Spontaneous splenic rupture due to splenic vein thorombosis

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Abstract

Medical causes for spontaneous rupture of a normal spleen is rare. Ultrastructural abnormality of veins with unexplained spontaneous thrombosis of splenic vein is a rare condition. All long forgotten cause of splenic rupture is enteric fever, which after the incoming of antibiotic era has significantly reduced. But, enteric fever should also be not forgotten as one of the cause of spontaneous splenic rupture. One of the rare complications occurring in enteric fever is venous thrombosis. Splenic vein thrombosis due to Anomalous bifurcation of the left tibio-peroneal artery and/or enteric fever which subsequently ruptured the spleen due blockage of afferent flow of blood is almost not yet reported We present a rare case of splenic rupture due to spontaneous splenic vein thrombosis.

Key words: Splenic rupture, Splenic vein thrombosis, Enteric fever,Anomalous bifurcation of the left tibio-peroneal artery.

Introduction

May-Thurnersyndrome(MTS) is a rarely diagnosed condition in which patients develop iliofemoral deep venous thrombosis (DVT) due to an anatomical variant in which the right common iliac artery overlies and compresses the left common iliac vein against the lumbar spine. This variant has been shown to be present in over 20% of the population [1]. More recently, a similar prevalence (22%–24%) of MTS was reported in a retrospective analysis of computed tomography scans in japan[9].Similarly, many undiagnosed anomalous condition of venous system still exist in our population and diagnosis is still an accidental finding.

In 2000, it was estimated that over 2.16 million episodes of typhoid occurred worldwide, resulting in 216 000 deaths, and that more than 90% of this morbidity and mortality occurred in Asia[2]. Although improved water quality and sanitation constitute ultimate solutions to this problem, vaccination in high-risk areas is a potential control strategy recommended by WHO for the short-to-intermediate term [3]. Although the disease is not common in industrialised countries, it remains an important and persistent health problem in developing nation [4]. Hospital-based studies and outbreak reports from India indicate that enteric fever is a major public health problem in this country, with Salmonella enterica serovarTyphi*(*S. Typhi) the most common aetiologic agent but with an apparently increasing number of cases due to *S.* ParatyphiA (SPA). Because risk factors such as poor sanitation, lack of a safe drinking water supply and low socio economic conditions in resource-poor countries are amplified by the evolution of multidrug resistant salmonellae with reduced susceptibility to fluoroquinolone, treatment failure cases have been reported in India, which is associated with increased mortality and morbidity [5].

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Case report

Here we present a case of a 27 year old female patient who was admitted to casuality of Rajarajeswari medical college and hospital on 22-09-2013 with complaints of fever for 15 days and dark skin lesions in the left lower limbs for 7 days. She was treated elsewhere for typhoid fever. But, since the fever and clinical condition did not improve, she had come for admission in our hospital. Patient had history of generalized fatigue and headache for 3 days. No other associated symptoms suggestive of respiratory system, cardiovascular system, central nervous system involvement. Past history was not significant. She is a mother of 10 month old male baby. No history of recurrent abortions.

On examination at the time of admission, patient was conscious, oriented, co-operative, moderately built and nourished with height of 154 cm, weighing 56 kg. Her pulse was 92beats/min, blood pressure was 90/62 mm Hg. Temperature was 101'F, had pallor, 3 punched out lesions of size 1 cm with surrounding erythema + in left lower limb. Per abdomen examination showed mild splenomegaly about 3 cm below the left costal margin. Other systemic examination was normal (Figure 1).

On investigation, patient's hemoglobin was 8.5g%, RBC count was 2,76,000 cells/cu mm, MCV was 91.3fl, ESR was 30 mm at the end of one hour. Total leucocyte count was 18,400 cells/cu mm and platelet count was 5,000 cells/cu mm.Widal was positive S.typhi 'O' – 1:160 and S.typhi 'H'- 1:160, Dengue serology – negative, smear for MP- negative, CRP-negative, HIV 1 AND 2- negative, Renal function test – within normal limits, liver function test – Total bilirubin-1.3mg/dl, direct bilirubin-0.7mg/dl, SGOT-254 U/L, SGPT-297U/L, ALP-139U/L, Sr.TOTAL PROTEIN-5.5g/dl, Sr.Albumin-2.9 g/dl, Na^+-128 meq/l, K^+-3.9meq/l, Cl^--99meq/l. Treated for typhoid withinj.ceftriaxone antibiotic and symptomatic treatment was given. Six random donor platelets were transfused. Patient improved clinically, on 3rd day of hospitalization, patient was afebrile, vitals stable, platelet counts – 50,000 cells/cu mm.

