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Case Report

Hidden in plain sight: omental tuberculosis disguised as ovarian cancer: a case report

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ABSTRACT

We present the case of a 39-year-old woman with no significant past medical history who developed a six-month history of bilateral lower limb pain, progressive weakness, weight loss, abdominal distension, and intermittent fever. Clinical examination revealed pallor, hepatomegaly, right-sided pleural effusion, and a firm pelvic mass. Radiological evaluation suggested peritoneal carcinomatosis with possible skeletal metastases; however, pleural fluid cytology was negative for malignant cells. Histopathological analysis of an omental biopsy revealed epithelioid granulomas with caseous necrosis, and Ziehl–Neelsen staining confirmed acid-fast bacilli, establishing the diagnosis of disseminated tuberculosis. The patient showed significant improvement with antitubercular therapy. This case points out the requirement of considering tuberculosis in presentations that mimic malignancy, particularly in high prevalence countries.

Keywords: Disseminated tuberculosis, Omental tuberculosis, Peritoneal TB, Pleural effusion, Malignancy mimic

INTRODUCTION

Tuberculosis remains one of the most important infectious diseases worldwide and continues to contribute substantially to morbidity and mortality, particularly in low- and middle-income countries. Although pulmonary involvement is the predominant form of disease, extrapulmonary tuberculosis constitutes a considerable proportion of cases and often presents with nonspecific clinical and radiological findings that create considerable diagnostic uncertainty.[1,2] Peritoneal tuberculosis is an uncommon manifestation of extrapulmonary disease and frequently mimics advanced intra-abdominal malignancy, especially ovarian carcinoma and peritoneal carcinomatosis.

Patients may present with constitutional symptoms, ascites, omental thickening, lymphadenopathy, and elevated inflammatory markers, all of which can closely resemble metastatic malignancy.[1,3] In endemic regions, failure to recognise these atypical presentations may lead to delayed diagnosis, unnecessary oncological interventions, and increased patient morbidity. This case demonstrates the diagnostic complexity of disseminated tuberculosis presenting as suspected metastatic cancer and emphasizes the central importance of histopathological confirmation before initiating definitive therapy.[4,5]

Peritoneal tuberculosis is a relatively rare presentation that can mimic various intraabdominal pathologies. Its clinical, radiological and sometimes biochemical similarity to peritoneal carcinomatosis can easily mislead clinicians. In countries with a high tuberculosis prevalence, awareness of such atypical presentations is important to prevent delayed diagnosis and inappropriate treatment.[1,2]

CASE REPORT

A 39-year-old woman presented with a six-month history of gradually progressive bilateral lower-limb pain, generalised weakness, loss of appetite, and significant unintentional weight loss. She also reported progressive abdominal distension during the same period, associated with intermittent low-grade fever and easy fatigability.

There was no history of cough, hemoptysis, gastrointestinal bleeding, night sweats, or prior known tuberculosis exposure. She had no menstrual irregularities or gynaecological complaints. There was no family history of malignancy or chronic illness.

On examination, the patient appeared pale, emaciated, and lethargic, with features suggestive of chronic disease. Vital signs were stable and the patient was afebrile at the time of assessment. Abdominal examination revealed visible distension with fluid thrill, a firm palpable pelvic mass, and tender hepatomegaly. No clinical evidence of lymphadenopathy was observed.

Respiratory examination showed decreased breath sounds and stony dullness on percussion over the right infrascular region, denoting a right-sided pleural effusion. Cardiovascular and neurological examinations were unremarkable; The combination of constitutional symptoms, ascites, palpable pelvic mass, hepatomegaly, and pleural effusion prompted consideration of a disseminated systemic process, with peritoneal carcinomatosis as the initial clinical suspicion.

Investigations

Initial haematological workup revealed microcytic anaemia, suggestive of chronic illness or nutritional deficiency, along with an elevated erythrocyte sedimentation rate (ESR) and mildly raised C-reactive protein (CRP), pointing towards an underlying inflammatory or infective process. Biochemical evaluation demonstrated mild derangement of liver enzymes, which was likely secondary to systemic illness and hepatic involvement.

