Intrauterinely acquired pseudomonas septicemia in the neonate

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Abstract The case is presented of a premature infant with early onset pseudomonas aeruginosa infection, acquired in utero. The infection itself was fulminant, rapidly progressive without skin rash. Peripheral blood picture showed severe neutropenia, thrombocytopenia and anemia. Although early onset sepsis with this organism is extremely rare in newborns, it may pose a severe life-threatening challenge to premature infants.


Keywords: Neonatal septicemia, pseudomonas.

Pseudomonas infection in neonates occurs mainly as a result of contaminated environment, including manipulative instrumentation or prolonged ventilation. Intrauterinely acquired pseudomonas infection presenting as early onset sepsis in the newborn infant is extremely rare1,2,3 and in most cases it is accompanied with characteristic skin rash. We report a premature infant with unusual early onset pseudomonas infection with bone marrow aplasia but without any skin rash.

Case report A 32-week gestation female infant with birth weight of 1390 g was delivered with apgar score of 5 and 7 after one hour and five minutes. Mother was 38-years old, gravida 4, para 3, abortion nil, and was referred to a peripheral hospital as a case of premature uterine contractions, abdominal pain and fever (39.5°C) with chills of two days duration. Pregnancy was uneventful. She received nitrodrine hydrochloride, decadron and augmentin two days before delivery. Spontaneous labor began within two hours of membrane rupture. Amniotic fluid was meconium stained. Her white blood count was 21,000/cmm with normal differential and platelet counts. Fetal heart sounds during labor were normal. She delivered in Al Wasl Hospital within a few hours after transfer.

Initial examination of the infant revealed pallor, hypoactivity and mild tachypnea. Chest x-ray was consistent with moderate degree hyaline membrane disease. Baby had metabolic acidosis with pH 7.10, HCO3-12 and base excess -16.7 with normal PaO2 and PaCO2. Blood urea and electrolytes were normal. Complete blood count showed total white blood cells 1600/cmm with polymorphs 4%, lymphocyte 91%, monocytes 5%, reticulocytes 2.6%, nucleated red blood cells 32/100 white blood cells, platelets 29000/cmm and hemoglobin 5.9 g/dl. C-Reactive protein was 24 mg/l. Cerebrospinal fluid (CSF) findings were within normal limits. Ampicillin and gentamicin were started. Baby was transfused twice with fresh whole blood. Baby needed mechanical ventilation because of clinical deterioration and respiratory failure.

Complete blood count repeated at 20 hours of age showed total white blood cells 1200/cmm with polymorphs 4%, lymphocytes 96%, reticulocyte count 1.4%, nucleated red blood cells 95/100 white blood cells, platelets 6000/cmm and hemoglobin 14.4 g/dl. Dopamine infusion was started 10 μg/kg/min with 5% albumin 10ml/kg as infant had dropping blood pressure 29/17 (MAP 19mmHg). A double volume exchange transfusion was performed at 25 hours of age as a life saving measure but condition went on deteriorating and

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infant expired at 27 hours of age. Baby did not develop macular rash. Just before expiry, a bone marrow aspiration was done which was suggestive of aplasia with no evidence of malignant or infiltrative disease. Baby grew *Pseudomonas aeruginosa* from two blood cultures, deep ear, gastric and eye swabs, which were resistant to ampicillin but sensitive to gentamicin; CSF culture was sterile. Maternal high vaginal swab also grew *Pseudomonas aeruginosa*. Mother became afebrile on second post partum day and was discharged three days after delivery.

**Discussion** Pseudomonas aeruginosa is a gram-negative, motile, rod-shaped aerobic bacterium which multiplies in almost any moist environment including distilled water and some disinfectants. It is frequently responsible as a cause of late onset neonatal septicemia (after the first 48 hours of life and nosocomial in origin) but only rarely causes early onset septicemia (within first 48 hours of life and antenatally acquired).\(^1\) Pseudomonas species, being opportunistic pathogens usually invade compromised hosts.\(^5\) Premature infants being compromised hosts, do acquire pseudomonas infections more often than full term or adult individuals.

Pennis et al\(^3\) reported the case of a premature infant of 29-30 weeks gestation with birth weight 1490 g. Infant was born 22 hours after rupture of membranes. The mother was symptomatic with fever, chills and had greenish yellow foul smelling vaginal discharge. This infant was stillborn. Postmortem of lung tissue grew pseudomonas.

Kraus and Hunter\(^2\) reported a 3500 g, full term infant, with early onset pseudomonas infection which was acquired intrauterinely. The mother presented with fever, chills, lethargy, diarrhea and abdominal pain one day prior to delivery. The infant developed characteristic rash, respiratory distress and leukopenia soon after delivery. Cultures from blood, CSF, spleen and pleural exudate grew pseudomonas and infant expired. Although amniotic fluid and lochia cultures were sterile, maternal stool culture grew same species of pseudomonas.

Rovalo C and Bauer CR\(^1\) reported the case of a premature infant of 36 weeks gestation with birth weight 3880 g, born by lower segment cesarean section. Mother was nearly asymptomatic but complained of heavy ‘perspiration’ several weeks before delivery. Amniotic fluid was meconium stained. Baby presented with respiratory distress soon after, needing intubation and mechanical ventilation. Infant developed macular rash at 31 hours of age and total white blood cell count dropped to 4800/cmm. This baby survived but was handicapped by profound hearing loss.

Rudeman JW\(^6\) reported a 35 weeks gestation with birth weight 2140 g, born normally to a mother who did not have any symptoms. Baby developed respiratory distress within first hour of life accompanied by characteristic macular skin rash. Peripheral blood picture showed total white blood count 3200/cmm. Cultures taken from mother’s endometrium first post partum day grew pseudomonas aeruginosa.

Dennis CS\(^3\) reported nine cases of pseudomonas sepsis, four of which presented within 72 hours of life. Three of these infants presented with early onset respiratory distress and chest x-ray abnormalities consistent with hyaline membrane disease. None of the patients developed macular skin rash.

Our baby also presented with respiratory distress within few hours of life and chest x-ray had findings of hyaline membrane disease. Baby had severe leukopenia, thrombocytopenia and marked anemia but without characteristic macular rash as reported by others. Infant’s mother had history of fever, chills and abdominal pain two days before delivery and her high vaginal swab grew the same species of pseudomonas. Despite aggressive management the infant expired.

Although intrauterinely acquired pseudomonas septicemia is extremely rare in neonates it needs consideration in special circumstances. Leukopenia and/or pancytopenia is uncommon in the first few days of life. Most infants reported with early onset pseudomonas septicemia, presented with leukopenia. Our patient had severe pancytopenia with bone marrow aplasia. We feel infants with suspicion of sepsis accompanied with leukopenia, especially pancytopenia during first few days of life, carry a high probability of serious gram negative organism, e.g. pseudomonas. Characteristic skin rash for early onset pseudomonas septicemia may be absent as disease could be so fulminant that infant may succumb before its appearance. Prompt initiation of appropriate antibiotic therapy, depending upon hospital specific sensitivity, may minimize the mortality and morbidity.

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**References**


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