SHIGELLA SONNEI BACTEREMIA (REPORT OF A CASE)

GULSEN HASCELIK
ERDOGAN BERKMAN

SUMMARY: Shigella bacteremia is a rare event. A case of Shigella sonnei bacteremia in a 15-year-old patient suffering from congestive heart failure is discussed.

Key Words: Shigella sonnei, bacteremia.

INTRODUCTION

Infections due to Shigella species are generally considered to be confined to the gastroinstestinal tract (1) with extraintestinal infection being rare (2). Meningitis (3), osteomiyelitis (4) and bacteremia (5) have been described in newborns and children. Invasion of the blood-stream has been thought to be a rare and usually self-limited event (1). Such infection presents a high risk of death, particularly in malnourished children (5).

We describe here a case of Shigella sonnei bacteremia in a 15-year-old boy.

CASE REPORT

A 15-year-old boy was admitted to Hacettepe University, Medical School Hospital, on November 3, 1988, with dyspne, palpitation and watery diarrhea without mucus or blood. He was well until 7 days before admission. He had been operated because of Ebstein anomaly on April 22, 1988 and treated with digitalis since that day. Initial physical examination showed an oral temperature of 38°C, blood pressure 100/50 mmHg, regular pulse at a rate of 104 beats per minute and respirations 34 per minute. He was in respiratory distress and without signs of volume depletion. There were physical and radiographic findings of congestive hearth failure. Examination of the abdomen disclosed hepatosplenomegali.

His initial laboratory studies revealed a hematocrit value of 32 per cent and leukocyte count of 40600, with 92 segmented neutrophils, 8 lymphocytes. The urinalysis was normal, with the exception of excessive calcium carbonate crystals. Concentrations of carbon dioxide, chloride, sodium, glucose and potassium in the serum were within normal limits. The blood urea nitrogen (BUN) was 9 mg per 100 ml (N: 10-20 mg%), serum creatinine was 0.5 mg per 100 ml (N: 0.9-2 mg%), total serum protein was 8 gr per 100 ml (N: 6-8 gr%) with 2.9 gm albumin was 2.5 gr per 100 ml (N: 4-5.5 %). The serum biluribin alkalen fosfatase, serum glutamic oxaloasetic transferase (SGOT) and serum glutamic pyrivate transferase (SGPT) were also in normal values. Pharyngeal swab culture grew normal flora and stool culture grew Klebsiella spp. Shigella sonnei was identified in six blood culture bottles.

At the time of admission, the patient was started dipyridamol 3 x75 mg/day, digoxin 1 mg/day, salicylic acid 500 mg, cefotaximine 100 mg/kg/day, amikacin 15 mg/kg/day. Temperature elevations to 40.2°C orally and tachycrdia were present during the first 4 hospital days. His loose stool discontinued on 3rd day of admission. Because of high temperature, his antibiotic was changed to intra-venous sulbactam-ampicilline 200 mg/kg/day and was continued until the patient's death on the 10th hospital day.

Other diagnostic procedures performed in an attempt to locate a nidus of infection. A telecardiogram showed cardiomegaly and echocardiogram revealed no abnormalities. Cerebrospinal fluid was normal and cultures of it revealed no growth. Before the patient's death, the CO_2 was 27.9 mEq per liter; chloride, 86 mEq per liter; sodium, 125 mEq per liter; and potassium, 4 mEq per liter. Numerous subsequent blood cultures failed to grow any bacteria. His death ended attempts to find the source of infection.

Bacteriology: Culture pharyngeal swab grew normal flora and culture of stool grew Klebsiella spp. On the day of admission. No Shigella spp. Were found from the feces during hospitalization. Urine culture made at the time of the patient's admission to the hospital were sterile.

From Departments of Microbiology and Pediatrics, Hacettpe Universtiy, Ankara, Turkiye.

On the day of admission, 6 blood cultures made several hours apart were positive for a non-motile Gram-negative rod which was citrate, indole and urea negative, and which did not ferment lactose, sucrose. It agglutinated strongly with Shigella sonnei, Group D specific antiserum.

Antibiotic sensitivities, performed by the disk method, using the organism isolated from the blood, revealed marked *in vitro* sensitivity tomezlocillin, trimethoprim-sulphamethoxazole, netromycine, tobramycine, cephtazidime, sulbactam-ampicilline, ceftriaxone, cefotaxime and cefoperazone.