Ultrasonography of the abdomen, as the first-line imaging modality, showed free fluid in the peritoneal cavity, omental thickening accompanied by hypoechoic nodules, hepatomegaly and right-sided pleural effusion. These features raised suspicion of peritoneal carcinomatosis. To further characterize these data, a contrast-enhanced computed tomography (CECT) scan of the abdomen and pelvis was performed, revealing omental nodularity, diffuse peritoneal thickening, multiple enlarged abdominal lymph nodes and lytic lesions in pelvic bones and vertebrae, findings highly suggestive of disseminated malignancy with skeletal metastasis.

Given the unexpected bony lesions, magnetic resonance imaging (MRI) of the pelvis and spine was performed to evaluate alternative causes of the lytic lesions and to better define soft-tissue involvement. MRI confirmed nodular omental deposits, pelvic lymphadenopathy and vertebral lytic lesions, further confirming the impression of a malignant process.

Pleural fluid analysis showed an exudative, lymphocyte-predominant effusion with elevated ADA levels, which raised the possibility of tuberculosis but did not exclude malignancy. Cytological examination of the fluid was negative for malignant cells, which lowered but did not completely rule out cancer.

A definitive tissue diagnosis was established by omental biopsy, which revealed epithelioid granulomas with Langhans giant cells and central caseous necrosis on histopathology. The Ziehl–Neelsen staining demonstrated acid-fast bacilli, confirming disseminated tuberculosis involving the peritoneum, pleura and bone. This histopathological confirmation was key in redirecting management from an oncological approach to anti-tubercular therapy, thus avoiding unnecessary chemotherapy or surgery.

Diagnosis

Disseminated TB involving the peritoneum, pleura, and bone.

Treatment and outcome

Following histopathological confirmation of tuberculosis, standard first-line anti-tubercular therapy was initiated under directly observed treatment, short course (DOTS) supervision. The regimen included isoniazid, rifampicin, pyrazinamide and ethambutol for an intensive phase of two months followed by isoniazid and rifampicin for a subsequent phase of four months. Due to skeletal involvement, the therapy was extended to a total duration of nine months (Figures 1-3).

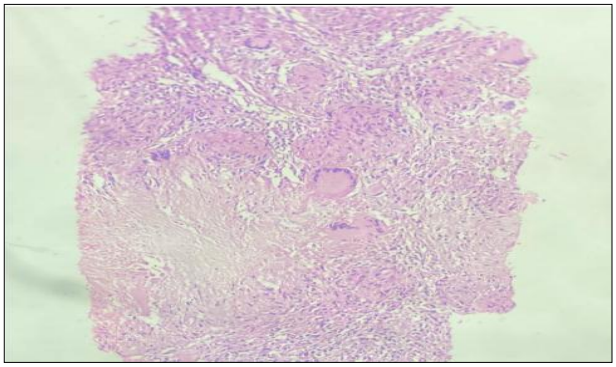

Figure 1: Granuloma composed of aggregates of epithelioid histiocytes, lymphocytes, Langhans giant cells and necrosis.

During the initial weeks of therapy, the patient experienced a marked clinical improvement, with a reduction in fever, an increase in appetite, and gradual weight gain. The abdominal distension progressively subsided, hepatomegaly decreased, and the pelvic mass was no longer palpable by the end of the intensive phase. Follow-up chest imaging showed near-complete resolution of the pleural effusion. Laboratory parameters improved with rising haemoglobin and normalisation of inflammatory. The patient was closely monitored for drug-related toxicity, including periodic liver function tests and

ophthalmological evaluation for ethambutol toxicity. Nutritional supplementation and iron therapy were administered to correct anaemia and improve overall health. At follow-up visits during the subsequent phase, the patient regained functional capacity and showed significant improvement in quality of life, supporting the diagnosis and showing the effectiveness of timely initiation of anti-tubercular therapy.

