Tuberculous Peritonitis Diagnosed by GeneXpert in Ascitic Fluid Despite Markedly Elevated Carcinoembryonic Antigen: A Case Report from Timor Leste

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ABSTRACT

Background: Tuberculous peritonitis is an uncommon form of extrapulmonary tuberculosis that can closely mimic intra-abdominal malignancy because of its nonspecific clinical presentation, ascites, weight loss, and elevated tumor markers. In selected patients, GeneXpert MTB/RIF testing of ascitic fluid may provide a rapid means of confirming *Mycobacterium tuberculosis* infection.

Case Presentation: A 24-year-old Timorese woman was referred from a peripheral health-care facility to Hospital Nacional Guido Valadares (HNGV) with progressive abdominal distension, abdominal pain, anorexia, and significant weight loss. Physical examination revealed gross ascites. Laboratory investigations demonstrated a markedly elevated carcinoembryonic antigen (CEA) level of 109 µg/L, raising significant concern for gastrointestinal or gynecological malignancy. Diagnostic paracentesis revealed exudative ascitic fluid. GeneXpert MTB/RIF testing of the ascitic fluid detected *Mycobacterium tuberculosis* with no evidence of rifampicin resistance. Cytological examination showed no malignant cells. Abdominal imaging demonstrated significant ascites but no definite intra-abdominal mass or other convincing evidence of malignancy. Based on the clinical presentation and molecular detection of *M. tuberculosis*, the patient was diagnosed with tuberculous peritonitis and commenced on standard anti-tuberculosis therapy. She subsequently demonstrated clinical improvement.

Conclusion: Tuberculous peritonitis should remain an important differential diagnosis in young adults presenting with ascites, weight loss, and abdominal symptoms in tuberculosis-endemic settings such as Timor-Leste, even when tumor markers such as CEA are markedly elevated. This case demonstrates that elevated CEA does not necessarily indicate malignancy and that GeneXpert MTB/RIF testing of ascitic fluid can contribute to rapid diagnosis in appropriate clinical settings. Early recognition of abdominal tuberculosis may prevent unnecessary invasive investigations or surgical intervention and facilitate timely initiation of effective treatment.

Keywords: Tuberculous peritonitis, Ascites, GeneXpert, Carcinoembryonic antigen, Extrapulmonary tuberculosis, Timor-Leste

INTRODUCTION

Tuberculosis (TB) remains a major global cause of infectious morbidity and mortality. Extrapulmonary tuberculosis accounts for approximately 15–20% of all TB cases in immunocompetent adults and occurs more frequently among immunocompromised individuals (1,2). Peritoneal tuberculosis is an uncommon form of extrapulmonary TB, accounting for approximately 1–2% of all TB cases. Its diagnosis is often challenging because the clinical and radiological features can closely mimic peritoneal carcinomatosis and ovarian or gastrointestinal malignancy (3–5).

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Patients with tuberculous peritonitis commonly present with nonspecific symptoms, including abdominal pain, fever, anorexia, weight loss, abdominal distension, and ascites. Microbiological confirmation is often difficult because acid-fast bacilli are infrequently detected in ascitic fluid, while mycobacterial culture may take several weeks. Molecular diagnostic methods, including GeneXpert MTB/RIF, have improved the rapid detection of *Mycobacterium tuberculosis* in selected extrapulmonary specimens.

Carcinoembryonic antigen (CEA) is commonly associated with gastrointestinal malignancy, particularly colorectal cancer. However, elevated CEA levels may also occur in benign inflammatory and infectious conditions, potentially creating diagnostic uncertainty. Distinguishing tuberculous peritonitis from malignant ascites can therefore be challenging, particularly in patients with massive ascites and markedly elevated tumor markers (5,6).

Case Presentation

A 24-year-old woman from Timor-Leste was referred from a peripheral healthcare facility to Hospital Nacional Guido Valadares (HNGV) with a several-month history of progressive abdominal distension, diffuse abdominal discomfort, anorexia, and weight loss.

She reported no previous history of tuberculosis or anti-tuberculous treatment and no known family history of tuberculosis. She was married and had one child. She did not smoke or report the use of alcohol or other recreational substances. She was unemployed and stayed at home caring for her child in a rural village.

Physical Examination on Admission

On admission, the patient was a young woman who appeared acutely ill, anxious, and distressed. She had marked abdominal distension associated with respiratory distress and chest discomfort. The abdomen was grossly distended, with bilateral flank fullness and bulging of the flanks in the supine position.

She was conscious but appeared pale and diaphoretic. There was no obvious peripheral cyanosis.

Vital Signs

Her blood pressure was 110/70 mmHg, heart rate 90 beats/minute, respiratory rate 28 breaths/minute, oxygen saturation 90% on room air, and temperature 36.5°C.

