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Original Research Paper

Neonatal sepsis caused by a rare pathogen *Comamonas* terigena in a tertiary care hospital: A case report

Microbiology

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ABSTRACT

Comamonas terigena has been rarely observed as an infectious agent in clinical practice. The organism has the low virulence potency and infrequently causes human disease. Comamonas species (previously classified within the Pseudomonas group) have widespread environmental distribution and also survive for a long time in hospital environments. 7The present case being reported is a one day old neonate who had developed respiratory distress following sepsis caused by this unusual pathogen. To the best of our knowledge, this is the first case of sepsis caused by C.terrigena being reported from this part of the country. Though this organism is difficult to isolate by manual methods which are mostly used by Indian laboratories, the emergence in hospitals strongly suggest the community acquired transmission of this rare organism. Hence stressing on the needs of cleaning and infection control practices to be followed by people outside the hospital settings also.

INTRODUCTION

Comamonas terigena has been rarely observed as an infectious agent in clinical practice. The organism has the low virulence potency and infrequently causes human disease. *Comamonas* species (previously classified within the *Pseudomonas* group) have widespread environmental distribution and also survive for a long time in hospital environments [1, 2]. The infection by *Comamonas terrigena* is under diagnosed and underreported, and hence considered a rare pathogen. Majority of the cases of sepsis by *Commamonas* have been caused by another species like *C. acidovorans* and *C. testosteroni*.[3-7]The present case being reported is a one day old neonate who had developed respiratory distress following sepsis caused by this unusual pathogen. To the best of our knowledge, this is the first case of sepsis caused by *C.terrigena* being reported from this part of the country.

CASE REPORT

A male new born baby delivered at 1:49 pm on 20/9/2016 in our institution presented with symptoms of respiratory distress at 6 am on 21/9/16. The baby, weighing 3.2kg was a full-term neonate and was born by Emergency LSCS done in view of fetal distress. Baby had shown spontaneous respiratory efforts and cried at birth. APGAR score at birth and 10 minutes later was 7/10 and 8/10 respectively.

Antenatal history: Mother was G1P0A0, 39 weeks with fetal oligohydramnios. Fetal heart rate was 90-11/min .She had complaint of fever 3 days back along with vague pain in abdomen. There was no history of leakage or bleeding per vaginum or rash or UTI. Antenatal Ultrasonography revealed mildly reduced anterior fontanel and loop of entangled umbilical chord around neck.

Day 0: On examination, the general condition of baby was sick. The baby was euthermic with absence of pallor or icterus and capillary filling time was normal. Peripheral cyanosis was present.Loud grunt could be heard and intercostal recession was seen. Heart rate and respiratory rates were 180/min and 74/min respectively. On chest auscultation, bilateral air entry was present. Cardiovascular system and per abdominal examination did not reveal any significant findings. On CNS examination, anterior frontanelle was depressed and reflexes were decreased. Significant laboratory findings were raised SGOT 127IU/L, elevated Prothrombin time 64 sec.CSF examination showed raised protein 110mg/dl, decreased sugar of 30mg/dl. At 6 am on 21/9/2016 loud grunting and peripheral cyanosis was noted. Based on clinical findings, a provisional diagnosis of respiratory distress with sepsis was made. The baby was put on CPAP, i.v. fluids and antibiotics were started and transferred to NICU for further management.

Day 1

After admission to NICU the APGAR score of baby was found to be 6/10.Heart rate was 178/min. Respiratory rate 78/min. Intercostal recession was seen and loud grunting was heard On initial investigations, complete hemogram showed decreased Hemoglobin and Total Leucocyte Count. C - reactive protein was reactive 42mg/dl. Blood culture was sterile For fluid and electrolyte correction, i.v. fluids and calcium gluconate were given. Injectable Piperacillin-Tazobactam 300mg i.v. over 12hours was started. Ionotropes were given to manage shock.

