

A RARE CASE OF FATAL FUNGAL MITRAL VALVE ENDOCARDITIS

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ABSTRACT:

With the continuing rise in the number of immune-compromised patients, the incidence of invasive mycoses has increased. There are various patterns of fungal infections. Because of limitations in ante mortem clinical diagnosis owing to lack of sensitive diagnostic tools, information regarding frequency and pathogenesis of fungal infections is largely dependent on autopsy. We report a case of 50 year old diabetic female with lower limb cellulitis died of subarachnoid haemorrhage which showed mixed cirrhosis and fungal endocarditis. This presence of fungal endocarditis in a non diseased heart with no major risk factor like previous cardiac surgery or illicit drug abuse, but along with cirrhosis and diabetes is rare. Also fungal endocarditis is commonly associated with hemorrhagic stroke but subarachnoid haemorrhage is very rare.

Keywords: *Aspergillus, Endocarditis, Liver cirrhosis, Subarachnoid haemorrhage.*

Key Messages: *The unusual feature of this case was fungal endocarditis (FE) in a non diseased heart with no major risk factor like previous cardiac surgery or illicit drug abuse but in cirrhosis and diabetes although reported but rare questions the pathogenesis of FE. Also fungal endocarditis is commonly associated with hemorrhagic stroke but subarachnoid haemorrhage is very rare.*

INTRODUCTION

Fungal endocarditis (FE) is rarely diagnosed early enough for prompt intensive therapy that has halved the fatality of bacterial endocarditis from 95%. Fungaemia still tends to be ignored when it is transitory, and serological tests are not widely used. The incidence of resultant FE is difficult to estimate.¹ 10 to 20% of clinical infective endocarditis are abacterial. 30% of those detected only at post-mortem had been blood culture-negative. Fungal endocarditis accounts for less than 5 % of all the community acquired infective endocarditis. The major predisposing factors are cardiac surgeries, multiple illicit drug abuse, immunosuppressant and valvular heart diseases. They commonly present as culture negative endocarditis with large vegetations. The common causative organisms include *Candida albicans*, non *albicans* species of *Candida*, *Aspergillus*.

CASE HISTORY

A 50 year old female presented with fever with chills since 4 days. H/O swelling and wound on the right leg on the medial aspect since 1 month which is non healing. Patient was a known case of diabetes mellitus. On examination: General condition was fair, vitals were stable, CVS: S₁S₂ heard with loud P₂. RS: breath sounds were bilaterally equal. P/A: soft non-tender, mild hepatosplenomegaly, CNS: drowsy, oriented, speech heavy, comprehension present. Local examination: Non – contaminated wound on the medial aspect of the right leg about 4X5 cm with sloping edges.

INVESTIGATIONS:

Hemogram showed thrombocytopenia with platelet count of 78000/cumm, Liver Function Test was deranged, Random blood sugar levels were raised (174 mg/dl), ESR was raised (134/min), INR was raised (2.15), LDH was markedly raised (2469 IU/L), CT brain : subarachnoid haemorrhage.

On Post-mortem examination: Heart was enlarged and globular. Mitral valve shows single large bulky friable protruding vegetations which was infiltrating into the cusps and involving the chordae tendinae and papillary muscles (Fig 1). Bilateral subarachnoid hemorrhage involved entire convex surface predominantly the lateral surface and also involved basal surface (Fig 3).

Few blood clots were noted at the origin of left cerebral artery and in Sylvain fissure. Liver was enlarged, external surface showed micro and macro nodules measuring 2mm to 1 cm. Capsule was stretched as the liver was enlarged. Cut surface was firm in consistency, yellowish brown in colour, nodular suggestive of mixed cirrhosis (Fig 2 & 9). On histopathological examination, sections of heart showed mild myocardial hypertrophy of the muscle with infiltration of acute inflammatory infiltrate and fungal hyphae (Fig 4). GMS stain for fungal hyphae showed branching at acute angles was stained black with sharp margins and cleared centre seen (Fig 6 & 7). Sections of brain showed oedema with congestion of meningeal vessels and subarachnoid haemorrhage (Fig 8). Sections of liver showed focal areas of fatty changes. There was formation of multiple nodules separated by bridging fibrosis portal to portal & portal to vein with extensive bile duct proliferation

and mononuclear inflammatory infiltrate in the septate.