Cardiovascular Examination

The peripheral pulses were weak but regular, with a capillary refill time of less than 2 seconds. The jugular venous pressure was not elevated. The apex beat was palpable in the fifth intercostal space, and the heart rhythm was regular. The first and second heart sounds were present.

Respiratory Examination

The patient had increased respiratory effort. Bilateral air entry was present, with no wheezing.

Abdominal Examination

The abdomen was markedly distended, with bilateral flank fullness and bulging of the flanks in the supine position. The umbilicus was stretched. On palpation, the abdomen had a doughy consistency with mild-to-moderate diffuse tenderness. Shifting dullness and a positive fluid thrill were present, consistent with a large volume of ascites. Assessment of hepatomegaly, splenomegaly, and other intra-abdominal organs was limited by the large volume of ascitic fluid.

Neurological Examination

The patient had a Glasgow Coma Scale score of 13/15. No focal neurological deficit was identified.

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Extremities

Bilateral pitting edema of the lower limbs was present. There were no clinical signs of chronic liver disease or peripheral vascular disease.

Investigations

Investigations	Result	Normal ranges
White blood cells	15.9X 10^9/L	3.5-11.0 x10^9/L
Neutrophils	80.1 %	50-70%
Hemoglobin	9.0 g/L	130-170 g/L
MCV	82.3 fL	80-100 fL
MCH	27.5 pg	27-32 pg
HCT	0.44 l/l	0.40-0.54 l/l
Platelets	540 x10^9/L	150-410 X 10^9/L
Sodium	137.5 mmol/L	135-145 mmol/L
Potassium	5.6 mmol/L	3.5-5.2 mmol/L
Blood urea nitrogen	5.6 mmol/L	3.0-8.0 mmol/L
Creatinine	54.4 umol/L	60-100 umol/L
Random Blood Glucose	5.0 mmol/L	3.0-7.7 mmol/L
ALT	26 U/L	<35 U/L
AST	14 U/L	5-35 U/L
ALKP	67 U/L	30-100 U/L
GGT	26 U/L	9-64 U/L
Uric acid	413.2 umol/L	137-452 umol/L
ESR	100 mm/ hour	Male < 15 mm/hour Female <20 mm/hour
CRP	120 mg/L	< 5 mg/L
Iron test	3.5 umol/L	6-31.3 umol/L
CA. 19.9	36.0 kU/L	0.0—40.0 kU/L
Alpha feto protein	3.6 ng/mL	0.0-<10.0 ng/mL
CA.125	30 kU/L	0-35 kU/L
CEA	109 ug/L	0.0-<5.0 ug/L
Gen-Xpert ascites fluid	MTB detected	MTB not detected
Rifampicin resistance	No detected	No detected
Cytology ascites fluid	No malignant cells detected	No malignant cells detected
Bacterial culture	No growth after 5 days	No growth
HBsAg test	Non-reactive/ negative for hepatitis B	Non-reactive
HIV test	Non-reactive/ negative for HIV	Non-reactive
SAAG	<1.1 g/dl (low gradient) non-portal hypertension	>1.1 g/dl: portal hypertension <1.1 g/dl: non-portal hypertension

Investigations

Laboratory investigations included a complete blood count, peripheral blood film, renal function tests, serum electrolytes, liver function tests, serum albumin and total protein, inflammatory markers, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), and screening for HIV, hepatitis B, and hepatitis C.

Tumor markers, including carcinoembryonic antigen (CEA), cancer antigen 125 (CA-125), cancer antigen 19-9 (CA 19-9), and alpha-fetoprotein (AFP), were assessed because of the clinical suspicion of intra-abdominal malignancy.

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Abdominal ultrasonography and contrast-enhanced computed tomography (CT) of the abdomen and pelvis were performed to further evaluate the cause of the ascites and identify possible intra-abdominal or pelvic malignancy.

Diagnostic abdominal paracentesis was subsequently performed. Ascitic fluid was analyzed for cell count and differential, protein and albumin concentration, Gram staining, bacterial culture, acid-fast bacilli (AFB), GeneXpert MTB/RIF, mycobacterial culture where available, and cytological examination for malignant cells.

Investigations

Initial laboratory investigations demonstrated a markedly elevated serum carcinoembryonic antigen (CEA) level of 109 µg/L, which raised clinical suspicion for an underlying intra-abdominal malignancy.

Abdominal ultrasonography demonstrated moderate-to-massive ascites. Diagnostic paracentesis yielded straw-colored, slightly cloudy, exudative ascitic fluid.

