treated promptly. Counselling on the use of diaphragms should stress avoidance of prolonged use.

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Enterococcal endocarditis in early infancy

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Infective endocarditis is uncommon in newborns and infants. The microorganisms most frequently involved are gram-positive, but gram-negative and mixed flora may be encountered.[1-16] Although *Streptococcus faecalis* causes 10% to 15% of cases in adults[17] and some cases in older children[2] it has never before been identified as the cause in neonates or children under the age of 2 years. The only case of enterococcal endocarditis in a newborn reported in the literature was not sufficiently documented to warrant the diagnosis.[18] In our 3-month-old patient the disease was fatal.

Case report

A 3-month-old girl was readmitted to our hospital because of poor feeding, lethargy and respiratory difficulty. She had been born after 31 weeks of uncomplicated gestation; the delivery was by cesarean section because of breech presentation, and her weight was 1.13 kg. Mild respiratory distress at that time had prompted her transfer to our hospital. A catheter placed in the umbilical artery on the first day of life was removed the next day. At the age of 1 month a heart murmur had prompted electrocardiography, echocardiography and chest roentgenography; all the results were within normal limits, and the murmur was judged to be innocent. At 7 weeks she had melena due to necrotizing enterocolitis. A blood culture was positive for *S. faecalis*, which was sensitive to ampicillin and gentamicin. These antibiotics were administered for 7 days. The remainder of the hospital course was uneventful, and she was discharged at the age of 2½ months, weighing 2.21 kg. She did well in the first 2 weeks at home, but then rectal bleeding was noted. A few days later she started to feed poorly and became lethargic.

When readmitted, she was pale, lethargic and in marked respiratory distress. Her heart rate was 180 beats/min, but while the peripheral pulses were easily felt in the upper extremities they were not palpable in the lower limbs. Similarly, her blood pressure, obtained by the Doppler method, was 100 mm Hg in both arms, but it could not be obtained in the legs. The precordium was hyperdynamic; the first heart sound was normal, the second was single and loud, and a third was loud, making a gallop rhythm. A systolic murmur was heard along the left sternal border and the back, where it seemed to spill into diastole; there was also a low-pitched mid-diastolic murmur at the apex. Fine rales were heard over both lung fields. The infant's abdomen was distended, the liver reaching 4 cm below the right costal margin and the spleen 5 cm below the left. Generalized edema was present. We noted a large bruise on her left foot.

The pressure difference between her upper and lower limbs suggested an aortic obstruction, and the mid-diastolic murmur was compatible with mitral valve obstruction. Because her cardiovascular system had been normal earlier and there was now clinical evidence of sepsis and heart failure, a diagnosis of infective endocarditis involving the mitral valve and the aorta was considered.

Blood samples again yielded *S. faecalis*, and chest roentgenograms revealed cardiomegaly, pulmonary edema, entrapment of air and areas of atelectasis. Electrocardiography suggested combined atrial enlargement, while an echocardiogram (Fig. 1) showed decreased mitral valve motion, with thick, multilayered echoes. The left ventricle and left atrium were evidently enlarged, the right ventricular wall was thickened, and there was a little pericardial effusion.

Abnormal laboratory values were: hemoglobin level 4.6 g/dl, hematocrit 15.9%, leukocyte count 21.6 × 10^9/l, reticulocyte count 9.8% and serum bilirubin level 2.5 mg/dl (42.8 μmol/l). A Coombs' test gave a negative result. There were coliforms in the urine but no organisms in the cerebrospinal fluid.

We began treating the infant with oxygen, ampicillin, gentamicin, cloxacillin, transfusions of packed red cells, digoxin and diuretics, but her improvement was only transient. Bleeding at the sites of puncture and laboratory evidence of intravascular coagulopathy prompted the administration of platelets and exchange transfusions. On the third day cardiac catheterization revealed pulmonary venous desaturation, pulmonary hypertension and elevation of the right ventricular and the right and left atrial pressures; the left ventricle was not entered. Cineangiograms showed marked dilation of the left atrium and a thickened, irregular mitral valve. The descending aorta was not well visualized.

The baby's condition continued to deteriorate as seizures and renal shutdown developed. Her poor general condition precluded surgery. Cardiorespiratory arrest occurred on the fourth hospital day.

