CASE REPORT

Clinical management of advanced schistosomiasis: a case of portal vein thrombosis-induced splenomegaly requiring surgery

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SUMMARY

We report for the first time in the Philippines a case of portal vein thrombosis in a 12 year old Filipino boy with advanced schistosomiasis. The boy was referred to the Research Institute for Tropical Medicine (RITM), Manila, due to a rapidly enlarging spleen post-praziquantel treatment. At RITM, liver function tests were within normal limits but complete blood examinations showed pancytopenia and abnormal coagulation times. Serum markers for hepatitis A, B and C were negative. Abdominal MRI revealed schistosome-induced periportal fibrosis. The main portal vein appeared thrombosed with characteristic cavernous transformation of the right portal vein. Varices were seen in the oesophagus, gastrohepatic ligament, and splenic hilum. The spleen was markedly enlarged, with parenchymal foci representing Gamna-Gandy bodies. The patient underwent splenectomy. Histopathologic findings in the liver showed moderate pipestem fibrosis and schistosome egg granulomas. The patient was discharged from the hospital in excellent clinical condition.

BACKGROUND

Schistosomiasis affects approximately 240 million individuals globally causing roughly 70 million disability-adjusted life years.^{1 2} Most of the deaths are caused by liver disease induced by the *Schistosoma* parasite.³ In schistosomiasis, adult worms reside in the mesenteric vasculature where they lay eggs.⁴ Although these ova are intended to traverse into the bowel lumen to complete the parasite life cycle, many of them are carried upstream to the portal vein where they get trapped in the presinusoidal venules. In this site, the eggs ignite a granulomatous inflammatory response which results in the deposition of fibrotic macromolecules around the portal tract causing obstruction to blood flow. Blockage in the portal blood flow results in the development of portal hypertension, collateral blood vessels and splenomegaly.⁵ Splenic enlargement can also be caused by massive lymphocyte hyperplasia induced by chronic inflammation.⁶

The three main species of *Schistosoma*, namely *Schistosoma mansoni*, *S. japonicum* and *S. mekongi* cause a significant hepatosplenic form of schistosomiasis.³ *Schistosoma mansoni* is endemic in tropical and subtropical regions of Africa, South America and the Caribbean, *S. japonicum* in China,

the Philippines and Indonesia, and *S. mekongi* along the Mekong river tributaries.³ Among these three species, *S. japonicum* is remarkable in terms of the egg output per day and the egg laying pattern by female worms. Many more eggs are laid per day by *S. japonicum* compared with other species.^{7 8} These eggs are also deposited in aggregates in the target end organs. The sheer number of eggs and the pattern of deposition by *S. japonicum* are believed to be responsible for the more severe target organ morbidities compared with the other intestinal forms.⁹

In the Philippines, schistosomiasis affects 28 provinces along the south eastern part of the country with recent endemic foci reported in the northernmost and central areas.^{10 11} Currently, there are approximately 865 000 individuals infected and an additional seven million at risk of infection.¹² Significant morbidity and mortality due to granuloma formation in the liver, resulting in hepatic fibrosis and complicated cases of portal hypertension have been documented.^{13–16} We report the successful clinical management of a case of advanced schistosomiasis in a 12-year-old boy from the province of Northern Samar, The Philippines.

CASE PRESENTATION

A 12-year-old Filipino boy, residing in a well-known schistosomiasis-endemic area, presented in 2013 with gross abdominal enlargement, hepatosplenomegaly, diarrhoea and abdominal pain. A Kato-Katz thick smear stool examination was performed and a diagnosis of schistosomiasis was made. The patient was subsequently treated with a split dose, 60 mg/kg, of praziquantel (PZQ) on day 1 and day 14.

Five years prior to admission, the patient reported a 1-month history of febrile illness with bloody diarrhoea occurring 5–6 times/day. He was brought to the local health centre where he was given empirical treatment for the bloody diarrhoea and the condition resolved. One month after the episode of diarrhoea subsided, the mother noted that the child's abdomen started to slowly enlarge. His daily activities remained unaffected despite his enlarging abdomen.