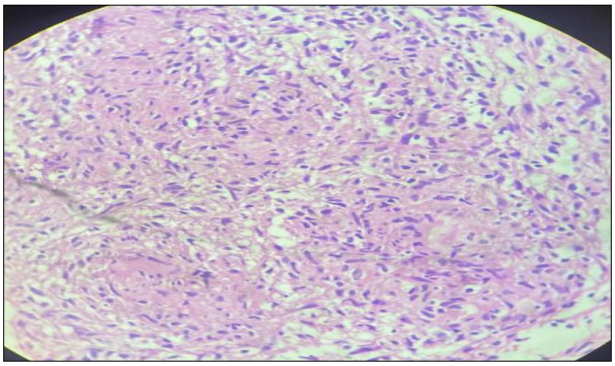

Figure 2: Confluent granulomas.

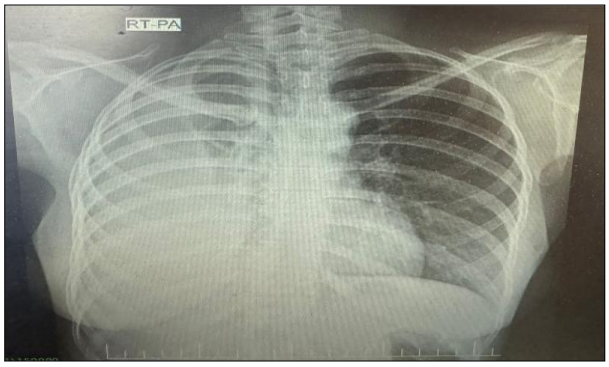

Figure 3: Chest X-ray showing massive right sided pleural effusion.

DISCUSSION

Disseminated and peritoneal tuberculosis continue to represent major diagnostic challenges because of their ability to imitate malignant, inflammatory, and autoimmune disorders both clinically and radiologically. The disease often evolves insidiously over several months, with constitutional symptoms such as fever, weight loss, anorexia, malaise, and progressive abdominal distension predominating during the early phase. These nonspecific manifestations frequently delay diagnosis and may initially direct evaluation toward occult malignancy.[1,2] In the present case, the coexistence of ascites, pleural effusion, omental nodularity, pelvic mass-like lesions, lymphadenopathy, and vertebral lytic lesions strongly suggested disseminated intra-abdominal carcinoma with skeletal metastasis. Such radiological overlap illustrates why peritoneal tuberculosis has long been described as a 'great mimicker' in clinical medicine.

The pathogenesis of peritoneal tuberculosis is multifactorial and can occur through hematogenous dissemination from a latent pulmonary focus, lymphatic spread from infected abdominal lymph nodes, ingestion of infected sputum or direct extension from adjacent gastrointestinal or genitourinary structures.[3] Importantly, many patients do not have active pulmonary disease at presentation, which adds to the complexity of the recognition. In endemic regions, clinicians therefore must maintain a high level of suspicion even in immunocompetent individuals without classic respiratory symptoms.

Radiological imaging serves an essential function in the initial assessment, but lacks specificity. Computed tomography findings such as omental caking, irregular peritoneal thickening, mesenteric nodularity, ascites and enlarged lymph nodes are often interpreted as carcinomatosis.[1,4] Similarly, elevated adenosine deaminase levels in pleural or ascitic fluid support the possibility of tuberculosis but are not independently diagnostic. Microbiological confirmation may also be difficult because extrapulmonary samples often contain low bacillary loads. Consequently, tissue biopsy remains the gold standard for diagnosis. The demonstration of epithelioid granulomas with caseous necrosis and Ziehl–Neelsen positivity in the present patient was decisive in redirecting management away from unnecessary oncological therapy toward curative anti-tubercular treatment.

