LATE PROSTHETIC VALVE ENDOCARDITIS DUE TO CARDIOBACTERIUM HOMINIS, AN UNUSUAL COMPLICATION

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Abstract

We report a case of prosthetic valve endocarditis caused by *Cardiobacterium hominis* in a patient who had undergone atrial septal defect closure and mitral valve replacement of the heart in 1978. He presented with pyrexia of unknown origin and congestive cardiac failure. Investigations revealed infective endocarditis of prosthetic valve in mitral portion. Blood culture samples grew *C. hominis*. The patient was empirically started on vancomycin and gentamicin intravenously and ceftriaxone was added after isolation of the organism. Though subsequent blood cultures were negative, patient remained in congestive cardiac failure and died due to complications.

Key words: *Cardiobacterium hominis*, HACEK, prosthetic valve endocarditis

*Cardiobacterium hominis*, is a member of the HACEK group (*Haemophilus* species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens* and *Kingella kingae*) of gram negative bacilli. The organism is an unusual cause of human disease, notorious for causing culture negative endocarditis. Only 11 cases of prosthetic valve endocarditis by *C. hominis* have been described internationally. To the best of our knowledge, there are no case reports from India documenting *C. hominis* infection.

Prosthetic valve endocarditis is an endovascular, microbial infection occurring on parts of a valve prosthesis or on reconstructed native heart valves. Management of infection of an intracardiac device or prosthesis is challenging and calls for a multidisciplinary approach. We herewith report a case of late prosthetic valve endocarditis due to *C. hominis*, complicated with congestive cardiac failure, 26 years after valve replacement.

Case Report

A 61-year-old male patient was referred to our hospital for evaluation of pyrexia of unknown origin. In 1978 he had undergone atrial septal defect closure and mitral valve replacement with 28 mm Carpentier Edward bioprosthetic valve. Clinical examination revealed that he was febrile (101°F), pale, had irregular pulse rate of 96/min, respiratory rate of 18/min and blood pressure of 130/90 mmHg. He was in congestive cardiac failure with severe mitral regurgitation, severe pulmonary hypertension and tricuspid regurgitation. Neurologic examination was normal. Laboratory investigations revealed hemoglobin 9.06 g/dL, ESR 80 mm/hour, total WBC 10.3K/µL, creatinine 1.4 mg/dL, platelet count 110 K/µL, albumin 2.5 g/dL, globulin 5.9 g/dL, AST 28 IU/L, ALP 92 IU/L. ECG showed rate of 100/min in atrial fibrillation with extreme right axis and features of right ventricular hypertrophy. Echocardiogram showed severe valvular and paravalvular mitral regurgitation, normal sclerotic aortic valve, moderate tricuspid regurgitation, severe pulmonary arterial hypertension and no definite evidence of endocarditis. At this point of time, a differential diagnosis of intravascular hemolysis secondary to paravalvular regurgitation versus infective endocarditis was made. Subsequent urine analysis did not reveal urobilinogen in excess or hemoglobinuria. Trans esophageal echocardiogram done next day showed vegetations in prosthetic mitral valve (Fig. 1).

The patient was started on vancomycin (1 gm 12 hourly) and gentamicin (40 mg 8 hourly) after collecting blood samples.

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Figure 1: Trans esophageal echocardiogram showing vegetations in prosthetic mitral valve

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for culture. In addition, ceftriaxone (2 gm OD) intravenously was also started. Patient became afebrile from day five of starting antibiotic therapy. Patient’s renal function started deteriorating, necessitating discontinuation of vancomycin and gentamicin. Intravenous penicillin (20IU four hourly) was started. Patient underwent dialysis for correction of renal functions. Subsequent blood cultures were negative. But, throughout the course of hospitalization he remained in congestive cardiac failure. In addition he also developed pneumonia due to *Klebsiella pneumoniae* and needed ventilatory support. In spite of all supportive measures, patient deteriorated day by day and died due to complications of renal failure and secondary pneumonia.