Ascitic Fluid Analysis

Ascitic fluid investigations showed:

- GeneXpert MTB/RIF: *Mycobacterium tuberculosis* detected
- Rifampicin resistance: Not detected
- Cytology: No malignant cells identified
- Bacterial culture: Negative after 5 days

Abdominal Ultrasonography

Abdominal ultrasonography demonstrated moderate-to-massive complex ascites containing multiple fine internal echoes and thin septations. Diffuse smooth peritoneal thickening with associated omental thickening was observed. Multiple enlarged mesenteric lymph nodes were present, some demonstrating central hypoechogenicity suggestive of necrosis.

The liver, spleen, pancreas, and kidneys were normal in size and echotexture. The uterus and adnexa were also normal in appearance. No focal hepatic lesion or definite intra-abdominal solid-organ mass was identified.

Overall, the sonographic findings were suggestive of peritoneal tuberculosis; however, peritoneal carcinomatosis or other peritoneal malignancy could not be excluded based on imaging alone.

Contrast-Enhanced Computed Tomography

Contrast-enhanced CT of the abdomen demonstrated massive ascites with diffuse smooth peritoneal enhancement and thickening, associated with omental and mesenteric inflammatory changes. Multiple enlarged necrotic mesenteric lymph nodes with rim enhancement were also identified. These findings were highly suggestive of tuberculous peritonitis.

No focal colonic mass, bowel obstruction, or significant colonic wall lesion was identified. There was also no radiological evidence of malignancy involving the other intra-abdominal solid organs.

Peripheral Blood Film

Peripheral blood film examination demonstrated predominantly normocytic, normochromic red blood cells with mild anisocytosis and no significant abnormal red-cell morphology. The white blood cell morphology was unremarkable, although neutrophilia was present. Mild thrombocytosis was also observed. No blasts or other abnormal cells were identified.

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The overall findings were compatible with a chronic inflammatory process and were interpreted in conjunction with the patient's clinical and laboratory findings.

Diagnosis

The diagnosis was established based on the patient's clinical presentation, ascitic fluid GeneXpert result, radiological findings, and laboratory investigations. The detection of *M. tuberculosis* in ascitic fluid by GeneXpert MTB/RIF, with no rifampicin resistance detected, provided microbiological confirmation of tuberculosis.

The patient was diagnosed with **tuberculous peritonitis presenting with massive ascites**, associated with **moderate anemia** and **moderate-to-severe malnutrition**.

Treatment and Clinical Outcome

The patient was commenced on standard treatment for drug-susceptible extrapulmonary tuberculosis according to the national tuberculosis treatment guidelines. Treatment consisted of an intensive phase of 2 months with isoniazid, rifampicin, pyrazinamide, and ethambutol, followed by a 4-month continuation phase with isoniazid and rifampicin, for a total treatment duration of 6 months. Treatment was administered under the directly observed treatment, short-course (DOTS) program.

Nutritional management was initiated because of her moderate-to-severe malnutrition. She received a high-calorie, high-protein diet, together with multivitamin and mineral supplementation. Pyridoxine 50 mg orally once daily was administered during anti- tuberculous therapy. Oral nutritional supplementation with F-100 (Formula 100) was provided under the supervision of the hospital nutrition team. Nutritional therapy was monitored closely because of the potential risk of refeeding syndrome.

The anemia was considered to be predominantly related to chronic inflammation. Management focused primarily on treatment of the underlying tuberculous infection and nutritional support, including dietary sources of iron, folate, and vitamin B12.

Because of the massive ascites and associated abdominal discomfort and respiratory symptoms, therapeutic abdominal paracentesis was performed. Approximately 2.5 L of straw-colored, slightly cloudy ascitic fluid was drained. The fluid was submitted for biochemical, microbiological, molecular, and cytological investigations, including assessment of the serum-ascites albumin gradient (SAAG).

During hospitalization, the patient's abdominal distension gradually decreased following initiation of anti-tuberculous treatment. Her appetite and general condition improved progressively. She was discharged after 28 days of hospitalization and continued regular outpatient follow-up while completing the 6-month anti-tuberculous treatment course.

At completion of treatment, she had returned to her usual daily activities without reported complications related to tuberculous peritonitis.

DISCUSSION

Tuberculous peritonitis is an uncommon manifestation of extrapulmonary tuberculosis and accounts for approximately 1–2% of all tuberculosis cases. It occurs predominantly in regions with a high tuberculosis burden and may affect young adults (1,2). The clinical presentation is often nonspecific, with abdominal pain, abdominal distension, fever, anorexia, weight loss, and ascites being among the most common manifestations (2,3).

The differential diagnosis of ascites in a young woman is broad and includes tuberculous peritonitis, ovarian and gastrointestinal malignancies, peritoneal carcinomatosis, and chronic liver disease (3). Considerable overlap exists between the clinical and radiological features of tuberculosis and malignancy, which can result in diagnostic delay and inappropriate treatment (3,4).