INVESTIGATIONS

The boy was referred to the Research Institute for Tropical Medicine, Manila, for follow-up

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assessment and care due to his rapidly enlarging spleen and deteriorating health status post-PZQ treatment. On physical examination his heart rate was 89 bpm, blood pressure 90/60 mm Hg, respiratory rate 20 cycles/min and core body temperature 36.6°C. His height was 92 cm, weight 25.5 kg and abdominal circumference 73 cm. The palpebral conjunctivae were pale but sclerae were non-icteric. Both the heart and lung fields were unremarkable on auscultation. On abdominal examination, gross abdominal enlargement was noted along with a palpable liver, and splenomegaly (Hackett's graduation ≥ 5). No tenderness was evident.

Haematological testing showed leukopenia (white cell count $2.9 \times 10^9/L$), anaemia (haemoglobin 8.6 g/dL) and mild thrombocytopenia (platelet count $136 \times 10^9/L$). Serum electrolytes were within normal limits. Serum glutamic oxaloacetic transaminase (SGOT) was low (19.83 U/L), but serum glutamic pyruvic transaminase and alkaline phosphatase were within the normal range. Total protein and albumin-to-globulin ratio were normal. Direct, indirect and total bilirubin values were, likewise, within normal limits. Blood urea nitrogen and creatinine were also normal. However, the clotting, prothrombin (PT) and partial thromboplastin times were prolonged. Results of urinalysis were unremarkable. Stool examination, using the Kato-Katz thick smear technique, was positive for *Schistosoma* (10 eggs/g of stool), *Ascaris* and *Trichuris* ova. The patient's serum was screened for hepatitis A, B and C (anti-HAV IgM, HbcAb, HbeAg, HbsAg, and anti-HCV) but all were found to be negative. Ultrasound (US; portable model SONACE) examination of the abdomen showed a markedly enlarged spleen. The lower border of the spleen extended down to the level of the umbilicus. The liver was mildly enlarged in the area of the left lobe. The main branches of the portal vein were thickened and connected to the prominent fibrotic bands which partially divided the liver into lobules (figure 1). No ascites was demonstrated. A solitary stone was noted inside the gallbladder. Liver parenchymal fibrosis was classified as grade II using WHO ultrasonography guidelines.¹⁷

MRI of the abdomen with gadolinium showed a mildly enlarged left lobe of the liver (figure 2). The liver margins were lobulated. No focal liver lesion was seen. There were curvilinear tracts scattered throughout the liver parenchyma, consistent with portal fibrosis. The main portal vein was markedly attenuated and poorly visualised and thrombosed. There were several small tortuous vessels in the region of the right portal vein, secondary to collaterals/cavernous transformation. The spleen was severely enlarged extending to the left hemipelvis. There were multiple punctate low-signal foci scattered throughout the spleen, likely representing Gamna-Gandy bodies (figure 2). There were a few larger low-signal foci identified, measuring up to 2.2 cm, which may have been secondary to calcifications or hemosiderin deposits. The splenic vein measured approximately 8 mm in diameter. There was an 8-mm-dependent filling defect at the gallbladder neck, consistent with a calculus. Varices were identified within the distal oesophagus, gastrohepatic ligament and splenic hilum. A trace amount of perisplenic ascites was present. In sum, the findings were consistent with hepatosplenic schistosomiasis, with mild enlargement of the left hepatic lobe and diffuse scattered areas of portal fibrosis throughout the hepatic parenchyma. Portal hypertension was seen, with portal vein thrombosis (PVT) and cavernous transformation of the portal vein (CTPV). Massive splenomegaly and varices within the distal oesophagus and gastrohepatic ligament were noted.

Figure 1 (A) Ultrasound image of the liver showing the moderately thickened branch of the main portal vein (white arrow). The liver parenchyma appeared normal. (B) Ultrasound picture of the spleen which shows the markedly enlarged organ with dilated splenic vein (black arrow).

DIFFERENTIAL DIAGNOSIS

Superimposed chronic viral hepatitis was suggested but was ruled out by negative hepatitis serum screening. Massive splenomegaly in schistosomiasis can also be caused by hyperplasia induced by phagocytosis of disintegrated worms, ova and toxins. However, this situation is expected to be seen only in patients with heavy schistosome infections whereas the intensity of infection in this patient was light. PVT was the third condition considered.

TREATMENT

With the findings of severe portal hypertension and pancytopenia due to hypersplenism, the patient was transferred to the University of the East Ramon Magsaysay Memorial Medical Centre for surgery. After correction of anaemia, clotting, bleeding and PT time abnormalities, the patient was operated on. Figure 3 shows the picture of the abdomen of the patient before and after the operation with marker lines indicating the borders of the enlarged spleen. Intraoperative findings showed the markedly enlarged spleen and a normal appearing liver (figure 3). A splenectomy was performed and a wedge liver biopsy was taken. The gallbladder was removed along with the calculus.