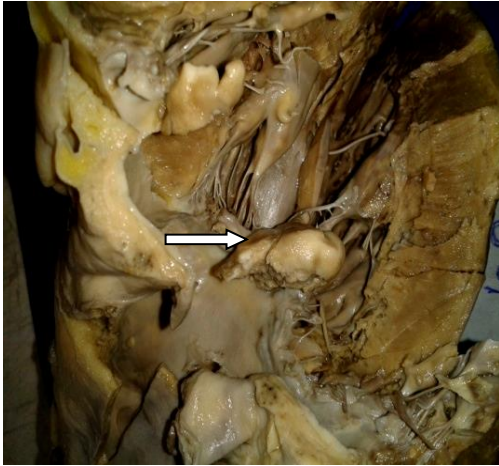


FIG.1: Mitral valve showing bulky friable vegetation.



FIG.2: Mixed cirrhosis of liver.

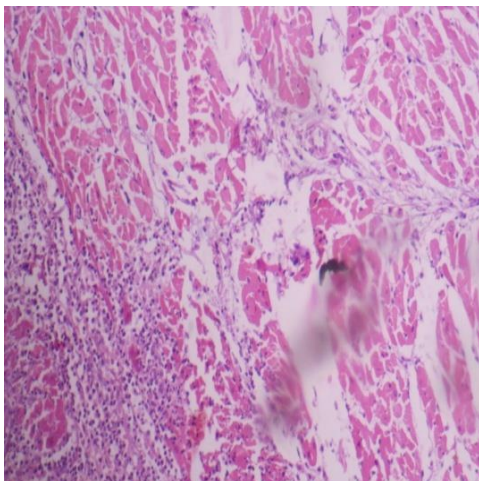


FIG.3: Subarachnoid haemorrhage.

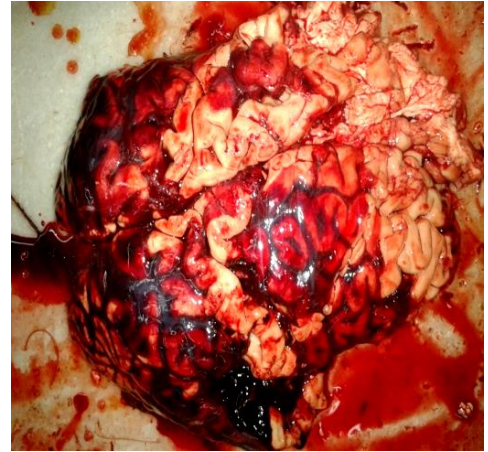


FIG.4. H&E section of heart with cardiac muscle hypertrophy.

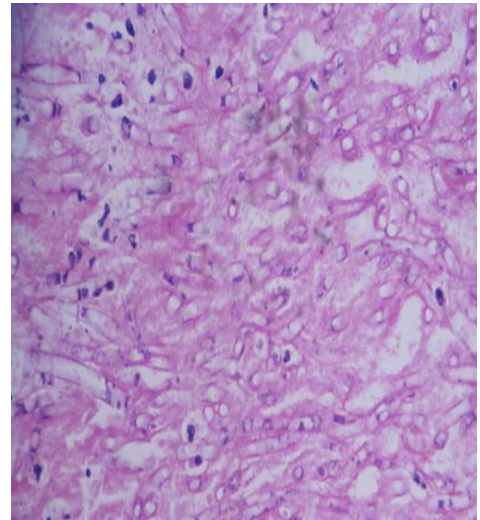


FIG. 5: H & E section from vegetation showing fungal hyphae.

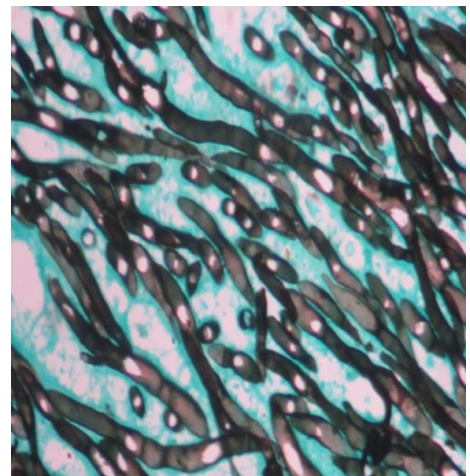


FIG. 6: Fungal hyphae seen as darkly stained strands against light green background of unstained normal cardiac tissue.