Autopsy revealed necrotizing en-

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terocolitis without perforation, as well as many fibrin thrombi in the lungs. The clinical diagnosis of infective endocarditis involving the mitral valve was confirmed: the anterior and posterior leaflets were redundant, and large nodules of friable tissue were attached to the edges. Microscopic examination of the valve showed an acute inflammatory reaction and clumps of gram-positive cocci among the fibrin and necrotic debris covering the valve. The thoracic aorta distal to the ductus arteriosus site was totally obstructed by a large embolus composed of material like that found on the mitral valve, and here, too, were clumps of gram-positive cocci. Lower nephron necrosis was seen in both kidneys.

Discussion

To date 85 cases of infective endocarditis have been described in infants less than 6 months old.[1,3-8,10-16] *Staphylococcus aureus* and *Streptococcus* sp. caused 10 cases each, *Diplococcus pneumoniae* 3 cases, group B *Streptococcus*, β-hemolytic *Streptococcus*, *Staphylococcus* sp. and *Pseudomonas* 2 cases each, *S. epidermidis*, *Bacillus pyocyaneus*, *Neisseria gonorrhoeae* and *Escherichia coli* 1 case each and mixed flora 2 cases (*Proteus mirabilis*, *Klebsiella pneumoniae* and β-hemolytic *Streptococcus* in 1, and *S. epidermidis* and *Serratia* sp. in the other); no organism was recovered in 8 cases, and in 40 the organism was not reported. In two reports on this condition in children the number of patients under 6 months of age and the etiologic agent in this subgroup were not stated.[2,9]

The clinical diagnosis of infective endocarditis in infancy is difficult, for in this age group the condition often occurs without any underlying cardiac abnormality.[2-6] Predisposing factors include umbilical catheters and central infusion lines,[6,11,13] sepsis,[2,6,8] prematurity[11,16] and, possibly, traumatic delivery.

M-mode echocardiography has been useful in revealing vegetations in adults[1,9] and infants[12,14,16] and has helped in the management of these patients. Such vegetations, however, can be too small to be detected by this technique.[19]

The prognosis of the disease in infancy and childhood has improved since the introduction of antibiotics,[15] but it continues to be guarded.[2,16] Complications are mainly due to embolization to various organs.[15] Arterial thrombosis extending to the aorta has been described,[1,13] although it did not appear to compromise the circulation as in our patient.

In conclusion, one should suspect infective endocarditis when unexplained cardiac failure develops in an infant with sepsis even in the absence of underlying heart disease. In our patient the *S. faecalis* detected in the blood at 7 weeks of age probably persisted in spite of antibiotic therapy and finally caused septicemia and endocarditis at 3 months.

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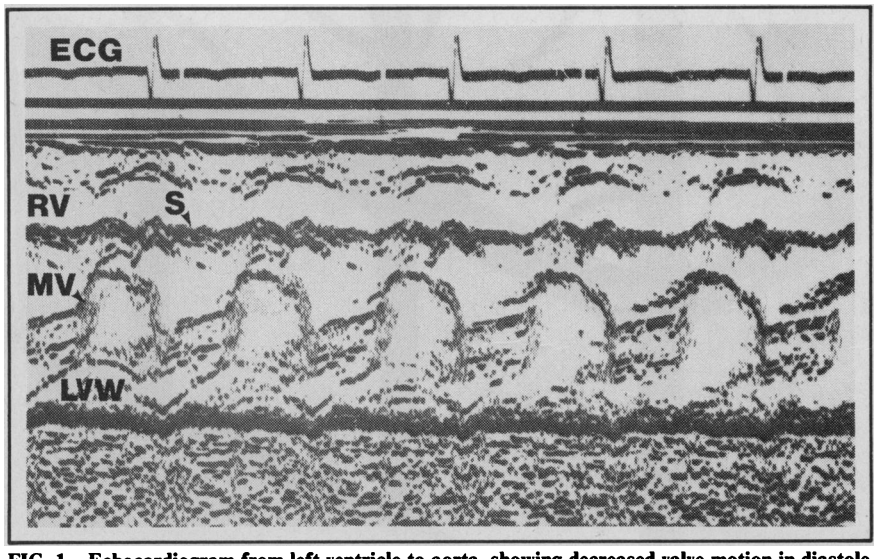

FIG. 1—Echocardiogram from left ventricle to aorta, showing decreased valve motion in diastole and thick multilayered echoes in systole, all strongly suggesting mitral valve vegetations. ECG = electrocardiogram; RV = right ventricle; S = interventricular septum; MV = mitral valve; LVW = posterior left ventricular wall.

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