

A 12-year-old boy presented with fever and left-sided upper abdominal pain

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Article Info

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Presentation of Case

Dr. Mohuya Mondal: A 12-year-old boy of non-consanguineous parents presented with the history of fever and left sided upper abdominal pain for 1 month. His fever was high grade, intermittent, associated with chills and rigor. The abdominal pain was dull aching in nature without any radiation, aggravating or relieving factors. His bowel habit was normal. The child had no history of jaundice, cough, respiratory distress, burning sensation during micturition, earache, skin infection, contact with tubercular patient, blood transfusion or parenteral medication.

On examination, she was fretful, febrile, moderately pale, and anicteric. Tachycardia was present. Per abdominal examination revealed tenderness at the left hypochondriac region. There were splenomegaly (4 cm) and non-tender hepatomegaly (just palpable). Ascites was absent. Other systemic examinations were normal.

The complete blood count showed moderate anemia, neutrophilic leukocytosis and thrombocytosis (Table I). The erythrocyte sedimentation rate was high. Urine routine microscopy and widal test, serum alanine amino transferase were normal. Immunochromatography for kala-azar, immunochromatography for malaria and serology for dengue virus were negative. Blood and urine culture showed no growth of the organism. Montoux test was positive and the X-ray chest and abdomen were normal.

The ultrasonography of the abdomen showed spleen was enlarged, size-12.8 cm, multiple small hypoechoic lesion in splenic parenchyma, large one 8.2 x 0.6 mm, suggestive of multiple splenic abscess (Figure 1).

Based on history, examination findings and investigations, the diagnosis was splenic abscess. As other common causes of fever and upper abdominal pain were excluded (urinary tract infection, malaria, kala-azar, dengue) and the ultrasonography of the abdomen was suggested as splenic abscess.

Provisional Diagnosis

Splenic abscess most probably due to tuberculosis

Differential Diagnosis

Kala-azar

Dr. Md Rukunuzzaman: Prolonged fever with splenomegaly are the common presenting feature of kala-azar where the patient resides or travels into the kala-azar endemic area. Kala-azar associates with the hyperpigmentation, anemia, weight loss, hepatomegaly and abdominal discomfort due to splenomegaly. Kala-azar may be present with other co-infection like tuberculosis or abscess or inflammation to any other site of the body. Diagnosis of visceral leishmaniasis is made by the clinical features of the disease in an endemic area. Screening test is done by using "rK39 rapid diagnostic test". Some serological tests have been used to diagnosis the kala-azar include globulin content of the blood, aldehyde test, direct agglutination test and polymerase chain reaction to diagnose leishmaniasis. Some indirect tests are also available like antibody detection by enzyme linked immunosorbent assay and immunofluorescent antibody test. The diagnosis is confirmed by the demonstration of *Leishmania donovani* body from the spleen or bone marrow or lymph node aspiration or by doing polymerase chain reaction or culture from the aspirate.¹ The slit-skin smear or skin biopsy can be used in case of skin involvement.

Dr. Parisa Marjan: After evaluating the patient's presenting features, physical findings and the laboratory tests results, our patient had no history of residing or travelling to the kala-azar endemic area, weight loss or hyperpigmentation. Immunochromatography for kala-azar was negative and the blood count revealed no feature of cytopenia expect anemia. Ultrasonography of the abdomen showed the enlargement of spleen (size-12.8 cm), multiple small hypoechoic lesions within the splenic parenchyma (large one 8.2 x 0.6 mm), suggestive of multiple splenic abscess.



Table I

Investigation reports of patients

Parameters	Findings	References
Hemoglobin (g/dL)	8.7	12.5 - 14.5
White blood cell count ($\times 10^9$ /L)	11	4 - 11
Differentials		
Neutrophil (%)	62	40-80
Lymphocyte (%)	30	20-40
Eosinophil (%)	2	01-06
Monocyte (%)	6	02-10
Platelet count ($\times 10^9$ /L)	430	100-400
Erythrocyte Sedimentation Rate (mm in 1 st hour)	86	0-10
Urine routine microscopy		
Pus cell (cell/High power field)	1-2	0-5
Red blood cell (cell/High power field)	0	0-5
Widal test		
TO	<1:80	<1:80
TH	<1:80	<1:80
Serum alanine aminotransferase (U/L)	17	30-65
Montoux test (mm)	10	
Blood culture sensitivity	Negative	
Urine culture sensitivity	Negative	
Immunochromatography for kala-azar	Negative	
Immunochromatography for malaria	Negative	

Dr. Subarna Das: Why it is not a case of malaria?