On the 5th day of hospitalization, patient developed sudden onset of abdominal pain, left hypochondrial region to begin with later on it was diffuse, with tachypnea and tachycardia, severe pallor followed by abdomen distension within 3 to 4 hours. An Emergency Ultrasound abdomen showed free fluid in the abdomen. A diagnostic ascitic tap revealed haemorrhagicaspiration, organomegaly cannot be made out. Patient was immediately shifted to ICU, planned to do emergencylaparotomy to look for suspected intestinal perforation, haemorrhage. Laparotomy revealed ruptured spleen which was three time larger than normal spleen with thrombosed splenic vein.The ruptured spleen was removed and the patient survived the procedure.Patient was conscious, oriented, vitals stable post operatively. Early ambulation of patient was started. Rare causes for splenic vein thrombosis (lower limb venous thrombosis thought and send for special blood investigations) (Figure 2).

On the 5th post-operative day, patient developed pain and swelling of left lower limb. On examination, patient's vitals stable, left lower limb swelling upto the thigh, local temperature increased. Emergency bedside venous and arterial Doppler studyand subsequent CT-angiogram of the left lower limb showed:multiple venous thrombosis in left external iliac, femoral, popliteal, tibial, anterior tibial and posterior tibial veins, Long saphenous vein; Arterial- Anomalous bifurcation of the left tibio-peroneal trunk noted with bifurcation distally just above the ankle joint. Patient was started on anti-coagulants.Patient improved symptomatically on the 13th post-operative day. A repeat ultrasound venous and arterial Doppler of left lower limb showed: normal study. (Figure 3).

