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A fatal case of septicemia caused by multi-drug resistant (MDR) Serratia marcescens; a case report from a tertiary care hospital

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Abstract: There has been an increase in hospital acquired infections due to organisms like *Pseudomonas spp.*, *Acinetobacter spp* and other members of enterobacteriaceae. *Serratia spp.* is now emerging as an important nosocomial pathogen with the ability to rapidly spread in the hospital environment. Outbreak due to *Serratia spp.* have been reported especially in the neonatal age group. However, we are reporting a fatal case where an adult man succumbed to his illness due to multidrug resistant (MDR) *Serratia marcesens* septicemia in an intensive care unit (ICU) setting. **Keywords:** *Serratia marcesens*, septicemia, multidrug resistant.

INTRODUCTION

Serratia marcescens has been recognized as a cause of hospital-acquired infection for the last two decades and accounts for 16% of nosocomial infection[1]. Many nosocomial infections like septicemia, pneumonitis and meningitis are caused by multiple antibiotic-resistant strains of S. marcescens, [2]. Serratia sppis rapidly spreading in the hospital environment posing danger to hospitalized patients, and hospital personnel should be vigilant in preventing nosocomial outbreaks due to this organism[1]. Various factors that appear to predispose to serratia infection included prior corticosteroid therapy, the post-operative status. mechanical respiratory manipulation, instrumentation of the genitourinary tract, multiple and "broad-spectrum" antibiotic therapy, and chronic, debilitating disease[3]. Various outbreaks have been reported in the neonatal age group but we are here reporting a fatal case of Serratia marcescens septicemia in an adult man.

CASE REPORT

A 52 year old male was admitted in the trauma ICU of the hospital. He was admitted after road traffic accident and was put on mechanical ventilation. Amikacin and ceftraixone was started as an empirical treatment. As our hospital's protocol, endotracheal aspirate was sent for culture on day 2 of hospitalization. Endotracheal aspirate culture was performed semiquantitatively using Maki's and Cleri's technique which grew non lactose fermenting colonies. On gram's staining gram negative bacilli was seen. They were motile, oxidase negative organisms. The organism was subjected to various biochemical tests. The organism did not produce indole, nitrate was reduced to nitrite, citrate positive, alkaline slant with acidic butt on triple sugar iron agar, urease was produced, and glucose, maltose, mannitol, and sucrose were fermented with acid production. The organism was sub-cultured on nutrient agar; they produced red pigment which was non- diffusible in the medium. The organism was provisionally identified as Serratia spp and was further tested in Microscan Walk Away 40 plus system in gram negative combo panel and was identified as Serratia *marcescens*. The antibiotic susceptibility testing was performed by Kirby Bauer disc diffusion testing for the following antibiotics, ampicillin, gentamicin, amikacin, ciprofloxacin, ceftriaxone, cotrimoxazole, piperacillintazobactum, aztreonam, and imipenem. The isolate was subjected to double disc synergy testing for extended spectrum beta lactamase detection (ESBL) by cefotaxime and cefotaxime with clavulanic acid. There was no zone around cefotaxime and cefotaxome with clavualnate due to production AmpC beta-lactamases.

After three days of intubation, patients developed respiratory distress and high grade fever. Samples were sent for pathologic, biochemical and microbiological investigations. After investigations, Haemoglobin was 10.2g%, total leucocyte count (TLC) - 15400 mm3, Platelet-1,98000 and ESR- 35 mm at the end of first hr. The biochemical investigations revealed that random blood glucose sugar was 64mg%, Liver function test, kidney function tests and electrolytes within normal limits. Total proteins were normal but globulins were increased. Dengue serology (NS-1 antigen and IgM antibody ELISA) was negative and HIV was non-reactive. Widal was performed and the titers were negative. Urine culture was sterile. Blood

culture was obtained in Bact Alert was sent for culture and antibiotic sensitivity testing.

Blood culture showed positive signal after 24 hours of inoculation and broth was inoculated on Blood agar and Mac-Conkey agar and incubated at 37 °C for 16 - 18 hours. Blood agar showed beta-hemoltyic colonies and Mac-conkey agar showed non lactose fermenting colonies which was also identified as *Serratia marcescens* by conventional and automated method. Isolates obtained from blood and endotracheal aspirate were resistant to all the major group of antibiotics and was susceptible only to imipenem. Patient succumbed to his illness within 2 days development of fever.

DISCUSSION

Serratia marcescens has emerged as a nosocomial pathogen with high affinity for damp environment and hence some of the sources of this organism are humid rooms, plastic nebulisers, disinfectant solutions, contaminated blood bags and mouth-wash solutions. However, spread via contaminated hands was considered as most important mode of transmission when no environmental source was identified[4]. In this case, the organism must have got access to the patient's respiratory system via endotracheal tube through contaminated hands leading to colonization by Serratia marcescens which later got entry into the blood stream leading to septicemia and hence, patient presented with high grade fever and later succumbed to his illness in-spite of antibiotic treatment as the isolate was multi-drug resistant.

Serratia marcescens was isolated from both endotracheal suction catheters and blood culture with same antibiogram profile suggesting that both these isolates were clone of a single strain which first colonized the endotracheal tube and through the tube; it got access to the patient's circulatory system leading to septicemia. Serratia spp. has high potential for transmission of resistance via plasmids responsible for high level of drug resistance in a hospital setting as seen in this isolate.Serratia also possesses chromosomally coded AmpC beta-lactamases which is in concordance with the results of double disc synergy testing for ESBL detection showing no zones around cefotaxime and cefotaxime with clavulanate[5]. It could also acquire resistance plasmids from other members of enterobacteriaceae present in the hospital environment or from patient's own respiratory commensal flora.

Gentamicin was initially considered as drug of choice for treatment of Serratia infections but now there is upsurge in resistance to gentamicin. The resistance was found to be around56-81%[6,7]. Then, amikacin replaced gentamicin and became drug of choice for treatment of nosocomial infections but use of amikacin is limited to due to ability of *Serratia spp*to develop resistance during the therapy[8]. So, it would no longer be wise to choose amikacin empirically in a nosocomial infection due to *Serratia spp*. unless in vitro testing showed that the isolate was susceptible. In the present case, the isolate were resistant to all the major classes of anti-microbial agents like ampicillin, gentamicin, amikacin, ciprofloxacin, ceftriaxone, cotrimoxazole, piperacillin-tazobactum, aztreonam and sensitive only to imipenem. So, amikacin and ceftriaxone were not effective and patient succumbed to his illness.

Serratia spp. are associated with various nosocomial infections like ventilator associated infections, septicemia, cellulitis post biopsy etc.[2]. So, clinicians should be informed about the possibility of infections caused by Serratia spp. To decrease the morbidity and mortality caused by Serratia spp., strict hospital infection control policy should be implemented along with rapid detection and identification of the organism with the antibiotic susceptibility. It will help in treating organism at time by right drug, thus decreasing the spread of organism in the hospital and further help in improving patient care.

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