Dr. Dipanwita Saha: Malaria is one of the common causes of fever with hepatosplenomegaly.² Suggestive clinical symptom with the history of travelling to the malaria endemic zone, the case is highly suspected of

malaria. Enlargement of the liver and spleen result from the inflammatory response to malaria parasitic infection.³ Demonstration of malaria parasite in blood film and rapid diagnostic test for detection of malaria parasite confirm the diagnosis of malaria. As our patient has no such travelling history to the malaria endemic zone, blood film does not show malaria parasite and immunochromatography for malaria is negative, so we exclude malaria.

Dr. Archana Shrestha Yadav: What are the causes of splenic abscess?

Dr. Marjan: Infection in other site of the body, like-pneumonia, otitis media, skin infection or abscess, urinary tract infection, osteomyelitis, meningitis, pelvic infection, etc.

Dr. Kamrun Nahar: What are the organism causing splenic abscess?

Dr. Dipanwita Saha: *Streptococcus viridians*, *Staphylococcus aureus*, *Mycoplasma*, *Haemophilus*, *Klebsiella*, *Salmonella*, *Micobacterium tuberculosis*, *E coli*, *Pseudomonas*, etc.⁴

Dr. Luthfunnahar: What are the risk factors responsible for the splenic abscess?

Dr. Mohuya Mondal: Abdominal trauma, infective endocarditis, hemoglobinopathy like- sickle cell disease and thalassemia, immunosuppressive condition, HIV infection, etc.⁵

Dr. Hazera Akter: What are the investigations you have to do to identify the risk factors and have you found any risk factor?

Dr. Marjan: We did blood culture and echocardiography for infective endocarditis, which were normal. Sickling test was done which was negative. Iron profile - blood iron, ferritin, total iron binding capacity were within normal range. Hemoglobin electrophoresis showed hemoglobin A1- 94.2%, hemoglobin A2- 8.8%, suggestive of B thalassemia trait, enzyme linked immune sorbent assay for HIV was negative. So, we found B thalassemia trait as a risk factor.

Dr. Sayma Munmun How thalassemia predisposed to splenic abscess?

Dr. Saha: Thalassemia patient has increased susceptibility to infection which attributed to immune abnormality. The patient has altered chemotaxis and phagocytosis of macrophage and neutrophil. There are impaired activity of T lymphocyte, decreased number and activity of natural killer cell and B lymphocyte, impaired immunoglobulin secretion and suppression of complement system.⁶

Dr. Saidul Islam Sumon: Sickle cell disease is the risk factor of splenic abscess. Why?

Dr. Mondal: Sickle-cell anemia occurs as a result of

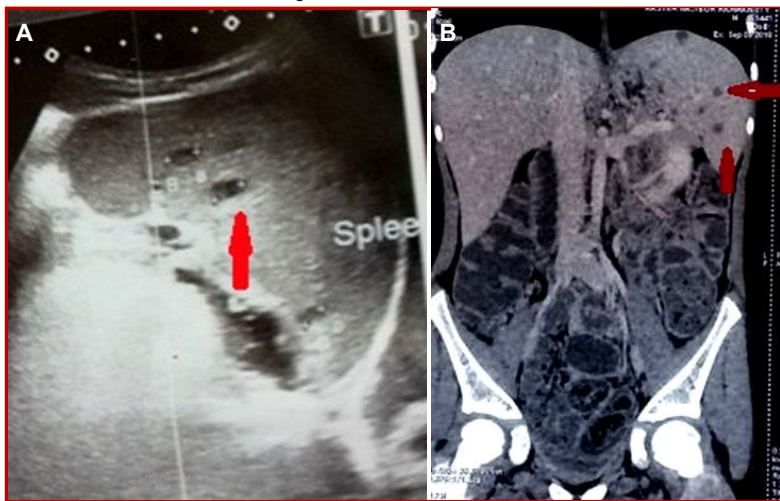


Figure 1: Ultrasonography (A) and CT (B) scan of the abdomen showing multiple splenic abscess (red arrows)

deformity of the hemoglobin. Here, there is a single change of amino acid, valine in exchange of glutamic acid at the 6th position of the hemoglobin of β -chain. This change leads to sequestration of the hemoglobin in the decreased oxygen saturation environment, resulting in deformed red cells and microvascular blockage. This causes the following results like cellular shrinkage, inflammatory consequences, and reperfusion damage which are the pathogenesis leading to splenic infarction. Produces mechanical asplenia, which makes them vulnerable to systemic infections and in the presence of infarction of spleen predisposes to splenic abscess.⁷

Dr. Subir Ananda Biswas: Why splenic abscess can occur in infective endocardites?

