Case Report

Renal allograft transplant recipient with ruptured hydatid native kidney

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Abstract

Echinococcosis of the kidneys in a renal transplant recipient is extremely rare and its occurrence being related to immunosuppression is a possibility which needs further characterisation. Ruptured renal hydatid in a renal transplant recipient is not reported so far to our best knowledge. We present a 42-year-old renal allograft receipient who presented one year after transplant with left flank pain, palpable left lumbar mass and gross hydatiduria. Investigations revealed a ruptured native hydatid kidney. Patient was managed with a combination of chemotherapy and left native nephrectomy and discharged in a satisfactory condition.

Key Words: Hydatid cyst, hydatiduria, renal transplant

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INTRODUCTION

Parasitic infections, although important complications of organ transplantation that are often overlooked in the differential diagnosis of post-transplantation pyrexial illnesses, are much less prevalent than bacterial and viral infections. Hydatid disease is a rare parasitic disease mainly involving the liver and lungs. Renal echinococcosis comprises about 2% of all hydatid disease in man.[1] We hereby report a case of ruptured native hydatid kidney in a renal transplant receipient, which could have been encouraged by immunosuppressive therapy. To our knowledge this is the first case of ruptured native hydatid kidney in a renal transplant recipient.

CASE REPORT

A 42-year male, non-diabetic, known case of hypertension, end-stage renal disease (ESRD), had a renal transplantation done in 2010 with mother as donor. The cause of ESRD was not established after detailed investigations. Patient was on post-transplant immunosuppressive therapy in the form of mycofenolate mofetil and tacrolimus. He presented to our out-patient department with history of left flank pain. Clinical examination was unremarkable except that his abdomen showed left flank fullness and palpable non-tender ballottable mass. Ultrasound (USG) abdomen revealed a septate calcified cystic lesion almost involving whole native kidney measuring 11 × 10 cm suggestive of hydatid kidney. Transplanted right kidney was normal. IgG ELISA for hydatid serology was suggestive with titers of 1:320. Contrast enhanced CT abdomen showed a well-calcified walled lesion with multiple internal septae suggestive of hydatid disease kidney. Patient was put on albendazole and subsequently managed with puncture, aspiration, injection, and re-aspiration technique (PAIR) to which patient did not respond. Patient was advised nephrectomy which he refused. Patient was continued on albendazole and 3 years later patient presented with colicky abdominal pain and passage of whitish membranes in urine. CT abdomen was advised which confirmed the previous findings of hydatid disease kidney with change in size [Figure 1]. USG revealed daughter cysts and membranes in the urinary bladder [Figure 2]. Gross urine examination showed cysts and

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membranes of echinococcus granulosus [Figure 3]. Patient was successfully managed with left nephrectomy and was discharged on albendazole. The follow-up improvement was noticed by symptomatic improvement and decreasing titers on IgG ELISA.

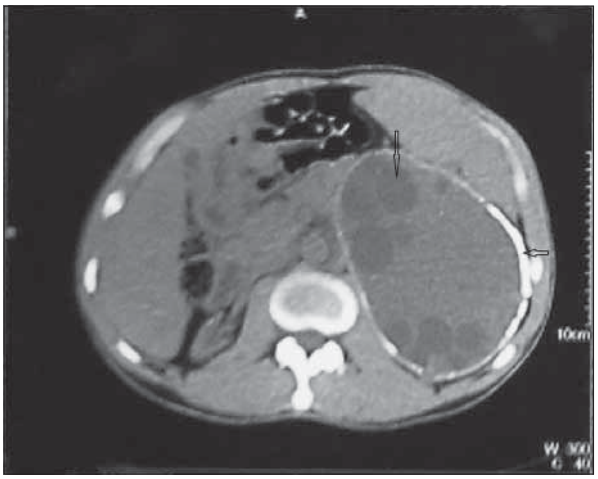

Figure 1: CECT abdomen showing hydatid cyst in native kidney with walled calcifications

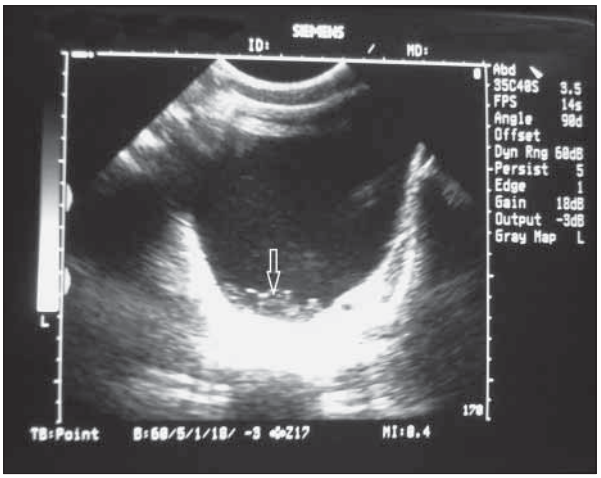

Figure 2: Ultrasonography abdomen showing ruptured daughter cysts and membranes within the urinary bladder

Figure 3: Gross urine sample collected in a bottle showing daughter cysts and membranes in naked eye examination

DISCUSSION

About 5% of human pathogenic parasites have been reported to cause significant illness in transplant recipients and approximately 10 different parasitic infections have been reported to infect renal transplant receipient.[2] Echinococcus infection in a renal transplant receipient is extremely rare and hydatid disease of the kidney in a renal transplant recipient is a new infection and has not been reported in literature except in one case report.[3]

Hydatid disease may be asymptomatic or may present as lumbar pain and abdominal mass. Cystic rupture into the collecting system is uncommon and usually presents as microscopic hydatiduria. Gross hydatiduria is rare.[4] It is not clear how the hydatid embryo reaches the kidney in cases of primary hydatid disease but it is postulated that it must pass through the portal system into the liver and retroperitoneal lymphatics.[3]

In our patient, no evidence of hydatid disease was found on pre-op evaluation. The occurrence of hydatid disease could have been because of immunosuppression could only be speculated. A decrease in antibody production has been reported with mycophenolate mofetil.[5] Also there is a role of cellular immunity in controlling earlier stages of larval development.[6] Hildreth *et al.* showed that cortisone treatment drastically increased both the number of Echinococcus cysts in mice and the average size of each cyst when treatment was administered at an early stage.[7] Immunosuppressant drugs may allow proliferation of metacestode remnants or proliferation of previous inapparent metastases.[8] However some reports have shown no effect of immunosuppression on the development and progression of hydatid disease.[9,10] Studies are warranted to see the effects of immunosuppression on the development of this parasitic infection in transplant patients.

Our patient did not respond to PAIR and was finally managed with nephrectomy which is required in most of the cases.

In conclusion, we report the first case of a well-defined ruptured native hydatid kidney in a renal transplant patient which was successfully managed by nephrectomy. Although indirect evidence suggests the role of immunosuppression in encouraging the development of hydatid disease, direct evidence is lacking which needs further characterisation.

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