# **Aspergillous Myocardial Absceses**

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### 36.1 Clinical History

A 38-year-old male, nondiabetic and nonhypertensive, had been admitted in 3 private health-care facilities in the past 9 days for a left gluteal abscess. It had developed within a day of an intramuscular injection for an unspecified febrile illness. On the first day of the third admission (that lasted for 7 days), he had gone debridement of a large wound, which had now formed in the gluteal region. He developed acute renal failure and was put on hemodialysis. He was then shifted to our tertiary-care center due to financial constraints.

On examination, he was afebrile, conscious, and well-oriented with reference to time and place. The pulse was 110 per minute and blood pressure was 106/90 mmHg. At this point of time, there was no fever. There was a large wound  $20 \times 15$  cm over the left gluteal region; the wound floor showed muscle, which was partly obscured by purulent exudates. There was an extension  $15 \times 5$  cm over the lower back, which was also covered with purulent material. The investigations have been tabulated (Table 36.1). On the

#### Table 36.1 Investigations

Hematological	<sup>a</sup> Hemoglobin 6.8 g/dL
	<sup>a</sup> Total leukocyte count 17,550/cmm
	Differential count—Neutrophil
	predominant, with band forms,
	metamyelocytes and
	metamyelocytes and cytoplasmic
	toxic granules
	<sup>a</sup> Platelet count 3.15 lakhs/cmm
Biochemical—	<sup>a</sup> Random blood glucose 90 mg/dL
Routine	<sup>a</sup> Serum creatinine 4.1 mg/dL
	<sup>a</sup> Blood urea nitrogen 57.9 mg/dL
	<sup>a</sup> SGOT 201.1 U/L
	<sup>a</sup> SGPT 80 U/L
	<sup>a</sup> Total bilirubin 0.7 mg/dL
	<sup>a</sup> Sodium 134 mEq/L
	<sup>a</sup> Potassium 4.1 mEq/L
	<sup>a</sup> Chloride 116 mEq/L
Radiological	Chest computed tomography—
Ū.	Bilateral pleural effusions with
	basal consolidations
Others	Urine examination: Proteinuria 1+,
	pus cells 50–60 per high power
	field
	Wound culture: Acinetobacter
	species
	Blood culture: Negative

<sup>a</sup>Mean values

Check for updates

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**Fig. 36.1** (a) Scanned section of the left ventricular free wall showing a central area of breakdown; (b) Intramural coronary arteritis flanked on either side by abscesses (H&E  $\times$  100); (c) The lumens are occluded by fresh fibrin thrombi with associated necroses of their walls (H&E  $\times$ 

second day of admission, another extensive debridement was performed under general anesthesia. He was administered intravenous broadspectrum antibiotics and was on hemodialysis. But the patient did not improve and developed altered sensorium and anuria. There was gradual deterioration and he expired 9 days after the operation.

### 36.2 Autopsy Findings

A complete autopsy was performed on a thin built male. The debrided wound was still covered with dirty slough and suppurative exudates. The heart was of normal size and weighed 240 g. All the chambers, valves, and coronary arteries were normal. Transverse slices of the ventricles, however, revealed multiple foci of congestion with breakdown of tissue in the left ventricular wall

250); (d) Necrotic wall infiltrated by septate, slender, basophilic hyphal forms (H&E × 400); (e) A fairly circumscribed area of myocardial abscess formation (H&E × 250); (f) Fungal elements admixed with neutrophils (H&E × 400)

(Fig. 36.1a). On histology, these areas showed intramural coronary arterial radicles containing fresh fibrin thrombi with destruction of their walls and the presence of radiating lightly basophilic slender septate hyphal forms (Fig. 36.1bd) resembling those of Aspergillus species (See Chap. 19). Surrounding such foci of necrotizing arteritis were many myocardial abscesses with hyphal filaments (Fig. 36.1e, f). Similar fungal micro-abscesses were also present in the brain (Fig. 36.2a-c). Surprisingly, the consolidated lung did not show any fungal elements even on the Gomori methenamine silver stain. Other findings were acute tubular necroses and pigment cast nephropathy (Fig. 36.2d) with focal microabscesses (no fungi seen) in the kidneys. Neutrophils were seen in the sinusoids of the liver and spleen.