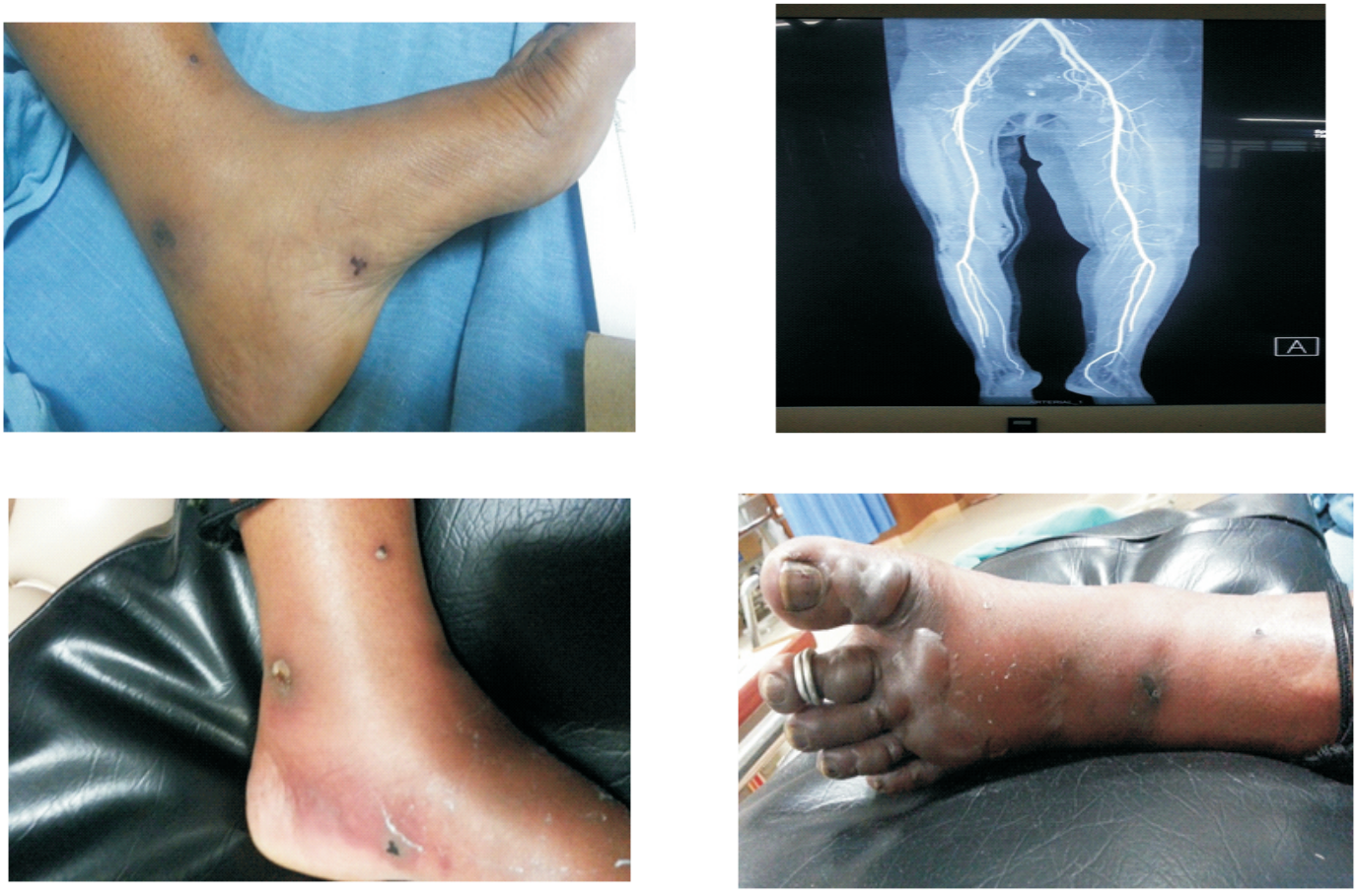

Figure 1. Multiple purpuric lesions.

