



Case Report

Isolated renal mucormycosis in an apparently healthy immune competent adult

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ABSTRACT

Mucormycosis usually presents as an opportunistic infection in diabetics and immunocompromised individuals. Though involvement of the kidneys has been reported in up to 20% of disseminated disease, there are only few case reports of isolated renal mucormycosis in the literature. We report a case of isolated renal mucormycosis (IRM) in a healthy adult with no antecedent risk factors.

KEYWORDS: Renal mucormycosis, Adult, Immunocompetent.

INTRODUCTION

Zygomycosis is now being reported with increased frequency as a cause of infection in immunocompetent healthy individuals. The clinical presentation of this fungus is most commonly rhino-cerebral, followed by primary pulmonary, disseminated, gastrointestinal and cutaneous forms [1, 2]. Renal involvement usually occurs in disseminated form, isolated renal involvement in immunocompetent host and requires high index of suspicion for diagnosis and prompt treatment with nephrectomy and Amphotericin should be considered for favourable outcome.

CASE REPORT

A 35 years old male presented with a history of persistent left flank pain with hematuria for 10 days. He had fever at the onset which subsided after 2 days following intravenous antibiotic therapy for suspected pyelonephritis based on presence of leucocytosis, pyuria and enlarged left kidney on ultrasound at another medical centre. Urine cultures were sterile there. The patient did not have diabetes mellitus, other co-morbidities, or history of any drug or alcohol abuse.

On admission at our center, he was afebrile, heart rate was 120 per minute and blood pressure was normal. He had left flank tenderness (pain score of 6/10 on Wong Baker scale) and no organomegaly or ascites. Investigations showed a

haemoglobin of 14.1gm/dl, total leucocyte count of 17890 cell/cmm (neutrophil 86%, lymphocytes 9%, eosinophils 2%, monocytes 2%, myelocytes 1%), ESR 42 mm/hr. Serum creatinine was 1.5 mg/dl, blood urea nitrogen 23.8 mg/dL, serum bilirubin 3.5 mg/dl (direct 2.2 mg/dl), serum aspartate transaminase 43 IU/L, alanine transaminase 104 IU/L, alkaline phosphatase 244 IU/L and fasting blood glucose 89 mg/dl. HIV ELISA, HBsAg and HCV Ab were negative. Urinalysis showed a pH of 6.4 and plenty of white blood cells and red blood cells/high power field. Urine and blood bacterial cultures at admission were subsequently sterile.

Non contrast computed tomography (CT) showed an enlarged left kidney suggestive of left pyelonephritis with no hydro-uretero-nephrosis. Contrast enhanced CT showed enlarged left kidney with a large non-enhancing hypodense area suspicious of infarct or abscess and inflamed adjacent structures (Fig 1), with evidence of left renal vein thrombosis. The patient showed no improvement with IV cefoperazone –sulbactam. Renal vein thrombosis secondary to worsening pyelonephritis or vascular pathology was considered. He was started on anticoagulation for a possible thrombophilic disorder.

Cystoscopy showed no evidence of any obstruction, normal bladder and proximal urinary system. Tc-99m dimercaptosuccinic acid (DMSA) renal scan showed enlarged left kidney with reduced and patchy cortical tracer uptake and multiple cold areas, with a normal right kidney.

The patient continued to be toxic, developed diffuse swelling of the left flank with excruciating pain requiring high doses of analgesics and sedation. He was therefore taken up for nephrectomy of the non functional kidney: the left kidney showed patchy areas of infarction with no abscess and the entire left retro-peritoneum was inflamed. Post operatively, he seemed better for a day when his pain subsided and renal functions normalized (serum creatinine 1.2 mg/dl). He continued to have leukocytosis (33,300 cell/cmm) and hence antibiotics were continued.

On 2nd postoperative day he developed respiratory acidosis, bradycardia and cardiac arrest and was resuscitated. His oxygen requirements continued to rise. CT chest with contrast showed pulmonary embolism despite patient being on anticoagulants. He developed ascites and anuria requiring continuous renal replacement therapy.

On 3rd postoperative day histopathology report on left kidney was received, showing renal tissue with extensive necrosis and suppuration with interstitial inflammation by lymphocytes, plasma cells and neutrophils. The necrotic area showed multiple pauci-septate, broad, ribbon like fungal hyphae with right angle branching consistent with mucormycosis (Fig 2, 3). Vascular invasion was also noted. Fungal culture of the removed kidney showed growth of grey-white downy colonies on Sabouraud's dextrose agar consistent with *Mucor* species. Bacterial cultures were negative.

A diagnosis of isolated left renal mucormycosis was made. He was started on conventional Amphotericin B at a dose of 1 mg/kg/d on the third postoperative day. However he showed poor response to treatment, developed disseminated intravascular coagulation and expired.