Dr. Marjan: Splenic abscess is a rare complication of infective endocardites. Abscess occurs secondary to hematogenous spread from an infective focus elsewhere in the body. Infective endocarditis can be associated with systemic embolization and is associated with about 10% incidence of splenic abscess.⁸

Dr. Rana Kumar Biswas: Why HIV infection predisposes the splenic abscess and which organism is mostly responsible?

Dr. Dipanwita Saha: Due to declining of CD4+ level and subsequent suppression of cell-mediated immunity, vulnerability to form splenic abscess, mostly by *Mycobacterium tuberculosis* bacilli.⁹

Dr. Fahmida Islam: What was the initial management given to the patient?

Dr. Mondal: We had given the conservative management. We gave injection ceftriaxone and injection flucloxacillin intravenously.¹⁰ Antipyretic and tepid sponging were given for fever.

Dr. Rafia Rashid: Have you taken surgical consultation and what was the decision regarding the aspiration of abscess or splenectomy?

Dr. Marjan: Yes but as the abscess were multiple, small in size and organized on ultrasonography and CT scan of the abdomen and having chance of bleeding due to its highly vascularity, surgery department advised to give conservative management and follow-up the patient subsequently.

Dr. Sharif Md. Habibur Rahman: What was the finding of subsequent follow-up?

Dr. Saha: After initial antibiotic therapy, the patient was febrile for 3 days and the abdominal pain was reducing. But after 3 days, the fever reappeared and the abdominal pain increased. So, after giving the antibiotic therapy for 2 weeks, as the patient remain symptomatic, we gave the antitubercular therapy.

Dr. Ruhina Tasmeen: On which basis the antituber-

cular therapy was given?

Dr. Mondal: As tuberculosis is very common in our country, the patient was not responding to the antibiotic therapy for 2 weeks and his erythrocyte sedimentation rate was raised and Montoux test was positive, so we started antitubercular therapy. Finally the patient was asymptomatic after giving 1 week of antitubercular therapy.

Dr. Naznin: What is the fate of untreated splenic abscess?

Dr. Marjan: Untreated abscess may rupture into the peritoneal cavity causing peritonitis. It may erode diaphragm and rupture into the pleural cavity or may erode colon.¹¹

Dr. Fahmina: What was the management regarding thalassemia?

Dr. Saha: After consulting with the Pediatric Hemato-oncologist, we gave the nutritious diet, folic acid supplementation, maintenance of hygiene. If required, the child was advised to give blood transfusion and iron chelator therapy.¹²

Dr. Mondal's Diagnosis

Splenic abscess

Discussion

Dr. Md. Rukunuzzaman: The splenic abscess is very uncommon in children. The incidence rate ranges 0.2 to 0.7%. It may be defined as single or multiple intraparenchymal or subcapsular collections of purulent discharge in the splenic parenchyma.¹³⁻¹⁴

The diagnosis of splenic abscess is often delayed in children due to its rarity and non-specific clinical features and polymorphic symptomatology.¹⁵ The splenic abscess may be single or multiple (24.8% cases). The splenic abscess formation is prevented by the phagocytic process of the reticuloendothelial system and white cells. The defective self protection mechanism of the host, infective endocarditis, splenic injury, sickle cell anemia, thalassemia, and immune suppression aggravates to splenic abscess formation. The common causative agents are *Staphylococci*, *Streptococci*, *Enterococcus* and *Klebsiella*, *Escherichia coli*, *Pseudomonas*, *Salmonella typhi*, *Candida* and *Mycobacteria*.¹⁶⁻¹⁷

Tuberculosis is a very common in developing countries like Bangladesh, but it is very uncommon in primary involvement of spleen.¹⁸ It is such a disease which can involve any organ or site. This condition mimics other abdominal pathology due to its non-specific feature. Abdominal involvement may occur in the gastrointestinal tract, peritoneum, lymph

nodes or solid viscera. Isolated involvement of the abdominal solid organ occurs in 15-20% of all patients with abdominal tuberculosis. Only 15% of patients have concomitant pulmonary tuberculosis. The clinical features may vary in abdominal tubercular patient according to different type.¹⁹ In tubercular splenic abscess, a probable mechanism might be that the red pulp of the spleen is relatively devoid of phagocytic activity and helps the entrapped, slow-growing mycobacteria to escape from the reticuloendothelial system of the spleen.