Figure 2 MRI of the liver and spleen: (A) the black arrow shows cavernous transformation around the right portal vein (CTPV), and the white arrow shows periportal fibrosis running along the secondary branch of the portal vein. (B) The white arrows show the curvilinear periportal fibrosis. (C) The white arrow shows hyperintense splenic vein. (D) White arrow showing hypointense portal vein (thrombosed). (E) White arrow showing hyperintense right portal vein with CTPV. (F) The white arrow showing oesophageal varices. (G) The white arrow showing gastric varices. (H) The black arrows showing Gamma Gandy bodies.

Histopathological findings of the liver showed ‘pipestem’ fibrosis with moderate portal inflammation and parasitic granulomas consisting of macrophages, epithelioid cells, giant cells, lymphocytes, eosinophils, plasma cells, fibroblasts and calcified *Schistosoma* ova aggregates. In the spleen, there was severe congestion noted, and some foci of granuloma formation with schistosome eggs (figure 4).

OUTCOME AND FOLLOW-UP

Two weeks after the operation, the patient was discharged in excellent clinical condition and his future looks bright.

DISCUSSION

Enlargement of the spleen in schistosomiasis japonica is usually due to portal hypertension. The degree of splenic enlargement reflects the degree of hepatic fibrosis and consequently the severity of portal hypertension. When this patient was examined by US there were findings of periportal and grade II liver parenchymal fibrosis. The spleen was also markedly enlarged. There was no ascites. No history of bleeding from the upper gastrointestinal tract was noted. The patient was subsequently treated with a split dose (60 mg/kg in total) of PZQ over a 2-week period. Three months later, the patient underwent a second US. No change in the grade of hepatic fibrosis was seen, but the spleen was noted to be much larger than before and there was

marked deterioration in the general health of the patient. At that point, other conditions that may have been causing the progressive enlargement of the spleen, despite treatment, were considered. Superimposed chronic viral hepatitis was suggested but was ruled out by negative hepatitis serum screening. Histological findings showed pipestem fibrosis with moderate portal inflammation, parasitic granuloma and calcified *Schistosoma* ova. Massive splenomegaly in schistosomiasis can also be caused by hyperplasia induced by phagocytosis of disintegrated worms, ova and toxins. However, this situation is expected to be seen only in patients with heavy schistosome infections whereas the intensity of infection in this patient was light.

PVT was the third condition considered. Pathogenesis of patients with PVT with portal hypertension can be related to unbalanced haemostasis and slowing of portal flow. PVT is a well-known complication following surgical intervention (ie, splenectomy and distal splenorenal shunting) for schistosomiasis and possibly arises due to the fall in portal vein pressure after surgery.^{17 18} This complication had also been reported in a 46-year-old patient with portal hypertension due to *S. mansoni* without previous surgery for complications of portal hypertension.¹⁹ Despite treatment with PZQ, the patient had to undergo repeated sessions of sclerotherapy for recurrent bleeding from ruptured oesophageal varices. MRI of this patient showed PVT.

Figure 3 (A) and (B) show the abdomen of the patient with the dotted lines delineating the borders of the liver and spleen before operation, and the border of the liver after operative removal of the spleen. (C) shows the intra-operative appearance of the liver and spleen. The liver was normal looking while the spleen was markedly enlarged. (D) shows the gross appearance of the spleen, post-splenectomy.

The patient underwent surgical procedure consisting of splenectomy, oesophagogastric disconnection and suturing of the varices. The patient was discharged from hospital in good clinical condition. The clinical course of our young patient suggests the development of PVT as a direct result of portal hypertension. Although thrombus inside the portal vein was not demonstrated, as in the case due to *S. mansoni* aforementioned, there were three MRI findings that pointed to the occurrence of PVT

in this patient. First, the main portal vein was markedly attenuated and poorly visualised, and thus thrombosed. Second, there were small tortuous vessels in the right portal vein region which suggested the presence of CTPV. CTPV is a sequela of PVT.²⁰ CTPV occurs in patients with PVT due to the obstruction to the flow of portal blood leading to the development and dilation of multiple small vessels in and around the recanalising main portal vein. The cause of CTPV is not known, but it can occur in healthy livers with chronic non-cirrhotic and non-tumoural PVT.^{21–22} Third, small siderotic nodules of the spleen, called γ -Gandy bodies, have been seen on MRI in patients with portal vein or splenic vein thrombosis.²³ Although the same lesion has been reported for haemolytic anaemia, leukaemia, acquired haemochromatosis and paroxysmal nocturnal haemoglobinuria, this patient had no history of any form of blood dyscrasia.²³ To the best of our knowledge, this is the first case of PVT-induced splenomegaly due to advanced schistosomiasis reported for the Philippines.