Fig1: Contrast CT KUB showing bulky Left kidney with large hypodense areas and patchy enhancement

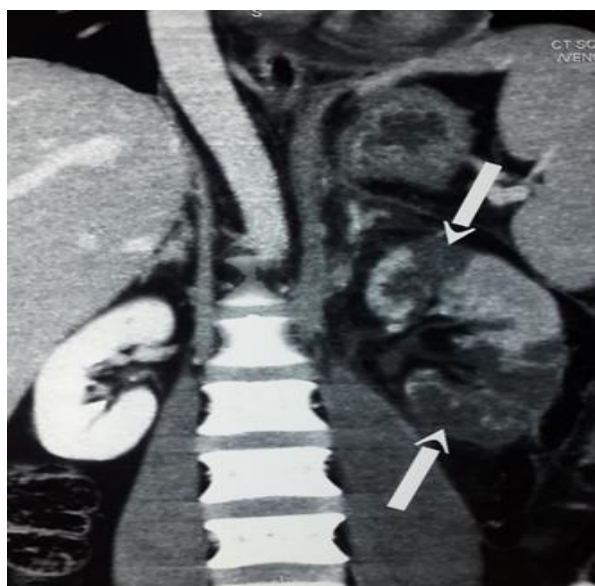


Fig 2: Section showing pauci-septate broad ribbon like fungal hyphae (Periodic acid Schiff stain; x40)

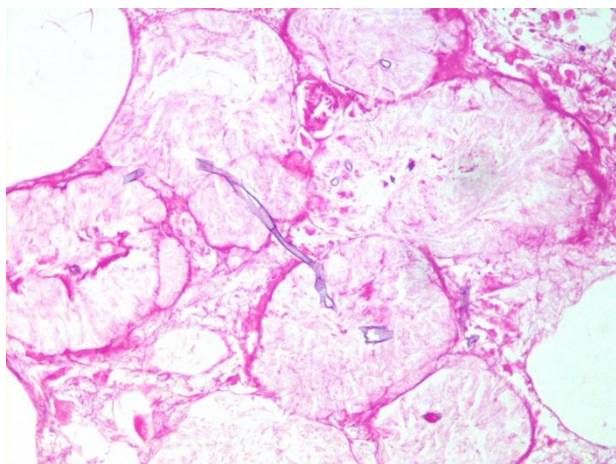
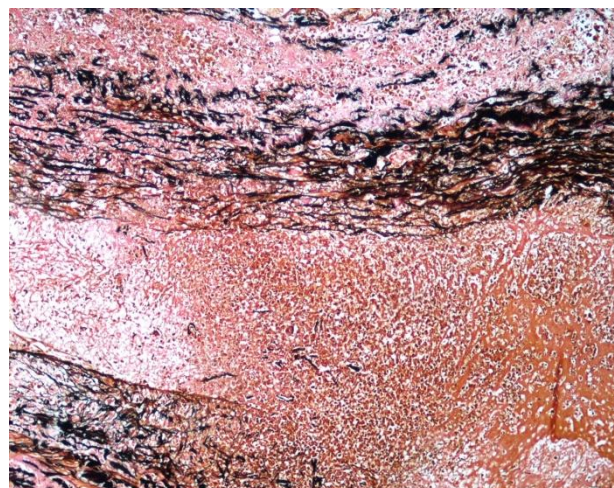


Fig 3: Section showing multiple broad ribbon like fungal hyphae, (Gomori Methenamine-Silver stain; x10)



DISCUSSION

Mucormycosis is an invasive infection caused by fungi of the class Zygomycetes, order Mucorales viz. *mucor*, *rhizomucor*, *absidia*, *apophysomyces* etc. Invasive mucormycosis is largely restricted to those with underlying immunocompromising illness, most commonly diabetes, lymphoproliferative disorders, and transplant recipients. Our patient did not have any of these recognized risk factors.

Isolated renal mucormycosis (IRM) has rarely been reported in healthy adults with minimal constitutional symptoms, especially from India [3-5]. Most patients with IRM have presented with fever (88%), flank pain (70%), tenderness and gross hematuria (70%) [6]; our patient had a similar presentation. Acute renal failure is due to direct hyphal invasion of renal vessels causing near total or total occlusion of arteries and veins and subsequent thrombosis with cortical and medullary infarction. Blood and urine cultures are usually negative and therefore diagnosis almost always requires histopathologic evidence of fungal invasion of the tissues. If the CT scan reveals a “diffuse patchy nephrogram” or an enlarged kidney with poor enhancement and focal or diffuse enhancement, biopsy should be considered.

The pathogenesis of IRM is not clear. Haematogenous dissemination to kidneys and a lower urinary tract infection with retrograde spread have been suggested [7]. Histologically there is extensive angio-invasion with resultant vessel thrombosis and tissue necrosis. This angio-invasion is associated with the penetration through endothelial lining of blood vessels and haematogenous dissemination of fungus from the original site of infection to other organs which could explain the pulmonary embolism and the rapid progression in our patient.

Successful therapy of IRM requires extensive debridement of infected and necrotic tissue, administration of Amphotericin B, and reversal of underlying immune compromising conditions if any [7]. There are isolated reports of successful management of IRM with Amphotericin B alone, but a combined approach of Amphotericin B with nephrectomy seems to offer the best chance for cure [8]. Our patient expired despite this approach, probably because of delay in diagnosis and rapid dissemination. Overall survival is estimated to be 65% [3].

CONCLUSION

Isolated renal mucormycosis may affect healthy immune competent hosts and presents with neutrophilic leucocytosis,

sterile pyuria, hematuria and suggestive radiographic findings (evidence of severe localized pyelonephritis, vascular thrombus). A high index of suspicion for the entity and prompt treatment with nephrectomy and amphotericin is required in view of the poor prognosis associated with delayed diagnosis and therapy.

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