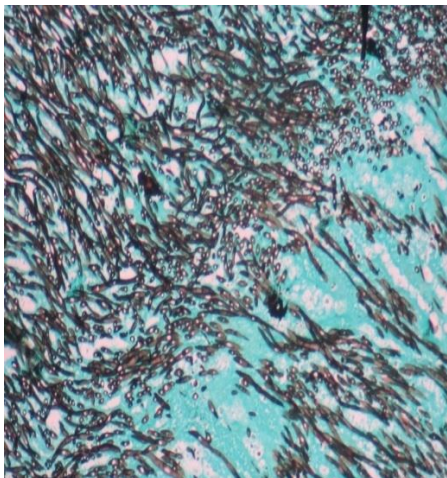


Fig. 7: Fungal hyphae seen as darkly stained strands against light green background of unstained normal cardiac tissue.

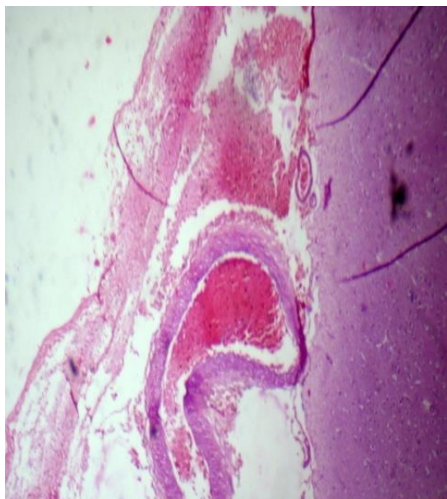


FIG.8: Sections of brain showing oedema with congestion of meningeal vessels and subarachnoid haemorrhage.

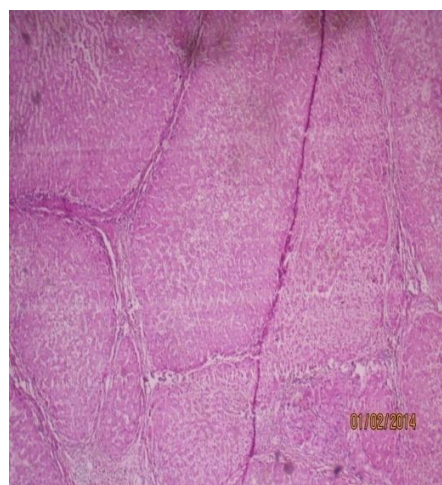


FIG.9: Sections of liver show focal areas of fatty changes. There is formation of multiple nodules separated by bridging fibrosis portal to portal & portal to vein with extensive bile duct proliferation and mononuclear inflammatory infiltrate in the septa.

DISCUSSION

Fungal endocarditis is not rare. It usually develops in patients with abnormal or surgically traumatized hearts, to whose blood fungi have gained access, perhaps during temporary (often iatrogenic) impairment of host defences. In our case as patient was diabetic and had lower limb cellulitis, the broad spectrum antibiotics used for treatment allowed fungus to establish itself through indwelling intravenous catheters for nutritional or diagnostic purpose, other mechanisms like toxin irritated gut by mycelia invasion, yeast presorption, environment fungi can reach the blood via indwelling catheter sleeve thrombi in endocardium traumatized by tubes that reach the heart as described by M. Seelig et al study of fungal endocarditis its risks and treatment. Although the antifungal therapy cleared fungus rapidly, it can establish itself in the endocardium, where it grows slowly.¹

Thus, clinical and laboratory procedures (including blood and urine cultures) that have permitted early diagnosis and treatment of bacterial endocarditis, are not reliable in fungal endocarditis. As this is a case was diabetic, which itself is an immunosuppressed state, fungal infections are common. This case also has cirrhosis which may be secondary to infective aetiology like Hepatitis virus and nosocomial infections like staphylococcus, streptococcus, enterococcus, pseudomonas as no serology testing was done when patient was alive and post mortem serology testing was not conclusive of any aetiology. Infectious endocarditis is seen in up to 60 % cases of cirrhosis these are seen due to immune dysfunction as noted by Bonnel A R et al which reported 10% IE associated with cirrhosis.² R B Hsu et al reported 26 cases of infective endocarditis with cirrhosis in which hepatitis virus was isolated in 20 cases.³

Cirrhosis is also known to predispose to infectious endocarditis as reported by Fernandez Guenrero ML et al which concluded Staphylococcus aureus as most common organism, but fungal endocarditis is extremely rare.⁴ In presence of both these co morbid conditions the terminal event causing death was sub arachnoid haemorrhage which was, on post-mortem we detected fresh clots at Circle of Wills which suggests aneurysmal bleed. Hemorrhagic stroke and infective endocarditis go hand in hand as suggested by Yanagiri-hara C et al, their study too showed presence of sub arachnoid haemorrhage, but was rare.⁵

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