Acta Persica Pathophysiologica

Inferior vena cava thrombosis in a patient with abdominal tuberculosis

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ARTICLE INFO

Article type:
Case Report

Article history:
Received: 10 December 2017
Accepted: 10 January 2018
Published online: 5 February 2018

Keywords:
Abdominal tuberculosis
Inferior Vena Cava thrombosis
Hypercoagulability

ABSTRACT

Tuberculosis (TB) is one of the most widespread infectious diseases and a major health problem of the developing world, responsible for a large number of deaths each year. Isolated inferior vena cava thrombosis is a rare complication of abdominal TB. Venous thrombosis occurs due to either local invasion directly damaging the endothelial lining or by inducing hemostatic changes through acute phase reaction that causes a hypercoagulable state. Drug treatment with anti-TB drugs and anticoagulants such as low molecular weight heparin are effective treating the TB with coagulative disorders.

Case Report

Core tip: Patients with abdominal TB have an increased risk of developing inferior vena cava thrombosis which can cause hepatic and other systemic thromboembolic complications.

Please cite this paper as: Majid Z, Bux Soomro G, Bux A, Mandhwani R, Luck NH, Mubarak M. Inferior vena cava thrombosis in a patient with abdominal tuberculosis.. Acta Persica Pathophysiol. 2018;3:e01.

Introduction

Tuberculosis (TB) is one of the most widespread infectious diseases and a major health problem of the developing world, responsible for a large number of deaths each year. Isolated inferior vena cava thrombosis is a rare complication of abdominal TB (1).

Case Presentation

A-12-year old female referred to the outpatient department of the hospital with a history of intermittent diarrhea since a year ago. Her loose stool occurred 4 to 6 times in a day, with watery in consistency, small in amount, yellowish in color and contained no mucous or blood. Along with this complaint, there was a history of gradual onset of periumbilical pain, which was mild to moderate in intensity and dull aching in nature. History of weight loss and low-grade intermittent fever was also present. There was no history of night sweats. There was no family history of TB too.

On examination, the patient appeared pale, dehydrated. Her systemic examination was unremarkable, with no lymphadenopathy in examination.

Initial laboratory findings were unremarkable apart from a raised ESR of 100 mm/h in first hour. Her chest x-ray was normal. We proceeded with a tuberculin test, which revealed an induration greater than 10 mm. Her tissue transglutaminase (tTG) serology was sent and turned out to be negative. Upper gastrointestinal (GI) endoscopy was normal. Then, we conducted an abdomen CT scan which showed thickening of the bowel wall involving terminal ileum, cecum and proximal ascending colon along with multiple enlarged calcified necrotic lymph nodes, detected in porta-hepatic, peri-pancreatic and mesenteric regions. The patient was underwent colonoscopy which revealed areas of ulceration, erythema and luminal narrowing in the ascending colon. Biopsy from involved area showed non-specific inflammatory changes.

She was empirical started on anti-tuberculous therapy (ATT) although histopathological report showed non-specific inflammatory changes. A second abdominal CT suggested a decrease in the mucosal thickening compared to the first abdominal CT scan. However, a recently

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developed thrombosis in IVC was seen in the intra-hepatic portion of the liver. This finding was confirmed by Doppler study of the liver vessels.

Thrombophilia profile was requested which showed decreased levels of antithrombin III and protein S levels. According to this findings, anticoagulants (heparin followed by warfarin) were started. One month after the scheduled combined treatment with ATT and anticoagulants, the Doppler US abdomen showed resolution of the inferior vena cava (IVC) thrombus. A few months later, the colonoscopy was repeated, with new findings of stricture formation in ascending colon. The stricture of ascending loop of colon was dilated by CRE balloon. After this procedure she electively underwent right hemicolectomy with ileocolic anastomosis. Biopsy of one lymph node was taken which was later confirmed as TB (Figure 1).