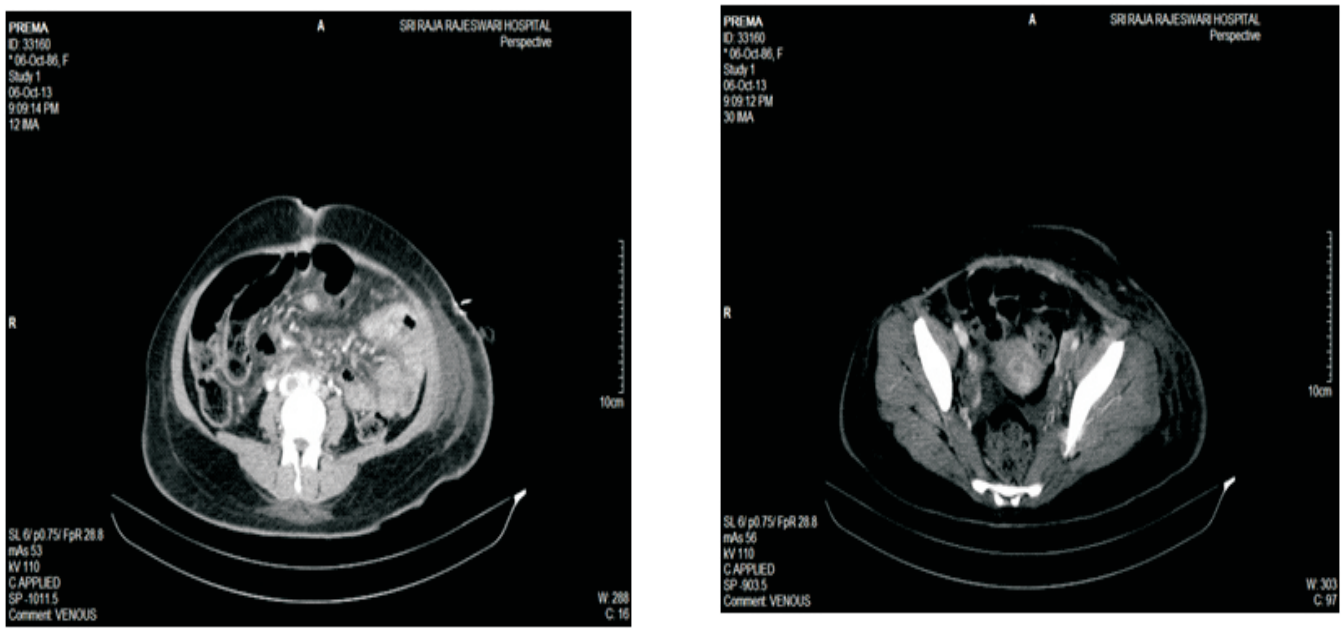

Figure 2. CT-angiography of lower limbs

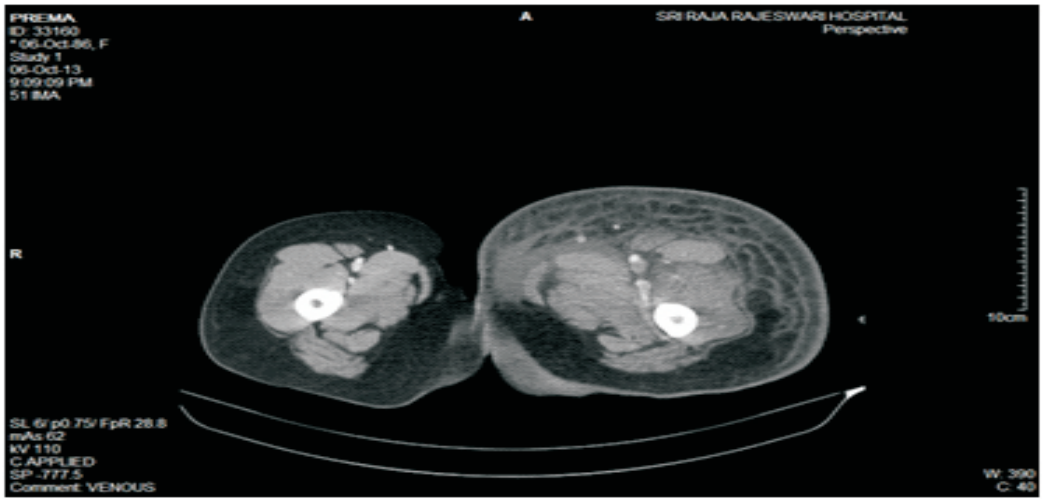

Figure 2. (Contd.) CT-Angiography of lower limb- Inferior vena cava, Iliac Vein, Femoral Vein Thrombus

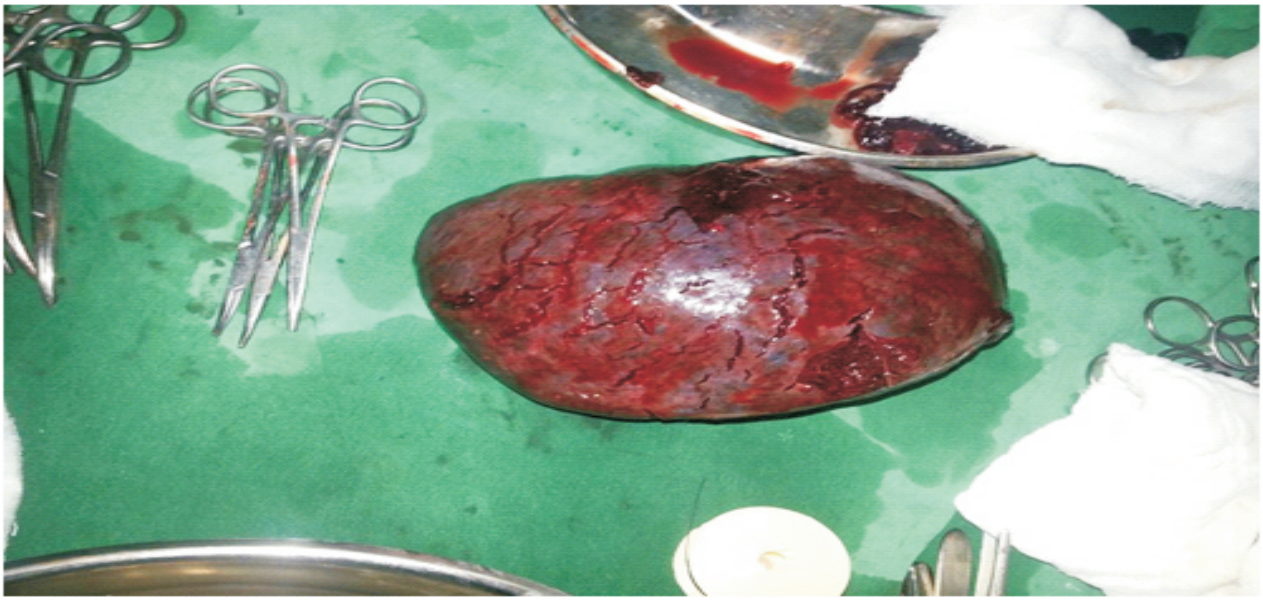

Figure 3. Emergency laparotomy revealed splenic rupture and thrombosed splenic vein

Discussion

May-Thurner syndrome, also known as iliac vein compression syndrome, Cockett syndrome, or iliocaval compression syndrome is caused when the left iliac vein is compressed by the right iliac artery, which increases the risk of deep vein thrombosis (DVT) in the left leg [10-12]. A history of persistent left lower extremity swelling with or without deep venous thrombosis in a woman between the 2nd and 4th decades of life, without an obvious cause, is highly suggestive of May-Thurner syndrome. The clinical suspicion can be confirmed with CT and iliac venography [11]. Similar compression syndrome may also happen due to compression of underling venous structures by distal left tibioperoneal trunk which has abnormal bifurcation. Since the underlying corresponding vein gets compressed, similar mechanism of May-Thurner syndrome, Cockett syndrome could have occurred. This is the only possible anatomical anomaly of lower limb arteries which can predispose to multiple venous thrombosis in my patient (Figure 4).