A high index of clinical suspicion is required for making a prompt diagnosis.²⁰ The classical clinical features include fever, chills, pain in the left hypochondrium and splenomegaly. Erythrocyte sedimentation rate may be high. A plain X-ray abdomen is usually non-informative. An abdominal ultrasound is the most commonly useful investigation that will demonstrate one or more hypo-echogenic image within the spleen which can contribute to an early diagnosis. Any intra-abdominal collection can also be identified. Computed tomography has now become the gold standard for the diagnosis of splenic abscess because of its high sensitivity and specificity. Both the modalities can be used for guided diagnostic and therapeutic aspiration.²¹⁻²³ Tuberculosis can be histopathologically marked by the presence of caseatous necrosis with granuloma formed by the epithelioid cells and multinucleated giant cells.²⁴⁻²⁷

The management of splenic abscess in children aims to preserve of the splenic tissue is for its immunological functions. The treatment could be done by antitubercular therapy with the consideration of splenectomy depending upon the response.²⁸ Isolated abscess of spleen in children can be well treated by needle aspiration and medication without splenectomy.^{29,30} Splenectomy is advocated as the standard management of splenic abscess. Complete or partial removal of the spleen is considered for failed percutaneous drainage of the abscess, multi-loculated and more than single abscess of spleen. The current treatment advocates the conservative management for isolated abscess by percutaneous drainage for preservation functions of the spleen in children if possible.³² Percutaneous drainage has fewer morbidity and post-splenectomy complications like infection and less immunological response. Splenic tuberculosis should be managed by antitubercular drugs as the first-line therapy with consideration of splenectomy as per response.^{31,33}

Final Diagnosis

Tubercular splenic abscess with thalassemia trait

References

1. Daher EF, Evangelista LF, Silva junior GB. Clinical presentation and renal evaluation of human visceral leishmaniasis (kala-azar): A retrospective study of 57 patients in Brazil. *Braz J Infect Dis.* 2008; 12: 329-32.
2. Ahammad F, Ali MY, Rahman SH. Aetiology of pyrexia with hepatosplenomegaly: Study of 50 cases in MMCH (Bangladesh). *Faridpur Med Coll J.* 2010; 5: 7-10.
3. Mangal P, Mittal S, Kachhawa K. Analysis of the clinical profile in patients with plasmodium falciparum malaria and its association with parasite density. *J Glob Infect Dis.* 2017; 9: 60-65.
4. Mustafa M, Menon J, Muniandy RK. Splenic abscess: Etiology, clinical spectrum and therapy. *IOSR J Pharm.* 2015; 5: 37-41.
5. Fotiadis C, Lavranos G, Patapis P, Karatzas G. Abscesses of the spleen: Report of three cases. *World J Gastroenterol.* 2008; 14: 3088-91.
6. Ricerca BM, Girolamo AD, Rund D. Infections in thalassemia and haemoglobinopathies: Focus on therapy-related complications. *Mediterr J Hematol Infect Dis.* 2009; 1: e2009028.
7. Al-Salem AH. Splenic complications of sickle cell anaemia and the role of splenectomy. *ISRN Hematol.* 2011; 2011: 1-7.
8. Elasar A, Al-Baradai A, Al-Harfi Z. Splenic abscess associated with infective endocarditis: Case series. *J Saudi Heart Assoc.* 2015; 27: 210-15.
9. Kalantari S, Paydary K, Soleimani A. Isolated splenic tuberculosis in a HIV-positive patient: A case report. *Arch Clin Microbiol.* 2011; 2: 3823.
10. Liu YH, Liu CP, Lee CP. Splenic abscesses at a tertiary medical center in Northern Taiwan. *J Microbiol Immunol Infect.* 2014; 47: 104-08.
11. Gupta S, Singh O, Hastir A. Splenic abscesses: Reports of two cases with review of the literature. *Ann Trop Med Public Health.* 2012; 5: 273-77.
12. Musallam KM, Rivella S, Vichinsky E, Rachmilewitz EA. Non-transfusion-dependent thalassemias. *Haematologica* 2013; 98: 833-44.
13. Chiang IS, Lin TJ, Chiang IC. Splenic abscesses: Review of 29 cases. *Kaohsiung J Med Sci.* 2003; 19: 510-15.
14. Nasr B, Chhaider A, Mabrouk MB. Diagnosis and treatment of splenic abscess (about 11 cases). *J Gastroenterol Hepatol Res.* 2013; 2: 9.
15. Faruque AV, Qazi SH, Arshad M. Isoated splenic abscess in children, role of splenic preservation. *Pediatr Surg Int.* 2013; 29: 787-90.
16. Kanwal SK, Kaur S, Kumar V. Multiple splenic abscesses in a child with enteric fever treated by percutaneous aspiration and antibiotics. *Trop Med*

