



Penicillin Resistant *Aerococcus viridans* Septicemia in an Immunocompetent Person: An Unusual Case Report

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Abstract

Aerococcus viridans is a catalase negative, gram positive coccus that is usually air borne and widely distributed in hospital environment. This is an unusual pathogen causing bacteremia and septicemia. *Aerococcus viridans* can be easily misidentified as alpha hemolytic streptococci because of similar colony morphology on blood agar, with staphylococci they share microscopic appearance and with enterococci they partly share antibiotic resistance pattern. Although aerococci were reported to be of low virulence but still it can cause severe infection in immunocompromised host. Infection in immunocompetent host is very rarely documented. Herewith we are reporting a case of septicemia caused by *Aerococcus viridans* in an immunocompetent host. To the best of my knowledge after web search of medical literature this is the first documented case of septicemia caused by *Aerococcus viridans* in an immunocompetent host from India. Patient treated successfully with appropriate antibiotic therapy.

Keywords: *Aerococcus viridians*; Immunocompetent; Septicemia

Introduction

Aerococcus viridans is a microaerophile, catalase negative, gram positive cocci, that is usually found singly, pairs, tetrads or in group like clusters [1]. *Aerococcus viridans* is mainly considered as pathogen of lobsters and rarely causes human infections. The organism is often misidentified as alpha hemolytic streptococci in routine laboratory practice. Hence correct species identification was very difficult in past and it was thought that aerococci to be a rare cause of human infection [2]. Now with the introduction of improved methods for species determination aerococci have been increasingly recognized as human. Herewith we are reporting a case of aerococcal septicaemia in a host without having obvious immunocompromised factors.

Case Presentation

A 25 year old female admitted to our hospital with chief complaints of high grade fever (104°F), pain in lower abdomen and fast breathing (38/min) since last 3 days. She was hypotensive with a blood pressure of 78/48 mmHg and pulse rate 102/min. There was history of vaginal delivery of a male baby in her district hospital seven days before admission to this hospital (tertiary care hospital). On examination other findings like cardiovascular system and central nervous system were within normal limits. Relevant laboratory parameters include; hemoglobin 12.8 gm/dl, total leukocyte count 16200/cmm, neutrophil 86%, lymphocyte 12%, eosinophil 2%, monocyte 0%, basophil 0%, total platelet count 3 lacs/cmm, erythrocyte sedimentation rate 48 mm after first hour, serum urea 26 mg/dl, serum creatinine 0.6 mg/dl, Na⁺ 136 mmol/L, K⁺ 3.3 mmol/L, leptospira IgM negative (by ELISA), dengue NS1 Ag negative (by ELISA), malaria parasite negative (by immunochromatography method). Chest X-ray normal, ECG normal, ultrasound of lower abdomen showed few product of conception in uterus. Blood was collected with complete aseptic precaution into aerobic and anaerobic blood culture bottle (Bact/ALERT/3D; BioMerieux, Marcy l'Etoile, France). Patient was started empirically with intravenous Imipenem 500 mg 6 h and intravenous vancomycin 500 mg 6 hourly to cover both gram negative and gram positive organisms. Aerobic culture bottle showed positive sign of growth after 72 h. Broth was then sub cultured on 5% sheep blood agar. After overnight incubation blood agar plate showed alpha hemolytic colonies which were 1 mm to 2 mm in diameter and circular in shape. Gram stain was done and it was gram positive coccus, arranged in pairs, tetrads and clusters (Figure 1). They were catalase and bile aesculin negative. Further identification was done by vitek-2 (fully automated identification system) using GP test card (Bio Merieux, Marcy l'Etoile, France). It was identified as *Aerococcus viridians*

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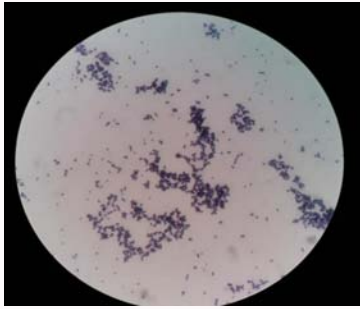


Figure 1: Gram stain was done and it was gram positive cocci, arranged in pairs, tetrads and clusters.