The major complications of portal hypertension in schistosomiasis are massive bleeding due to ruptured oesophageal varices and pancytopenia due to hypersplenism. Bleeding from oesophageal or gastric varices typically results in a high death rate. For bleeding oesophageal or gastric varices, endoscopy-guided injection sclerotherapy or band ligation of the bleeding varices are the most appropriate immediate treatment procedures.²⁴ However, the facilities and expertise for these procedures are not always available in many developing countries where schistosomiasis is endemic. In such a situation, vasoactive drugs that can lower the splanchnic vascular pressure can be used as a stopgap measure. Among the vasoactive drugs, propranolol, somatostatin and octreotide have been used in the control of bleeding varices in patients with portal hypertension due to schistosomiasis.^{25–29} One study has shown propranolol to be more effective if combined with endoscopic treatment in controlling variceal bleeding. It has been shown that propranolol in combination with endoscopic variceal sclerotherapy can prolong the period between the initial variceal injection and the recurrence of bleeding.³⁰ Somatostatin and octreotide, on the other hand, were demonstrated in clinical trials, to be at least as effective as other vasoactive drugs, but with the advantage of fewer adverse effects, making these drugs more ideal as adjuvant treatment to emergency sclerotherapy or band ligation of varices with active bleeding.³¹ The benefit in terms of prolongation of the period for rebleeding by an endoscopic procedure is best seen in patients with previous splenectomy compared with those without previous surgery.³²

In the case of oesophageal variceal bleeding not responding to endoscopic and medical treatment, particularly secondary prevention of bleeding from gastric varices, transjugular intrahepatic portosystemic shunt (TIPS) is indicated.³³ TIPS is a radiological procedure which involves connection of the portal and hepatic veins using a stent. The purpose of the TIPS is to decompress the portal venous system and prevent rebleeding. The procedure is also indicated in patients requiring repeated and frequent paracentesis.^{33–34} However, there are potential complications with the procedure such as occurrence of hepatic encephalopathy, deterioration of liver function and stent-related problems.^{33–35} For those cases with recurrent variceal bleeding that do not respond to intravenous infusion of drugs and to less invasive non-surgical procedures, surgery is indicated. A long-term follow-up of a randomised trial, comparing three types of surgery for complications of portal hypertension due to *S. mansoni*, has demonstrated that the best results are achieved with distal splenorenal shunting, and with oesophagogastric

Figure 4 (A–F) The histopathological findings of the patient's liver and spleen. (A) Early granuloma formation around *Schistosoma japonicum* eggs (H&E $\times 40$ magnification). (B) A matured granuloma (H&E $\times 10$). (C) A late granuloma in which most of the cells around the egg were replaced by fibroblasts and fibrotic materials (reticulum stain $\times 40$). (D) A much later granuloma in which the egg was already destroyed (reticulum stain $\times 40$). (E and F) Eggs trapped in the spleen with only minimal cellular reactions around the eggs (H&E $\times 10$ and $\times 40$, respectively).

devascularisation plus splenectomy.³⁶ In patients with markedly enlarged spleens with pancytopenia due to hypersplenism but with no history of bleeding from oesophageal or gastric varices and no ascites, as in our patient, splenectomy was deemed appropriate and potentially lifesaving.

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Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Schistosomiasis is a neglected tropical disease that can cause significant morbidity in about 5% of diagnosed cases.
- ▶ Enlargement of the spleen in schistosomiasis japonica is usually due to portal hypertension as a result of hepatic fibrosis.
- ▶ The major complications of portal hypertension in schistosomiasis are massive bleeding due to ruptured oesophageal varices and pancytopenia due to hypersplenism.
- ▶ In patients with markedly enlarged spleens with pancytopenia due to hypersplenism but with no history of bleeding from oesophageal or gastric varices and no ascites, as in our patient, splenectomy was deemed appropriate and potentially lifesaving.

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