- Public Health. 2012; 5: 245-47.
17. Lee MC, Lee CM. Splenic abscess: An uncommon entity with potentially life-threatening evolution. *Can J Infect Dis Med Microbiol.* 2018; 8610657: 6.
 18. Hasan M, Sarwar J, Bhuiyan J. Tubercular splenic abscess. *Mymensingh Med J.* 2008; 7: 67-69.
 19. Rai S, Thomas WM. Diagnosis of abdominal tuberculosis: The importance of laparoscopy. *J Roy Soc Med.* 2003; 96: 586-88.
 20. Dbi U, Ravisankar V, Prasad KK. Abdominal tuberculosis of gastrointestinal tract: Revisited. *World J Gastroenterol.* 2014; 20: 14831-40.
 21. Baron MJ, Kasper DL, Jameson JL. Intra-abdominal infections and abscesses. *Harrison's Principles of internal medicine.* 18th ed. New York, McGraw Hill Company, 1998, p 1082.
 22. Mohta A, Sharma SK, Sinha SK. Splenic abscess in children: A report of three patients. *J Indian Assoc Pediatr Surg.* 2003; 8: 113-15.
 23. Green BT. Splenic abscess: Report of six cases and review of the literature. *Am J Surg.* 2001; 67: 80-85.
 24. Divyashree S, Gupta N. Splenic abscess in immunocompetent patients managed primarily without splenectomy: A series of 7 cases. *Perm J.* 2017; 21: 16-139.
 25. Suri R, Gupta S, Gupta SK. Ultrasound guided fine needle aspiration cytology in abdominal tuberculosis. *Br J Radiol.* 1998; 71: 723-27.
 26. Fukunaga H, Murakami T, Gondo T. Sensitivity of acid-fast staining for mycobacterium tuberculosis in formalin-fixed tissue. *Am J Respir Crit Care Med.* 2002; 166: 994-97.
 27. Haq Z, Mahbub MS, Miah MT. Tuberculosis at an unusual site. *Bangladesh J Online.* 2010; 11: 67-69.
 28. Fooladi AA, Hosseini MJ, Azizi T. Splenic tuberculosis: A case report. *Int J Infect Dis.* 2009; 13: 273-75.
 29. Ray S, Kundu S, Goswami M. Isolated tubercular splenic abscess: Can we defer splenectomy? Our single experience with antituberculous therapy alone. *Indian Assoc Med Microbiol.* 2012; 30: 101-03.
 30. Sharma SK, Smith-Rohrberg D, Tahir M. Radiological manifestations of splenic tuberculosis: A 23-patient case series from India. *Indian J Med Res.* 2007; 125: 669-78.
 31. Ng CY, Leong EC, Chng HC. Ten-year series of splenic abscess in a general hospital in Singapore. *Ann Acad Med Singapore.* 2008; 37: 749-52.
 32. Jaiswal CS, Talreja LC, Chawla SL. Organ preservation in splenic abscess. *Med J Armed Forces India.* 2014; 70: 195-97.
 33. Dixit R, Ayra MK, Panjabi M. Clinical profile of patients having splenic involvement in tuberculosis. *Indian J Tuberc.* 2010; 57: 25-30.
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