Salmonella senftenberg Carrier State in a Neonate Following Septicemia

Salmonella serotypes are one of the frequent etiologic agents incriminated in nosocomial epidemics in neonatal intensive care units. Nursery outbreaks due to *S. typhimurium, S. anatum, S. newport, S. weltevreden* and *S. oranienberg* are associated with high morbidity and mortality(1). However, *S. senftenberg* is rarely reported as a cause of neonatal septicemia. Only four cases of *S. senftenberg* septicemia have been reported in the newborns from India(2,3). We report a similar case of neonatal septicemia who at follow up continued to excrete *S. senftenberg* in the stools.

A full term SGA, boy, weighing 2000 g was born to a third gravida by an emergency LSCS for fetal distress. The membranes ruptured 6 h prior to delivery and the amniotic fulid was meconium stained. The infant had Apgar scores of 3 and 6 at 1 and 5 min, respectively. During resuscitation thick meconium was aspirated from trachea. Soon after, the baby had respiratory distress and bilateral crepitations. A diagnosis of meconium aspiration syndrome was made and the child was treated with intravenous fluid, oxygen, ampicillin and gentamicin. Chest skiagram revealed retrocardiac pneumonitis. The blood culture was sterile. Respiratory distress settled by fourth day and feeds were initiated. On fifth day, swelling of left upper limb was noticed (thought to be due to extravasation). Subsequently, bluish red patches appeared over dorsum of left hand and wrist. By eighth day, few of these had pus-discharge and the rest showed black discoloration. The child developed fever, poor activity, increasing prefeed aspirates and abdominal distension with poor bowel sounds. The total leucoyte count was 20,200/cu mm with 86% polymorphs, platelet count was 1,20,000/cu mm and peripheral smear revealed bandemia and toxic granulations. Radiographs of chest and abdomen were normal. Oral feeds were stopped. Intravenous fulids, cefatoxime (100 mg/kg) and amikacin (15 mg/kg) were started. Local exclusive dressing with framycetin was done. *S. senftenberg* was isolated from blood, pus and rectal swab. It was sensitive to amikacin and resistant to ampicilin, gentamicin, chloramphenicol, cephalexin and co-trimoxazole. Efforts made to trace the source from nursery personnel, patients, environment, equipment and maternal wound or stool were unrewarding. Since the child showed improvement, same antibiotics were continued for two weeks. Fever and abdominal distension subsided within 5 days. Feeds were restarted on fifteenth day. During follow up, the child continued to excrete *S. senftenberg* in stools till 3 months of age but remained asymptomatic. Repeated blood cultures were sterile.

S. senftenberg infection, though predominantly zoonotic can affect man through contaminated food and water. *S. senftenberg* has survived in pork-pies and caused a large outbreak in Midlands(4).

All the four cases of neonatal septicemia caused by *S. senftenberg* reported from India were preterm or small for date. Three of them suffered severe birth asphyxia. The symptoms, predominantly alimentary, appeared between 3-7 days. The organism was sensitive only to gentamicin, amikacin and cephalosporins(2,3).

To the best of our knowledge, the development of carrier state following neonatal septicemia caused by *S. senftenberg* has not been reported so far. The present case continued to excrete the bacillus in the stool for 3 months. No treatment was offered, as symptomatic neonates excreting *Salmonella* in stools ultimately get rid of the infection. Moreover, it is difficult to eradicate the carrier state of most *Salmonella* serotypes(1). Cases of *S. senftenberg* septicemia need to be closely followed up to document the carrier state in view of its epidemiological implications as the index case may be a potent source of infection to the susceptible contacts in community.

P. Gupta,
M.M.A. Faridi,
M.V. Murali,
V.G. Ramachandran,
V. Talwar,
Departments of Pediatrics and Microbiology,
University College of Medical Sciences and GTB Hospital, Delhi 110 095.

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Persistent Pulmonary Hypertension in the Newborn

With reference to the letter entitled 'Persistent Pulmonary Hypertension in the Newborn' (PPHN)(1), we would like to offer the following comments.

Though the most probable diagnosis of the case would be PPHN following meconium aspiration syndrome (MAS), the definitive diagnostic criteria for PPHN include hyperoxia test, hyperoxia-hyperventilationt test, preductal versus post-ductal arterial PO_2, rapidly fluctuating arterial PO_2 or transcutaneous PO_2 demonstrated on continuous recording and use of contrast echocardiography to demonstrate patent ductus arteriosus (PDA) and patent foramen ovale (PFO)(2).

Hence, the full diagnostic criteria have not been established. No mention of the FiO_2 is there in the entire report. For diagnostic purposes, hypoxemia should persist even in 100% oxygen as the first step towards confirmation of PPHN. The most definitive test, hyperoxia-hyperventilation test has not been mentioned. A difference of 15 mm Hg or more from preductal and post-ductal sites are significant of right to left shunting only if both the PaO_2 values are around 100 mm Hg or less. This test has to be done in 100% oxygen or at lower FiO_2 concentrations adequate to prevent cyanosis. Demonstration of shunt via PFO and/or PDA using contrast