The response to therapy further reinforced the diagnosis. Early initiation of standard anti-tubercular therapy resulted in marked clinical improvement, including resolution of fever, reduction of ascites, improvement in nutritional status and recovery of functional capacity. This case therefore emphasises not only the curable nature of extrapulmonary tuberculosis when recognised promptly, but also the potentially serious consequences of misdiagnosis. From a wider clinical perspective, the report stresses the importance of merging clinical judgment with radiology, pathology and microbiological evidence in order to avoid premature labelling of advanced malignancies. Such multidisciplinary diagnostic reasoning remains vital in areas where tuberculosis prevalence continues to be high.[2,5]

The clinical presentation is typically insidious, with symptoms gradually evolving over weeks to months. Constitutional symptoms such as low-grade fever, progressive weight loss, anorexia, malaise and fatigue are common but nonspecific, often leading to initial consideration of chronic infections, autoimmune diseases or malignancy. The development of abdominal distension, palpable masses, or pleural effusion usually prompts further evaluation, but imaging findings of omental thickening, peritoneal nodularity, ascites, and lymphadenopathy frequently suggest a malignant etiology, particularly ovarian or gastrointestinal carcinoma. The presence of lytic vertebral and pelvic bone lesions, as

observed in this case, further strengthens suspicion of metastatic spread, creating a significant diagnostic dilemma. This emphasizes the principle that tissue diagnosis must precede the initiation of cancer-directed therapy.[1,3]

A step-wise diagnostic approach is fundamental. Ascitic fluid analysis typically reveals a straw-coloured, lymphocyte-dominant exudate with high protein content and adenosine deaminase (ADA) levels typically above 40 IU/l, findings which strongly support tuberculosis in endemic regions. However, ADA can also be elevated in lymphoproliferative disorders and other inflammatory conditions, so it cannot be used in isolation. Microbiological confirmation is challenging because Ziehl–Neelsen staining has low sensitivity in peritoneal fluid, and culture is slow. GeneXpert MTB/RIF provides rapid molecular confirmation but has variable yield in extrapulmonary samples. Therefore, histopathological evaluation remains the diagnostic gold standard. In this case, omental biopsy revealed epithelioid granulomas with caseous necrosis, and Ziehl–Neelsen staining demonstrated acid-fast bacilli, thereby confirming the diagnosis and preventing unnecessary oncological intervention.[1,3]

Therapeutically, peritoneal TB is highly responsive to anti-tubercular therapy (ATT), with most patients demonstrating symptomatic improvement within weeks. The recommended regimen consists of two months of isoniazid, rifampicin, pyrazinamide, and ethambutol (2HRZE) followed by four months of isoniazid and rifampicin (4HR). In the presence of skeletal involvement, as in this case, treatment is often extended to 9–12 months to ensure complete eradication and prevent relapse. Supportive care is equally important — including nutritional rehabilitation, management of anaemia, symptomatic relief for ascites, and careful monitoring for drug-related hepatotoxicity or neuropathy. Timely initiation of therapy not only promotes clinical recovery as well as spares the patient the physical, psychological and monetary burden associated with being wrongly treated for malignancy.[3]

This case also illustrates the importance of a coordinated, multidisciplinary approach — bringing together internists, radiologists, pathologists, and microbiologists — to reach an accurate and timely diagnosis. It reinforces the teaching point that tuberculosis must always remain high on the list of differentials when evaluating chronic ascites and omental lesions in endemic areas. Awareness that TB is a "great mimicker" is crucial to avoid misdiagnosis and its downstream consequences, including unnecessary surgery, chemotherapy, and patient distress.[2,5]

CONCLUSION

This case shows that disseminated tuberculosis can look very similar to advanced intraabdominal cancer, which makes diagnosis difficult in places where tuberculosis is common. At first, the radiology results looked like peritoneal carcinomatosis with bone metastases, so doctors suspected metastatic ovarian or gastrointestinal cancer. However, by following a careful diagnostic process and insisting on a tissue diagnosis, the team found the real cause and changed the treatment plan. This case highlights how important it is to get a tissue diagnosis before starting cancer treatment, especially for patients with ascites, omental thickening, pleural effusion, and general symptoms in areas where tuberculosis is widespread. Starting anti-tubercular treatment quickly led to major improvement and helped avoid unnecessary chemotherapy and invasive procedures. This report also reminds us to consider tuberculosis as a possible diagnosis when a patient's symptoms look like cancer. Finally, it shows that working together across clinical medicine, radiology, pathology, and microbiology can help solve tough diagnostic problems and improve patient care.

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