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In this patient, the markedly elevated serum CEA concentration of 109 μg/L initially raised concern for intra-abdominal malignancy. CEA is widely used as a tumor marker, particularly in colorectal and other gastrointestinal malignancies. However, elevated CEA concentrations have also been reported in benign inflammatory and infectious conditions, including tuberculosis (5). Therefore, an elevated CEA level should not be interpreted in isolation and cannot reliably distinguish malignant from infectious causes of ascites.

The analysis of ascitic fluid is an important component of the diagnostic evaluation of suspected tuberculous peritonitis. Conventional acid-fast bacilli microscopy has limited sensitivity because tuberculous peritonitis is frequently paucibacillary. Mycobacterial culture provides important microbiological confirmation but may require several weeks before results become available (1,6). These limitations may delay treatment, particularly in resource-limited settings.

GeneXpert MTB/RIF provides rapid molecular detection of *M. tuberculosis* complex DNA and simultaneously assesses rifampicin resistance. Although its sensitivity in ascitic fluid is lower than that observed in respiratory specimens, a positive result provides strong microbiological evidence of tuberculosis because of the high specificity of molecular detection (7,8). In this case, detection of *M. tuberculosis* in ascitic fluid by GeneXpert, together with the compatible clinical and radiological findings, facilitated early initiation of anti-tuberculous treatment.

The radiological findings further supported the diagnosis. Massive ascites, diffuse peritoneal thickening and enhancement, omental involvement, and necrotic mesenteric lymphadenopathy are recognized features of abdominal tuberculosis. However, similar findings may occur in peritoneal carcinomatosis and other intra-abdominal malignancies. The absence of a definite primary intra-abdominal mass and the absence of malignant cells on ascitic fluid cytology further reduced the likelihood of an underlying malignancy, although cytology cannot completely exclude malignant disease.

The combination of a markedly elevated CEA level and radiological features suggestive of peritoneal malignancy created a significant diagnostic challenge in this patient. Importantly, the positive GeneXpert result provided microbiological evidence supporting tuberculous peritonitis and allowed treatment to be initiated without waiting for mycobacterial culture results.

Tuberculosis remains an important public health problem in Timor-Leste. Diagnosis of extrapulmonary tuberculosis can be particularly challenging in settings where advanced imaging, tissue biopsy, histopathology, and specialized laboratory investigations are not readily available. This case demonstrates the importance of maintaining a high index of suspicion for tuberculous peritonitis in patients presenting with unexplained ascites, even when tumor markers are markedly elevated and imaging findings raise concern for malignancy.

This case also highlights the potential value of GeneXpert MTB/RIF testing of ascitic fluid in resource-limited settings. When interpreted together with the clinical presentation and radiological findings, a positive molecular test can provide rapid evidence of tuberculous infection and facilitate timely treatment.

LIMITATIONS

This report describes a single patient and therefore cannot establish the effectiveness of specific diagnostic or therapeutic strategies for tuberculous peritonitis.

Although the diagnosis was supported by the clinical presentation, radiological findings, and detection of *M. tuberculosis* in ascitic fluid by GeneXpert MTB/RIF, histopathological confirmation from peritoneal or abdominal tissue biopsy was not available. Tissue biopsy was not performed because of the limited availability of histopathological services in the local setting.

Ascitic fluid adenosine deaminase (ADA) testing was also not available. Although ADA can provide supportive evidence in suspected tuberculous peritonitis, its diagnostic performance varies according to the clinical setting and should be interpreted alongside microbiological, clinical, and radiological findings.

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The patient received standard anti-tuberculous treatment together with nutritional support and therapeutic paracentesis and subsequently demonstrated clinical improvement. However, the contribution of individual components of the management strategy cannot be determined from a single case.

Future studies involving larger patient populations and longer follow-up are warranted to evaluate the clinical characteristics, diagnostic approaches, treatment response, complications, and long-term outcomes of tuberculous peritonitis in Timor-Leste. Where resources permit, future studies should incorporate histopathological examination of peritoneal tissue and supportive investigations such as ascitic fluid ADA testing.

CONCLUSION

Tuberculous peritonitis can closely mimic intra-abdominal malignancy and should be considered in patients presenting with massive ascites, particularly in tuberculosis-endemic settings. Markedly elevated CEA levels may occur in tuberculosis and should not be interpreted as definitive evidence of malignancy. In this case, the combination of clinical findings, characteristic radiological features, absence of malignant cells, and positive GeneXpert MTB/RIF from ascitic fluid supported the diagnosis of tuberculous peritonitis.

Early consideration of tuberculosis and timely molecular testing of ascitic fluid may facilitate diagnosis and prompt initiation of treatment, particularly in resource-limited settings where tissue biopsy and mycobacterial culture are not readily available.

Declaration Of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/ her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest

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