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Case Report

Cortical laminar necrosis due to refractory status epilepticus in a kidney transplant patient with cryptococcal meningitis

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ABSTRACT

A 56-year-old diabetic male underwent kidney transplant in 2010. He was brought to the hospital with complaints of vomiting and altered sensorium of 10 days duration. Lumbar puncture revealed cryptococcal meningitis. He was promptly initiated on liposomal amphotericin B and flucytosine. Immunosuppressive agents tacrolimus and mycophenolate mofetil were discontinued. There was an initial improvement in his sensorium but a few days later, he developed super-refractory status epilepticus. Despite early and aggressive management of seizures with multiple anti-epileptic drugs including intravenous anesthesia with thiopentone, his seizures persisted. This unfortunately led to cortical laminar necrosis, a condition in which there is severe brain injury. This condition is commonly a consequence of hypoxic ischemic encephalopathy, hypoglycaemic encephalopathy, status epilepticus, or ischemic stroke, and most patients either progress to brain death or remain in a persistent vegetative state.

1. Introduction

Cryptococcus neoformans is an invasive fungus in organ transplant recipients, with reported incidence of cryptococcosis being 3–4%[1] in renal transplant recipients. It is the pathogen most frequently implicated in infections of the central nervous system (CNS) during the late post-transplantation period, and often the cause of subacute meningitis-encephalitis. Mortality rate is around 50% despite antifungal treatment.[1]

We report a patient who presented with the full spectrum of disseminated cryptococcal disease involving the CNS, lung, and skin, four years after successful kidney transplant. We were able to identify the pathogen from all three sites of involvement.

The main purpose of this case report is to highlight cortical laminar necrosis (CLN) as a rare neurologic complication

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secondary to refractory status epilepticus (RSE) in a case of cryptococcal meningitis (CM). We could find only 2 reported cases of CLN related to prolonged status epilepticus in the literature, and to our knowledge this is the first case in a renal transplant patient.

2. Case history

A 56-year-old male was brought to the hospital with complaints of vomiting and altered sensorium.

He was a known case of diabetes mellitus, hypertension, peripheral vascular disease, and ischemic heart disease for which he underwent CABG. In 2010, he developed ESRD for which he underwent living unrelated donor renal transplantation at another center. He was on triple immunosuppression with tacrolimus, mycophenolate mofetil, and prednisolone. Allograft function was stable.

On admission, he was conscious but drowsy. He was afebrile and hemodynamically stable. His skin showed multiple umbilicated papules on the nose, cheek, and dorsum of fingers (Fig. 1). Neurologically, there was no focal neurologic deficit with a Glasgow coma scale of 13/15.

Laboratory values on admission showed 1 + protein, 5–6 RBC/hpf, and 4–5 WBC/hpf on routine urinalysis. Hemoglobin was 15.1 g/dl, WBC 13,900 per cubic mm with 90% neutrophils and platelets 238,000 per cubic mm. His serum creatinine was 1.49 mg/dl while the rest of his renal and liver parameters were normal.

A CT Brain showed an old infarct in the right basal ganglia and no other significant abnormality. A CSF examination revealed 80 WBCs per cubic mm with 15% neutrophils and 85% lymphocytes; protein 104.2 mg/dl, and glucose of 5 mg/dl (parallel blood glucose of 100 mg%). The India ink preparation showed capsulated budding yeast suggestive of cryptococcal species (Fig. 1). A diagnosis of CM was made and he was commenced on liposomal amphotericin B (5 mg/kg/day) and flucytosine. Tacrolimus and MMF were discontinued.

An MRI Brain with gadolinium showed abnormal diffuse pachymeningeal thickening and intense enhancement along bilateral cerebral convexities, interhemispheric fissure, tentorium, and cerebellar folia suggestive of meningitis. There was no focal enhancing lesion or hydrocephalus.

After an initial improvement in sensorium, 2 days later he became excessively drowsy and dyspneic. An HRCT chest showed features suggestive of infective bronchiolitis. Bronchoalveolar lavage showed few pus cells, moderate number of RBCs and India ink staining confirmed capsulated budding yeast suggestive of Cryptococcus species.

A scraping from the right nasal skin lesion was positive for India ink staining, showing multiple capsulated budding yeast.

After another 12 h, he had generalized tonic clonic convulsions which were treated with midazolam and levetiracetam. He was intubated for airway protection. A CT Brain

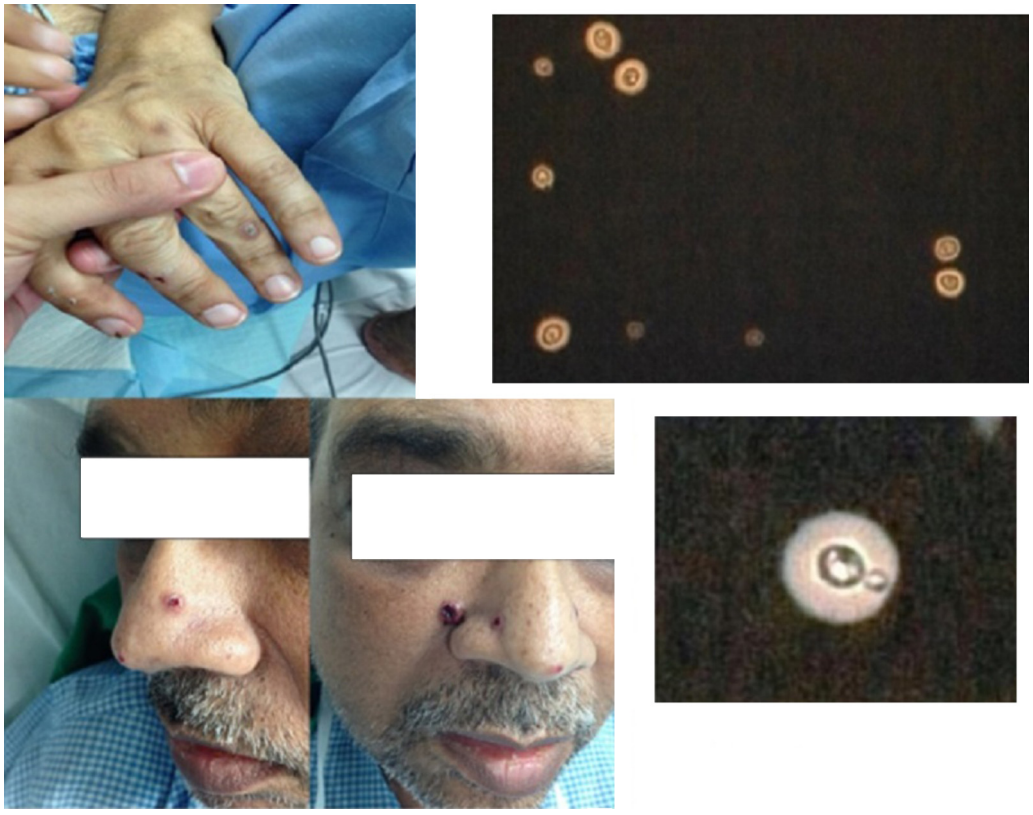

Fig. 1 – India ink preparation showing capsulated budding yeast and the skin showing cryptococcomas https://www.google.co.in/search?espv=2&biw=1366&bih=628&site=webhp&q=Cryptococcomas&spell=1&sa=X&ved=0CBkQvwUoAGoVChMIu8W96vv6yAIVhhiUCh3m-woO.