**Cause of Death:** Septicemia with fungal myocardial and cerebral abscesses.



**Fig. 36.2** Micro-abscesses seen in the cerebrum (a)  $H\&E \times 200$  and (b)  $H\&E \times 400$ ; (c) Scattered fungal hyphae (arrow) were present (Gomori methenamine silver

 $\times$  400); (d) Presence of reddish-orange pigment cases in the collecting tubules (H&E  $\times$  250)

## 36.3 Discussion

A young immunocompetent man developed cardiac and cerebral necrotizing suppurative lesions following a large nonhealing wound over his left lower extremity. Such infective cardiac lesions form a diverse group of disorders, caused by a variety of microorganisms with involvement of one or more layers of the heart. Myocardial abscesses are found in about 0.18-1.52% of autopsies performed in adults and are very often caused by Gram-positive cocci especially S. aureus. In most instances, they are seen in patients with valvular or mural infective endocarditis (See Part V) either as a direct extension of the infection or through septic coronary arterial embolization. Very rarely, the softened and necrotic myocardium following an acute infarction and coronary arterial interventions (See Chap. 29) are contributing factors for abscess formation. Myocardial abscesses can also develop through dissemination from a distant septic focus or as a manifestation of sepsis, which was the mechanism in the case presented. The organism identified within the abscesses was a fungus,

which had morphology of the *Aspergillus* species.

In general, unlike bacteria and viruses, fungi seldom produce infections in the immunocompetent hosts, except for superficial mycoses. Most of the invasive fungal diseases occur as opportunistic infections in individuals, whenever there is disruption of the immunological responses. The circumstances that permit penetration of immune barriers are immuno-deficient states, organ transplantation, hematological/non-hematological malignancies, administration of corticosteroids/ antineoplastic drugs/prolonged broad-spectrum antibiotics, malnutrition, extensive surgery, critically ill immunocompetent patients, and contaminated devices. In such patients, diagnosis of fungal disease is not easy and requires a battery of tests, which include microbiological isolation, histopathological demonstration of tissue invasion, detection of fungal antigens, and even molecular analysis. Very often, the diagnosis is made or confirmed after postmortem examination. After Candida species, the organisms included in the genus Aspergillus are the next common fungi that are capable for producing disseminated disease. The most common cardiovascular manifestation is infective endocarditis (See Chap. 19) and even with dissemination, the incidence of myocardial involvement is low, to the extent of 12%. These filamentous fungi are capable of producing saprophytic colonization, allergic reactions, and invasive disease depending on the host immunity. Since the spores of Aspergillus are ubiquitous and air-borne, pulmonary involvement is most frequent. In this case, however, the areas of bronchopneumonic consolidations failed to reveal the organisms even with special staining techniques, and very surprisingly, there were only myocardial and cerebral abscesses. This indicates that there would have been a hematogenous dissemination through the nonhealing wound. Even in and around the regions of the abscesses, the fungus showed the characteristic feature of angio-invasion with thrombotic occlusion of the intramural coronary arteries.

In most of the patients, the abscesses are scattered within the myocardium and clinical presentation would depend on the sizes and locations of the lesions. Few of these patients can remain asymptomatic, which is often the case with fungal dissemination. In some, the symptoms can be variable and vague, while others can present with conduction abnormalities, arrhythmias, valvular insufficiency (especially if the abscess is peri-valvular), congestive heart failure, myocardial rupture, or even sudden death. Clinical suspicion of fungal infection particularly in patients without obvious predisposing factors for such extensive, nonhealing wounds and the use of tissue biopsy and/or microbiological studies for fungi would have facilitated administration of appropriate antifungal therapy rather than broad-spectrum antibiotics. Unfortunately, material from the nonhealing wound was not sent for fungal cultures. Also, cardiac imaging would be useful for the detection of the life-threatening complication of fungal myocardial abscesses.

#### **Further Reading**

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