## Infective endocarditis by *Abiotrophia defectiva* presenting as acute coronary syndrome

*Abiotrophia defectiva* accounts for <1% of infective endocarditis (IE),[1] with <10 documented case reports from India. Interestingly, all these were insidious in onset with chronic duration and adequate time to plan for corrective surgery.[1–4] We report probably the first case of IE presenting with features of acute coronary syndrome by *Abiotrophia defectiva* from India.

A 65-year-old man on mechanical ventilation was referred from a local hospital with complaints of worsening dyspnoea and chest pain for 4 days. His blood examination was within normal limits. His random blood sugar was 428 mg/dl, and fasting sugar 419 mg/dl. HbA1c was 11.3%, urine sugar was strongly positive and ketones were present. On admission his renal function test was normal. Troponin I levels (2598 ng/L) were elevated and an echocardiogram showed severe aortic regurgitation with vegetations on the aortic valve and a normal chamber. Three sets of blood cultures were processed in BacT/ALERT microbial detection system. Gram-stain from flagged positive blood culture bottles showed Gram-positive bacilli. Chocolate and blood agar grew α-haemolytic colonies after 48 hours of incubation. There was no growth on MacConkey agar. Colony smear showed short slender irregularly stained pleomorphic Gram-positive bacilli with many showing clubbed ends (Fig. 1). It was catalase-negative, urease-negative and non-motile both at 37 ºC and room temperature. Acid-fast and modified acid-fast staining showed non-acid-fast bacilli. Albert staining did not show metachromatic granules. As VITEK identification gave inconclusive result, the isolate was sent for confirmation by MALDI-TOF and was identified as *Abiotrophia defectiva* (high confidence level 99.9).

On the day of admission, the patient was started on ceftriaxone 2 g i.v. and other supportive measures. By day 4 of hospital admission, his renal parameters worsened (urea 95 mg/dl, creatinine 3 mg/dl, decreased urine output), developed pulmonary oedema and died due to cardiac arrest.

There are several unique clinical and microbiological features pertaining to this case:

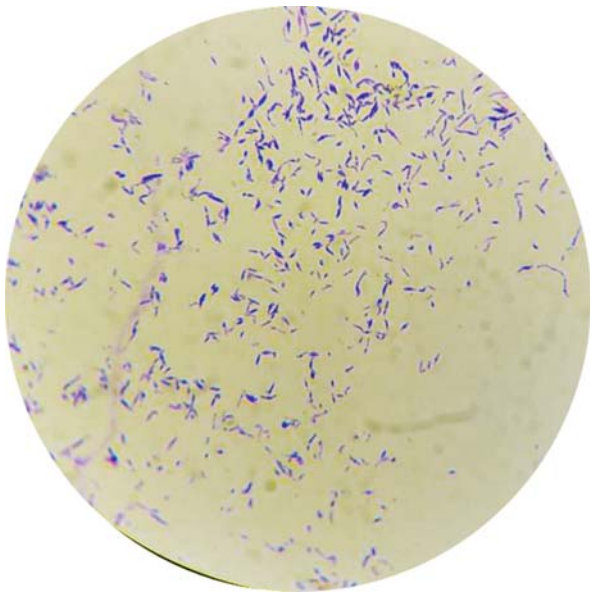

FIG 1. Smear showing Gram-positive bacilli with clubbed ends

1. Organism morphology: *Abiotrophia defectiva* has been classically described as a nutritionally variant streptococci[3] which are Gram-positive coccobacilli in chains.[2] In our patient, this organism showed irregularly stained Gram-positive bacilli with clubbed ends, which morphologically resembled *Corynebacterium* spp. This organism was ruled out by doing Alberts stain, catalase and urease test. Acid-fast staining and motility was also done to rule out other rare Gram-positive bacilli such as *Mycobacterium* spp. and *Listeria*.
2. Growth requirements: *Abiotrophia defectiva* is highly fastidious, dependent on either L-cysteine or vitamin B6 for its growth. They will not grow when subcultured on the usual enriched plates such as chocolate agar and blood agar.[5] In our laboratory, we routinely prepare chocolate agar by adding haemoglobin powder and IsoVitaleX to soyabean casein digest agar. As IsoVitaleX contains L-cysteine, they grew readily on chocolate agar. The growth on blood agar can be explained as we had used human blood agar due to interruption in supply of 5% sheep blood agar. Human blood may contain some factors that support the growth of this organism.
3. Clinical presentation: Most published cases of IE due to *Abiotrophia* were of insidious-onset with a good outcome. These reports emphasized on early diagnosis and treatment of this virulent organism for preventing mortality. [1–4]

Due to technical feasibility,[6] antibiotic susceptibility pattern for this organism was not attempted. The European Society of Cardiology recommends penicillin G, ceftriaxone or vancomycin for 6 weeks combined with an aminoglycoside for 2 weeks.[7] Ceftriaxone was started empirically for our patient. Gentamicin was not added later, as his renal parameters were worsening. The valvular vegetations causing left ventricular dysfunction must have resulted in acute coronary syndrome, resulting in the death of our patient, despite starting him on adequate treatment. Hence, we postulate that more than the virulence of this organism, it is the acute nature in which the disease had progressed giving hardly any time for cardiac compensatory mechanisms, which became life-threatening.

### REFERENCES


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