Four blood culture (BACTEC 9240) samples, two aerobic and two anaerobic, were collected before starting antimicrobial therapy. Two aerobic cultures flagged positive after 96.41h and 141.29h respectively. Gram smear of positive cultures showed pleomorphic gram negative bacilli. Subculture was done on MacConkey agar, blood agar and chocolate agar. Tiny, glistening opaque colonies with no hemolysis on blood agar and chocolate agar were noted after 72h of incubation at 37°C in a CO₂ incubator. There was no growth on MacConkey agar. Plates were further incubated up to one week and there was no pitting of media.

The isolate was nonmotile, oxidase positive, catalase negative. Gram smear of colonies showed pleomorphic gram negative bacilli with one or both ends swollen (Fig. 2).

Possibility of an organism belonging to the HACEK group was informed to the clinician. It fermented glucose, sucrose and maltose. It was nitrate negative, indole positive, urease negative, lysine, arginine and ornithine decarboxylase negative. The organism was identified as *Cardiobacterium hominis*, based on morphology, cultural characters and biochemical reactions. The isolate was β-lactamase negative.⁵

Sensitivity was done by Kirby-Bauer method on Mueller Hinton agar with 5% sheep blood supplement. Growth was scanty after three days of incubation. Sensitivity test was then attempted on blood agar in anaerobic jar and result was noted after 48h. The isolate was sensitive to penicillin, amoxycillin, gentamicin, amikacin, ceftriaxone, cefotaxime and vancomycin.

**Discussion**

*Cardiobacterium hominis* is a fastidious, gram-negative bacillus, which is present as normal flora of oropharynx. It is a member of the HACEK group, exhibiting the common characteristics of a slow growth rate and CO₂ requirement for optimal growth and causing similar clinical conditions. It is a facultative anaerobe. *C. hominis* should be distinguished from other members of the HACEK group and from *Pasteurella, Brucella, Streptobacillus moniliformis* and *Bordetella parapertussis*. The main characteristics of *C. hominis*, distinguishing it from other closely related organisms are absence of catalase activity, positive oxidase reaction, production of indole and absence of nitrate production.⁶

The virulence of *C. hominis* for humans is extremely low. Prosthetic valve endocarditis has conventionally been considered as early and late with 60 days regarded as the time limit for early cases because of differences between the microbiology and pathogenesis of infection in the two time periods. Early prosthetic valve endocarditis results from perioperative contamination and is thus a hospital infection. Not all these early infections present within 60 days of operation and this has led some investigators to suggest that the time limit for early disease should be extended to six months or even a year. Commonest organisms are likely to be *Staphylococcus* from patient’s own commensal flora and may be from operating room staff. Late onset infections are community acquired and are caused by contamination with low virulence organisms at surgery or by asymptomatic bacteraemia. A probable source of infection can be found in 25-80% of patients, the most frequent causes being dental procedures, urological infections and interventions and indwelling catheters. The most common organisms are *S. epidermidis, S. aureus*, viridans streptococci and enterococci.¹²

*In vitro* susceptibility testing may be difficult to perform and interpret because of the fastidious nature and slow growth of *C. hominis*.⁷ There are no breakpoints for this organism, nor have the correct medium or atmosphere and time of incubation been established.⁸ The organism is susceptible to penicillin and may be successfully treated with four to six weeks course of penicillin alone or in combination with an aminoglycoside.⁹ Sensitivity to vancomycin is variable. Resistance to erythromycin and vancomycin has been reported. These agents should be considered when there is a good *in vitro* sensitivity.¹⁰

HACEK organisms account for 10% or less of early and late prosthetic valve endocarditis. Clinically, these cases are characterized by a subacute or chronic course and often present with embolic lesion from large vegetation and...
congestive cardiac failure. In this case, the patient was in congestive cardiac failure at the time of admission. His clinical course was complicated with renal failure, which improved with dialysis. Risk of recurrent infection is common in patients with prosthetic valve endocarditis. Cardiac complications, advanced age and severity of illness are predictors of poor outcome. Hence, surgery was planned in this case after completion of antimicrobial therapy. He responded well to antimicrobial therapy and subsequent blood cultures were negative. But, congestive cardiac failure remained unresponsive to therapy. He died before completing the antibiotic course due to complications of renal failure and secondary pneumonia. This case illustrates the occurrence of infection due to *Cardiobacterium hominis* and highlights the importance of blood culture by a good blood culture system, which enabled the isolation of a rare and fastidious organism.

**References**


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