DISCUSSION

Shigella bacteremia is rare, occuring mostly in children (6). Probably much more bacillemia occurs and is missed because both physician and bacteriologist are convinced of the truth of aphorism, restated in bacteriology texts (7), that blood culture and serology are not helpful in shigellosis. In addition, the course the bacillemia does not correlate with the severity of the diarrhea (8).

There may well be an element of underestimation of the incidence of shigella bacteremia. This may be explained by several factors: 1) failure to obtain blood cultures at the proper time in the illness or not at all (8,9); 2) inhibition of bloodborne Shigella species by humoral factors and leukocytes (9); and 3) prior administration of antibiotics. It is possible that bacteremia is most likely to be detected during the initial phase of the illness (9), when patients frequently do not consult physicians or are treated symptomatically. In our patient, S. sonnei was first isolated from 6 blood cultures performed on the first admission day, but it did not grow on the last hospital days. Simultaneously, our patient was not treated with antibiotics prior to admission.

The circumstances which determine invasion of the blood-stream by shigella are unknown. The pathogenesis of shigellemia involves penetration of the bacterium through the gut epithelium to the lamina propria. Variations in virulence or inoculum size may explain occasional breakthrough to the blood. Since immunity against bacillary dysentery is believed to be mediated in the gut mucosa (1), inherited or acquired disturbances of local immunity may also increase the likelihood of bacteremia in certain individuals. The presence of underlying disease (8, 10), or a state of malnutrition (5), as well very old or young age (9, 10) may influence the normal balance resulting in invasion of the blood-stream by shigella. In our patient, the portal of entry for the shigellemia was probably the intestinal tract, although limited radiographic studies showed no lesions attributable to the organism. Invasion of the blood-stream probably occurred before admission, since S. sonnei was demonstrable within the first 12 hours of hospitalization. The possibility that stools obtained at the time of admission did contain Shigella can not be discounted. This likelihood seems improbable, since feces, enrichment broth and a selective medium (SS, Selenite F), with an excellent experimental Shigella isolation record were used, along with more conventional media in multiple culture attempts.

Since Shigella can cause bacteremia, blood cultures should be encouraged in febrile patients with diarrhea. Information gained from a blood culture isolate may be of great significance in determining the treatment, and possibly the outcome, of the disease.

REFERENCES

1. Dupont HL : Shigella species (bacillary dysentery), in Mandell GL, Gordon Douglas RG Jr, Bennett JE (eds): Principles and Practice of Infectious Diseases, New York, John Willey and Sons Inc, pp 1269-1274, 1985.

2. Barret-Connor E, Connor JD : Extraintestinal manifestations of shigellosis. Am J Gastroenterol, 53:234-245, 1970.

3. Jackson HP, Kilgore DG, Jr : Purulent meningitis caused by Shigella flexneri SC Med Assoc J, 67:347-350, 1971.

4. Rubin HM, Eardley W, Nichols BL : Shigella sonnei osteomiyelitis and sickle cell anemia, AJDC, 116:83-87, 1968.

5. Struelens MJ, Patte D, Kabir I, et al : Shigella septicemia: Prevalence, presentation, risk factors and outcome. J Infect Dis, 152:784-790, 1985.

6. Scragg JN, Rubidge CJ, Appelbaum PC : Shigella infection in African and Indian children with special reference to Shigella septicemia, J Pediatr, 93:796-797, 1978.

7. Burrows W : In Textbook of Microbiology, ed, 8, Philadelphia, WB Saunders Co, 1963.

8. Graber CD, Browning D, Davis JS : Shigellemia without Shigella diarrhea. Report of a case. Am J Clin Pathol, 46:221-224, 1966.

9. Kligler RM, Hoeprich PD : Shigellemia. West J Med, 141:375-378, 1984.

10. Neter E, Merrin C, Surgalla MJ, et al : Shigella sonnei bacteremia: unusual antibody response from immunosuppresive therapy following renal transplantation. Urology, 4:198-200, 1974.

Correspondence:
Gülsen Hascelik
Hacettepe University,
Department of Microbiology,
Ankara, TURKIYE.