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Abdominal lymphadenopathy: An atypical presentation of enteric fever

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ABSTRACT

This is a case report of a patient who presented to the Aga Khan University Hospital with generalized abdominal lymphadenopathy and high-grade fever. Due to ambiguous clinical findings, which were suggestive of either abdominal tuberculosis, or a lymphoma, the patient was started on empirical anti-tuberculous treatment due to the endemicity of tuberculosis in this region. The blood culture reports, however, were reported to grow colonies of *Salmonella paratyphi* A; thus the diagnosis of the patient was changed to enteric fever, and the patient improved on the subsequently started therapy of ceftriaxone 2000 mg bid. To the best of our knowledge, this is the first reported case of a patient suffering from enteric fever whose primary clinical findings were abdominal lymphadenopathy and fever.

1. Introduction

Enteric fever is a systemic disease caused by the bacterium *Salmonella typhi* (*S. typhi*). It has an estimated prevalence worldwide of 12–33 million cases and is a major problem especially in our part of the world including the Southeast Asia and Africa, where poor sanitation and lack of clean drinking water and food allow for easy spread of the pathogen^[1–3].

The bacterium grows intracellularly in the intestinal lymphoid tissue, presenting acutely with gastrointestinal symptoms and fever. The classic presentation is that of a prolonged fever, step-ladder in pattern, accompanied by malaise, abdominal pain and constipation in the first two weeks, followed by diarrhea in the third week^[4].

One of the intra-abdominal pathologies which may mimic typhoid infection is abdominal tuberculosis. Abdominal tuberculosis accounts for 11–16% of all extra-pulmonary tuberculosis cases, and predominantly affects the ileum and colon, though it may affect any part of the gastrointestinal system.

The pathogenesis itself can be explained by either the reactivation of a past dormant focus in the body or secondary to hematogenous or local spread from an infected organ. Swallowing of infected sputum is another postulated mechanism. Once established, the infection may manifest

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itself in a variety of ways, with the clinical spectrum ranging from completely asymptomatic to an acute abdomen to chronic non-specific symptoms of fever, weight loss, abdominal pain and diarrhea^[5].

We present the case of a young man suffering from enteric fever whose main presentation was abdominal lymphadenopathy. To the best of our knowledge, there have not been any reports documenting such an unusual presentation.

2. Case report

A 17 year old Pakistani male was admitted with high-grade fever associated with chills and rigors (40.5–41 °C) for 2 weeks prior to admission. During a course of oral antibiotics he had taken for 5 days, his symptoms had subsided, however the fever recurred on discontinuation of therapy.

On examination, the patient, a lean, thin individual, appeared toxic and dehydrated. He was tachypneic, tachycardiac and febrile at 38 °C, with an occasional spike up to 40 °C. He was started on symptomatic treatment. Biochemical tests on admission showed a low white blood cell (WBC) count, with neutrophilic predominance. His malaria work—up, conducted due to his non—specific presenting symptoms and its endemicity in Asia, was found to be negative. His liver function tests, however, were slightly deranged, with direct bilirubin of 0.5, Gamma—GT of 178, alkaline phosphatase of 205, and a lactate dehydrogenase of 2380.

A screening ultrasound done showed para-aortic, retroperitoneal and mesenteric lymphadenopathy, especially in

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the right iliac fossa and the aorto-caval regions. The CT scan of chest, abdomen and pelvis done to evaluate the extent of the lymphadenopathy showed significant thickening of the ileo-caecal junction, terminal ileum, caecum, and proximal ascending colon. It also showed aorto-caval, porta-hepatic, porto-caval lymph node enlargement, as well as mesenteric lymphadenopathy in the right iliac fossa (para-caecal). The lymph nodes were reported to be as large as 20×9 mm, 21×15 mm and 29×17 mm in the porta-hepatis, para-aortic, and right iliac fossa respectively. These radiological findings significantly increased the likelihood of the patient actually suffering from abdominal tuberculosis or lymphoma. The lymph nodes were considered to be too deep to access, even with ultrasound or CT guidance, thereby the plan to biopsy them was postponed unless necessary.

The ambiguous clinical findings seemed to lead to a diagnosis of either abdominal tuberculosis or a lymphoma. Due to the endemicity of the former in the region, as well as the extensive lymphadenopathy, it was thought likelier that the patient had abdominal tuberculosis, rather than lymphoma. Thus, the patient was started on empirical antituberculous therapy. However, 2 days later, the blood culture reports showed that the patient had actually been suffering from enteric fever, with the causative organism *Salmonella paratyphi* (*S. paratyphi*) A being sensitive to ampicillin, chloramphenicol, ceftriaxone, co–trimoxazole, and cefixime, and resistant to ciprofloxacin.

On the basis of the blood culture reports, the patient was re-admitted. His course of anti-tuberculous treatment was discontinued, and he was started on intravenous ceftriaxone 2000 mg BID as treatment of enteric fever for one week, followed by cefspan 400 mg for 2 weeks. His symptoms improved progressively, and within 4 weeks of diagnosis, his blood cultures were negative, the deranged biochemical markers had returned to normal and the previously visualized lymphadenopathy was not visible in the latest ultrasound, indicating resolution of the disease.

3. Discussion

Any patient presenting with fever of unknown origin in Pakistan is normally worked up for malaria, typhoid fever and tuberculosis, as these are the most prevalent in our part of the world. This case was unusual as this patient had diffuse lymphadenopathy. To the best of our knowledge, there has not been any report of a patient with enteric fever presenting with lymphadenopathy. In fact, it has been reported occasionally that if lymphadenopathy is present, then the suspicion of typhoid should decrease. Also, this patient did not have any disturbed gut motility. As in all atypical presentations, the only deciding factor was blood cultures.

Enteric fever in Pakistan is caused mostly by *S. paratyphi* A, and less commonly by *S. Typhi*. Timely diagnosis and prompt treatment is vital, as fatal complications may occur if it goes untreated. The diagnosis is primarily a clinical one, but a definitive diagnosis requires isolation from blood, bone marrow, stool or urine cultures[7], and the diagnosis of typhoid fever is not a simple one, due to its non– specific clinical features and lack of an immediate confirmatory test[8].

Initially the drugs of choice for enteric fever were ampicillin, chloramphenicol and co-trimoxazole, however when resistance developed to these drugs[8,9], ciprofloxacin was shown to be very effective. However, strains of *Salmonella* resistant to ciprofloxacin have started emerging, so empirical treatment of ceftriaxone is started on patients

who are considered likely to have treatment failure on ciprofloxacin as was done in our patient[10].

The variability and non–specificity of the symptoms of abdominal tuberculosis also makes this diagnosis a fairly challenging one to the physician, especially as the disease mimics the presentation of various diseases. A combination of investigations including *Mycobacterium* culture, imaging techniques using barium X–rays, ultrasound and CT scans, are conducted to reach a definitive diagnosis. In some cases such as ours, when even these are inconclusive, response to therapeutic trials of anti–tuberculosis therapy is the only basis of diagnosis^[11,12].

4. Conclusions

The possible variations in the clinical presentation of typhoid fever may make the early diagnosis and prompt treatment a challenging task for physicians. Thus, awareness of the possible aberrations in presentations is essential for a quick, correct diagnosis by the clinician.

Conflict of interest statement

We declare that we have no conflict of interest.

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