# A CASE OF HYDATID CYST DISEASE IN A RENAL TRANSPLANT RECEPIENT

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## INTRODUCTION

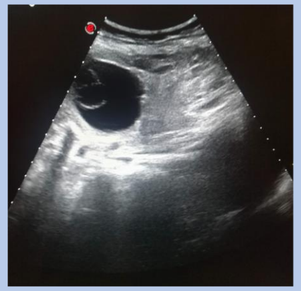
Hydatid cyst disease is an orally transmitted parasitosis caused by the larval form of the Echinococcus granulosus tapeworm that penetrates the intestinal mucosa and reaches the internal organs via the blood and lymphatic stream.

Cystic hydatid disease usually affects the liver (50 -70%), less frequently the lungs, the spleen, the kidneys, the bones and the brain(1-3%).

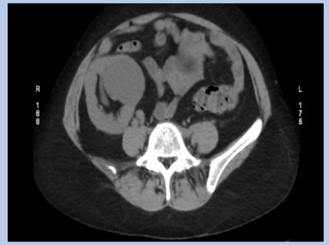
## CLINICAL PRESENTATION

A 48 year old lady who presented to our institute 16 years back with breathlessness and anuria- on examination she had bipedal edema and bilateral basal crackles ,her investigations revealed serum creatinine of 15.7 mg /dl ,hemoglobin of 4.2 g/dl and on further evaluation she was found to have ESRD. She was initiated on maintanence hemodialysis. She underwent live related renal transplantation in 1996 , her mother being the donor. She received prednisolone, azathioprine and cyclosporine. No induction therapy was given. Serum creatinine had normalized to 1.0 mg/dL on day 3. She has been on regular follow up with no major infections or immunological events . After 24 years, patient complained dull dragging pain on the right iliac fossa with no associated fever or dysuria. Ultrasound abdomen showed presence of transplant kidney in the right inguinal region, measuring 13 x 5.2 cm. There was a circumscribed round anechoic lesion noted in the upper pole of the transplant kidney. A few smaller cystic areas noted attached to the wall of this anechoic lesion. Thin echogenic septations also seen. Further evaluation with non contrast CT abdomen showed round homogenously hypodense lesion involving the cortex of the transplant kidney causing the contour bulge on the surface with no evidence of any peripheral or internal calcification, suggestive of hydatid disease in the renal allograft . Other investigations were, serum creatinine: 1.3 mg/dL, blood urea: 18 mg/dL, haemoglobin: 12.5 g/dL, total leucocyte count: 7200 /mm$^3$, blood and urine cultures were sterile.She was adviced surgical management but she opted for conservative treatment . We prescribed tablet albendazole 400mg twice daily for 28 days. The same was repeated for another cycle. As there was no reduction in the size of the hydatid cyst, we forestalled further doses.

## IMAGING



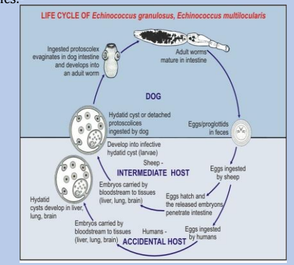
Ultrasound abdomen showed the long axis of the transplant kidney. There is a circumscribed round anechoic lesion noted in the upper pole of the transplant kidney with posterior acoustic enhancement. A few smaller cystic areas noted attached to the wall of this anechoic lesion. Thin echogenic septations also seen.



Non contrast CT scan axial image at the level of pelvis showing transplant kidney in the right iliac fossa. There is evidence of round homogenously hypodense lesion involving the cortex of the transplant kidney causing the contour bulge on the surface. There is no evidence of any peripheral or internal calcification. No evidence of extension of lesion beyond the confines of the transplant kidney

## DISCUSSION

Echinococcus granulosus (dog tapeworm) are of two types - Echinococcus granulosus and Echinococcus multilocularis. The eggs of Echinococcus granulosus ingested with contaminated food and water hatch in the duodenum releases hexacanth embryo which penetrates the intestinal layers, through portal circulation reaches the liver and form hydatid cyst. 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.[2]




We don't know if the immunosuppressive treatment encouraged the development of the hydatid cyst, as cellular immunity has a significant role in controlling earlier stages of larval development.[3] 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.[4]

In a patient with known liver hydatidosis who underwent heart transplantation ,the immunosuppressive treatment seemed to have no effect on the growth of the cysts, which were removed surgically 14 months later.[5] Antihelminthic properties of cyclosporine, perhaps via cyclophilin, have been reported in vitro by Colebrook et al. Administration two days prior to infestation, resulted in significant reduction in cyst establishment in mice, but the drug had no effect when administered 18 weeks after Echinococcus infestation. Liver transplantation, in unresectable cases are encouraging, with a 5 year survival rate of 76.2% and lack of recurrence of disease despite the immunosuppressive regimen.

Pre- and post-surgical treatment with albendazole or mebendazole has the advantage of reducing the risk of disease recurrence and intraperitoneal seeding of infection with spontaneous cyst rupture and spillage during surgery or needle drainage . Our present patient is the first patient of hydatid disease in the renal allograft. There are reports of hepatic hydatid disease and native kidney hydatid disease in kidney transplanted patients

## PUBLISHED REPORTS OF HYDATID DISEASE IN RENAL ALLOGRAFT RECIPIENTS

| S no | Age/Sex | Presentation | Site of hydatid disease | Serum creatinine (mg/dL) | Management |
|---|---|---|---|---|---|
| 1 | 44/M | 5 months post transplantation , 3 day history of fever with shivering,dysuria | Left hepatic lobe | 2.75 | Surgical removal of the hydatid cyst |
| 2 | 43/M | Detected in annual checkup | A magnetic resonance imaging revealed a heterogeneous, complex cystic mass on the right native kidney | Not reported | Surgery followed by albendazole 600 mg per day for six months. |
| 3 | 42/M | Left flank pain, left flank fullness and palpable non-tender ballottablemass | Ultrasound abdomen revealed a septate calcified cystic lesion almost involving whole native kidney measuring11 × 10 cm suggestive of hydatid kidney. | Not reported | Initial management with albendazole and puncture,aspiration, injection, and re-aspiration technique. As there was no response nephrectomy was aadvised 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. CTabdomen confirmed the previous findings of hydatid disease kidney with change in size. Left nephrectomy was done. |

## CONCLUSION

***Our present patient is the first patient of hydatid disease in the renal allograft. There are reports of hepatic hydatid disease and native kidney hydatid disease in kidney transplant patients . Although indirect evidence suggests the role of immunosuppression in encouraging the development of hydatid disease, direct evidence is lacking which needs further characterization. Surgery remains the first choice of treatment, with the potential for complete removal of the parasite. Pre- and post-surgical treatment with albendazole or mebendazole has the advantage of reducing the risk of disease recurrence and intraperitoneal seeding of infection with spontaneous cyst rupture and spillage during surgery or needle drainage***

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