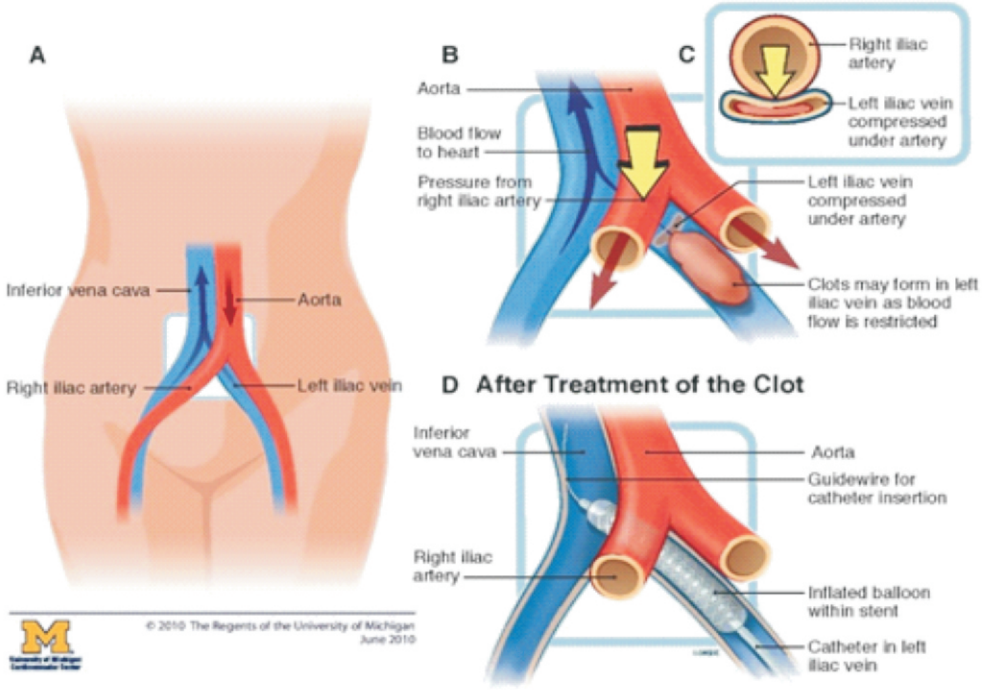

Figure 4. May Thurner Syndrome/Left Iliac Vein Compression with Clot (Thrombus and Stent Repair

Venous thrombosis due to enteric fever is long forgotten complication after the emergence of the antibiotic era, and often occurred in the fourth week of the illness [6]. Thrombosis mainly occurs in leg veins in the course of disease [7]. It's usually confined to left side and involves veins of calf. Involvement of femoral vessels may occur though not commonly. Earlier on involvement of femoral vein, long saphenous vein and popliteal veins was common but now with better care, the incidence of venous thrombosis has remarkedly decreased. S.Typhi has been isolated in some cases from the venous clot. In addition to venous thrombosis, rarely arteritis involving vessels of lower limbs or upper limbs may develop.

Conclusion

Thus anomalous left tibio-peroneal trunk can predispose to multiplevenous thrombosis and enteric fever has history to aggravate venous thrombosis. Both co-incidence in this patient such as mine has predisposed to sudden multiple venous thrombosis including the splenic vein, splenomegaly and has eventually ruptured the spleen. Such a life threatening incident can happen to any patient as the anatomical anomalies goes undetected in almost all patients unless it presents with unusual signs and symptoms. So the main purpose of this case report is to convey the information that multiple punched out skin lesions with swelling of left lower limb should not be ignored. An immediate Ultrasound Doppler study of lower limb arteries and veins should be done to rule out multiple venous thrombosis and congenital anomalous arteries. So that anti-coagulation therapy, if necessary can be started before the occurrence of further complications and life threatening events.

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