Figure 3: After overnight incubation at 37°C plate showed 1 mm to 2 mm yellow coloured colonies.

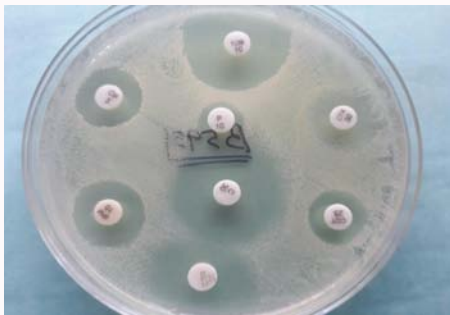


Figure 2: The isolate was found to be sensitive to vancomycin, gentamicin, tobramycin, chloramphenicol, etc and resistant to clindamycin, erythromycin, penicillin, co-trimoxazole, ciprofloxacin etc.

with 99% probability. There was no growth in anaerobic blood culture bottle even after 7 days of anaerobic incubation. The isolate was found to be sensitive to vancomycin, gentamicin, tobramycin, chloramphenicol, imipenem, ceftriaxone, ofloxacin and resistant to clindamycin, erythromycin, penicillin, co-trimoxazole, ciprofloxacin and levofloxacin (Figure 2). As per antibiotic sensitivity report both the antibiotic (vancomycin & Imipenem) were sensitive, so it was continued for 10 days. Urine sample was also collected with aseptic precaution in a sterile container and sent to microbiology department for culture and sensitivity. Direct microscopy of uncentrifuged urine sample showed 4 to 6 pus cells/hpf, and 2 to 3 gram positive cocci/hpf. Then sample was inoculated on CLED (cysteine lactose electrolyte deficient) media. After overnight incubation at 37°C plate showed to 2 mm yellow colored colonies (Figure 3). Gram stain was done and it was gram positive cocci with similar arrangements as on blood agar. It was catalase and bile aesculin negative. Further identification was done by vitek-2 using GP test card, and it was *Aerococcus viridans* with 98% probability. Isolation of *Aerococcus viridans* from both the clinical samples (blood & urine) confirmed its pathogenic role. Patient responded very well to antibiotic therapy. Dilatation and curettage was done to remove retained product of conception. Fluid resuscitation was also done to combat blood pressure. Blood and urine sample was taken 7 days after completion of therapy, and there was no growth. Patient is doing well during follow-up.

Discussion

Aerococcus was first described in 1953 with description of *Aerococcus viridans* as type isolate, present in dust and air. In 1967 a report was published consisting of 8 isolates of *Aerococcus* Like Organisms (ALO) from human infections, and it was distinct from *Aerococcus viridians*. The importance of *aerococcus* like organisms was clearly shown by christensen et al. in their series of works leading

to the definition of new species. In 1992 *Aerococcus urinae*, which comprises of most of the *aerococcus* like organisms was defined. In 2001 another clinically relevant species *Aerococcus sanguinicola* was defined. Later on two additional aerococcal species have been identified in humans i.e. *Aerococcus christensenii* from vaginal tract, which seems to be an uncommon cause of infections, and *Aerococcus urinaehominis* from urine, whose pathogenic role in human being is not clear till now [1]. Clinical microbiologists often failed to identify aerococci as it can be easily misidentified as alpha hemolytic streptococci, as both have similar colony morphology on blood agar (alpha hemolysis). It also misidentified as staphylococci, because of similarity in their microscopic appearance and with enterococci they partly share antibiotic resistance patterns. Now with the introduction of improved methods for species determination especially vitek-2 compact, Matrix-Assisted Laser Desorption Ionization Time of Flight-Mass Spectrometry (MALDI-TOF MS) and sequencing of 16S rRNA gene by PCR, aerococci have been increasingly recognized as human pathogen [3]. Normally aerococci appears to be of low virulence but they become pathogenic in certain vulnerable conditions. Genus aerococci is comprises of seven species of which three have been considered as important human pathogens. *Aerococcus urinae* causes endocarditis, urinary tract infection, septicemia, pyelonephritis, cellulitis, peritonitis, spondylodiscitis, lymphadenitis and soft tissue infections. *Aerococcus viridians* causes systemic infection in immunocompromised hosts including; endocarditis, septicemia, bacteremia, septic arthritis, urinary tract infection, spondylodiscitis, meningitis, para-aortic abscess, wound infection and osteomyelitis. *Aerococcus sanguinicola* causes invasive infections including; endocarditis, bacteremia, cholecystitis and urosepsis. Other rare species associated with human infections are *Aerococcus christensenii* and *Aerococcus urinaehominis* [4]. Risk factors for human infection have not been fully understood, but prolonged hospitalization, invasive procedures, prolonged antibiotic treatment, granulocytopenia, oral mucositis and implantation of foreign bodies have been described as major risk factors for systemic infection of *A. viridians* [5]. Aerococci are environmental isolates and its distribution is comprised of 5% to 10% of the bacterial flora of air and dust of housing premises (hospitals, classrooms, offices, factories etc). It is also found on raw vegetables, animal and animal products, as well as on human skins [6]. *Aerococcus viridans* appears as pairs, tetrads or clusters in gram stain and has worldwide in distribution. Most of the aerococci are sensitive to β -lactam antibiotics as well as several other antibiotics. The pattern of resistance however had shown some important differences among different species. *A. urinae* and *A. sanguinicola* shows low MICs for penicillin, cephalosporins and carbapenems. *A. viridans* shows higher MICs for penicillin,