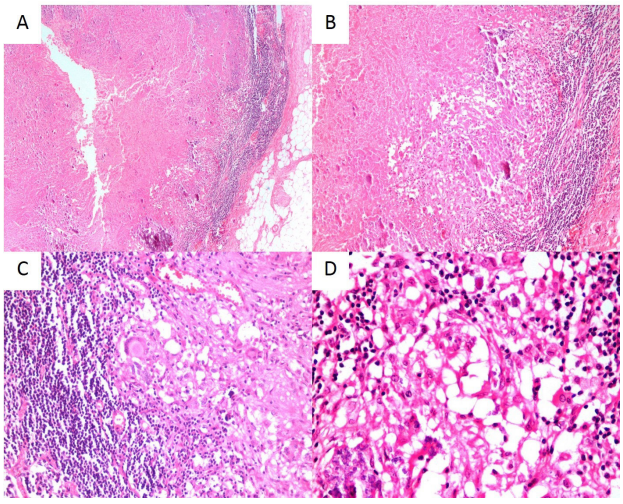

Figure 1. Histopathology of the lymph node. A. Low-power view showing a lymph node with large areas of caseation necrosis. (H&E ×40). B. Medium-power view showing ill-formed epithelioid granuloma at the interface between caseation necrosis and rim of lymphoid tissue (H&E ×200). C. Medium-power view showing an epithelioid granuloma with Langerhans giant cells (H&E ×200). D. High-power view showing poorly formed epithelioid granulomas surrounded by a sparkling of lymphocytes (H&E ×400).

Discussion

TB rarely may present with deep vein thrombosis (DVT). Although temporary increase in anti-cardiolipin antibodies, acute phase reactants along with hemostatic changes have been implicated in the formation of DVT in pulmonary TB. An early diagnosis along with immediate treatment is necessary since venous thromboembolism (VTE) can be life threatening (1).

Thrombus in pulmonary TB is mainly seen at the hepatic veins, central retinal veins, IVC along with the cerebral venous sinus (2).

The presence of DVT is directly proportional to the severity of TB (3), while it present in the early stage of the disease. This is mainly due to the hypercoagulability state along with the systemic inflammatory condition detected in TB. An increase in C4b binding protein that binds protein S also cause a hypercoagulable state. Another important association is between DVT and rifampicin, a drug commonly used in the treatment of TB (4).

Ganglionic forms of TB may compress IVC through enlarged lymph nodes and may lead to thrombus formation.

According to the hypercoagulable state seen in TB, patients should be anticoagulated with heparin prophylactically.

In cases of unexplained DVT, retroperitoneal TB lymphadenitis should be considered as a possible cause (5).

Thrombophilia due to hereditary causes occurs in patients under the age of 45 years. This condition may present as recurrent venous thrombosis at uncommon sites (6). A complete thrombophilia laboratory profile should be considered when IVC thrombosis is seen in young individuals (7).

The diagnosing of abdominal TB will be conducted by the biopsy of the peritoneal nodules, along with PCR of the ascites fluid (8).

Conclusion

Patients with abdominal TB have an increased risk of developing inferior vena cava thrombosis which can cause hepatic and other systemic thromboembolic complications. Treatment with anticoagulant along with ATT will treat the disease.

Since an IVC thrombosis is a rare complication of abdominal TB, it should be suspected in patients who do not respond well clinically with the initial anti-TB treatment.

Authors' contribution

ZM and GBS managed the patient and wrote the first draft. AB and RM wrote the final draft.NHL edited the patient.MM was responsible for the histopathology images.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors. The patient has given her informed consent regarding this case report.

Conflicts of interest

There were no points of conflicts.

Funding/Support

None.

References

1. Shah PA, Yaseen Y, Malik AH. Pulmonary tuberculosis with deep venous thrombosis. Webmed Central General Medicine 2011;2:WMC002093. http://www.webmedcentral.com/article_view/2093. Accessed March 12, 2013.
2. Kouismi H, Laine M, Bourkadi J, Iraqi G. Association of deep venous thrombosis with pulmonary tuberculosis. Egypt J Chest Dis Tuberc. 2013;62:541-3.
3. Ortega S, Artiles Vizcaíno J, Balda Aguirre I, Melado Sánchez P, Arkuch Saade ME, Ayala Galán E, et al. Tuberculosis as risk factor for venous thrombosis. An Med Interna. 1993;10:398-400.
4. White NW. Venous thrombosis and rifampicin. Lancet. 1989;2:434-435
5. Gogna A, Pradhan GR, Sinha RS, Gupta B. Tuberculosis presenting as deep vein thrombosis. Postgrad Med J. 1999; 75:104-5.
6. Wilde JT. The investigation of a patient with unexpected venous thrombosis. Postgrad Med J. 1995;71:720-4.
7. McAree BJ, O'Donnell ME, Fitzmaurice GJ, Reid JA, Spence RA, Lee B. Inferior vena cava thrombosis: a review of current practice. Vasc Med. 2013;18:32-43. doi: 10.1177/1358863X12471967.
8. Sayadinia M. Abdominal tuberculosis and thrombosis of inferior vena cava: a case report. Hormozgan Med J. 2015;18:547-552.