Day2,

Despite the therapy, the baby's condition continued to deteriorate. The case was reviewed by senior pediatrician this point the baby was intubated .Two unit of PCV was transfused in view of increased pallor and second sample for blood culture was sent. Combined respiratory and non respiratory acidosis was seen on arterial blood gas analysis .There were repeated alternate variations of increasing and decreasing spo2 in the same day. There was metabolic acidosis at the end of the day with rise of spontaneous respiratory efforts. Injectable Vancomycin (45mg i.v. 8 hourly) was started along with continuation of previous treatment and one point PCV was transfused. In the evening concentration of adrenaline was increased and the spo2 came to 93%.in evening the condition of baby was better with rise in spO2.

Day 3

General condition remained sick. Injectable Amikacin was started replacing Vancomycin after literature survey and discussion between treating clinicians and microbiologists. Rest of the treatment was continued as earlier. Blood culture sent on second day came out to be sterile. Fresh sample was sent for blood culture. On the basis of clinical and X ray finding the diagnosis of respiratory distress with sepsis with persistent pulmonary hypertension was made. On 9:30 pm 5 seconds of bradychardia was seen and there was a sudden disconnection which lead to the lowered readings. The concentration of the inotropes was again increased and the patient did find spo2 of 47%.

Day 4

Baby's general condition worsened on day 4 with further fall in spO2. While doing suctioning at 7pm baby had a dip in saturation for 5-7 seconds which revived later to normal. There was an alteration of SpO 2values throughout the day .There was sudden zero value of spO2 and no breath sounds at 7 pm though initially the baby was giving signs of improvement. There was failure to revive the child despite adequate efforts and he went into refractory shock due to

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respiratory failure and succumbed to death.

Two Blood cultures sent on third day were cultured aerobically for 24 hours. On nutrient agar, colonies were around 2mm round and convex with a smooth to wavy margin and a smooth to granular surface. No pigmentation was seen on the surface of media. It was non lactose fermenting on maconkey's medium. Identification of isolate was done b BD-Phoenix automated identification system on day five The organism was found susceptible to Ampicillin,PiperacillinTazobactam,Ceftazidime,Imipenem,Meropenem and Aztreonam.On grams stain of the organism it was a straight or slightly curved gram negative bacilli with size of around 0.5×3 µm.Hence it was considered as the pathogen responsible for sepsis

DISCUSSION

Comamonas strains are common inhabitants of soil, mud, and water, in both natural and polluted environments. It was isolated from contaminated soil in Slovakia. This bacterium showed remarkable biodegradation properties. Cells of *C. terrigena* strain N3H are capable of degrading the anion active surfactant dihexyl-sulfosuccinate and have been applied for this purpose immobilized in alginate gel 8. They have also been isolated from various clinical samples, the hospital environment, and horse and rabbit blood. Sources include blood, pus, urine, pharyngeal mucosa, kidneys, feces, burst appendix, intravenous tubing, and urinary catheters[9]. *Comamonas* strains are isolated from such samples using methods for the isolation of Gram-negative glucose-nonfermenters. This includes the use of a blood agar medium, such as tryptic soy agar plus defibrinated blood, and a selective enteric medium, such as Maconkey's agar. The commonly used incubation regime for primary Isolation media of 24 h at 35 C should be extended with 24 h at 30 C to permit growth of glucose-nonfermenters that grow slowly at 35 C and may be masked by other bacteria .*C. kerstersii* is a subgroup of Comamonas terrigena, and has been linked to cases of perforated appendices[10].

There have been few reports of sepsis caused by *C.testosteroni*, but after extensive search the author could find only one published reports of *C.terrigena*[11] which was acquired outside the hospital from an environmental source. The source of infection in our case could have been maternal as she also had reported fever and pain in abdomen 3 days before delivery. Though this organism is difficult to isolate by manual methods which are mostly used by Indian laboratories, the emergence in hospitals strongly suggest the community acquired transmission of this rare organism. Hence stressing on the needs of cleaning and infection control practices to be followed by people outside the hospital settings also.

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