and acquired resistance to penicillin is well documented [7]. In our case also penicillin was resistant. MICs to aminoglycosides are generally variable among aerococcal species. *A. urinae* generally display highest MICs for gentamicin. *A. viridans* was uniformly sensitive to vancomycin but now a day's vancomycin resistant *A. viridans* has been reported in medical literature. Antibiotics such as chloramphenicol, erythromycin, clindamycin and tetracycline rarely used against aerococcal infection but they are generally effective *in vitro*. Antibiotic susceptibility pattern were studied in order to differentiate *A. viridans* from *Aerococcus* Like Organisms (ALO). It indicates that *A. viridans* is sensitive to tobramycin and bacitracin and resistant to penicillin and furazolidone. In present case we also found same susceptibility pattern. Differentiation between *A. urinae* and *A. viridans* can be done by testing for Pyrrolidinyl Amino Peptidase (PYR), which is only positive for *A. viridans* and Leucine Amino Peptidase (LAP) which is only positive for *A. urinae*. Further automated identification system like vitek-2 is reliable for phenotypic identification and 16S rRNA PCR is gold standard for genotypic confirmation [8].

The normal habitat of *Aerococcus viridans* is not known but aerococci are found in the normal gut flora of chickens, and from this point it has been speculated that they might reside in the human gut flora [9]. From previous report it was found that *A. viridans* can produce bacteriocin which is logical for a bacterium that competes with other species of the normal flora. Aerococci can colonize the urinary tract and causes invasive infection because it has ability to form biofilm on foreign materials. Infective endocarditis should be highly suspected in a patient with *A. viridans* bacteremia, as this pathogen can activate and aggregate human platelets and bacteria can participate in the formation of infective endocarditis. The molecular mechanism behind platelets activation depends on the activation of specific antibodies towards the bacteria and complement activation [3]. Since virulence mechanisms of aerococcal species is not well studied so it will be highly interesting to analyze genome of different aerococcal species for the presence of reputed virulence factors. In our case source of infection might be air and dust present in the hospital premises as the patient had gone for delivery four days before she presented the present complaints.

For many years aerococcal species were identified by laboratories with special interest in these bacteria. But now a day there is increased awareness of aerococcal infections, combined with improved technology will lead to more correct identification as human pathogen.

Vitek-2 and MALDI-TOF-MS should be used instead of biochemical methods in order to correct identification of aerococcal species. Sequencing of the 16S rRNA gene remains the gold standard. Many basic questions about aerococci remain to be answered like its normal habitat, interaction with host, genomic study, virulence mechanisms and phylogenetic relationship [10]. Even though *A. viridans* is rarely associated with human infections but it could be potential causative agent of bacteremia and septicemia. Increased knowledge on aerococci among clinicians and clinical microbiologists will improve the management of patient suffering from aerococcal infection. However more study is needed to understand its pathogenesis and antibiotic susceptibility pattern which will help clinicians in optimal treatment of this rare pathogen.

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