Lopes AC. Tuberculose peritoneal: um diagnóstico diferencial de abdômen agudo: relato de caso. Rev Soc Bras Clin Méd. 2012; 10 (6): 544-546.
18. Cavalli Z, Ader F, Valour F, Saison J, Boussel L, Dumitrescu O et al. Clinical presentation, diagnosis, and bacterial epidemiology of peritoneal tuberculosis in two university hospitals in France. Infect Dis Ther. 2016; 5 (2): 193-199.
19. Guirat A, Koubaa M, Mzali R, Abid B, Ellouz S, Affes N et al. Peritoneal tuberculosis. Clin Res Hepatol Gastroenterol. 2011; 35: 60-69.
20. Ximenes ARG, Cavalcante Filho JRM, Bezerra DA, Guimarães Filho A, Adeodato WDC. Tuberculose peritoneal em paciente jovem imunocompetente: Relato de caso. Rev Med Minas Gerais. 2017; 26: 1-4. Disponível em: http://dx.doi.org/10.5935/2238-3182.20160149.
21. Fonseca LG, Campos FPF, Silva AF. Tuberculose peritoneal: experiência em um hospital geral. Autopsy Case Rep [Internet]. 2011; 1 (2): 3-9. Acesso em 16 jan 2020. Disponível em: http://doi.editoracubo.com.br/10.4322/acr.2016.002
22. Zaslavcky J, Gordon LM, Vasko I, Presenza T, Scattergood E, Meisli D et al. Tuberculous peritonitis in children: two cases reports highlighting the important role of imagining. Radiology Case Reports. 2018; 13: 862-866.
23. Su S, Qin S, Guo XY, Luo W, Jiang X. Assessment by meta-analysis of interferon-gamma for the diagnosis of tuberculous peritonitis. World J Gastroenterol. 2013; 19 (10): 1645-1651.
24. Chow KM, Chow VCY, Hung LCT, Wong SM, Szeto CC. Tuberculous peritonitis–associated mortality is high among patients waiting for the results of mycobacterial cultures of ascitic. Clin Infect Dis. 2002; 35: 409-413.
25. Uzunkoy A, Harma M, Harma M. Diagnosis of abdominal tuberculosis: experience from 11 cases and review of the literature. World J Gastroenterol. 2004; 10 (24): 3647-3649.
26. Fanlo P, Tiberio G. Tuberculosis extrapulmonar. An Sist Sanit Navar. 2007; 30 (3): 143-162.

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