Congenital Atrial Flutter

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Atrial flutter in infants is rare (Keith, Rowe, and Vlad, 1967) and was first reported by Lewis (1915) in an infant of 3 months. Carr and McClure (1931) reported atrial flutter in a newborn infant in whom the cardiac irregularity was noted occasionally before birth and in whom the heart rate and rhythm returned to normal on the 10th day after birth. Fetal tachycardia in such a case can lead to an erroneous prenatal diagnosis of fetal distress, and Blumenthal et al. (1968) have clarified the issue by documentation of the true nature of the lesion using fetal electrocardiography. The paucity of published reports suggests that this is an exceedingly rare condition and therefore worth while reporting 2 additional cases.

Case Reports

Case 1. A gravida 9 was admitted to Mill Road Maternity Hospital, Liverpool, in February 1968, with an unstable lie. The date of her last menstrual period was uncertain, but maturity was estimated at about 38 weeks. During the pregnancy she had been treated with oral iron for iron-deficiency anaemia, and at 26 weeks’ gestation she had an ‘influenza type’ illness, for which she received tetracycline. About that time also, 3 of her children had mumps. She had an x-ray for pelvimetry 8 days before admission. 8 days after admission the fetal heart was rapid and irregular, with a rate of about 170 a minute, and labour was induced by artificial rupture of the membranes, at which the liquor was noted to be clear. 2 hours later the fetal heart was recorded as greater than 200, with periods of irregularity, and she had an emergency caesarean section. The baby, a female weighing 3800 g., cried immediately at birth, and was scored 10 on the Apgar scale at 1 minute. There was no comment on the regularity of the infant’s heart. When seen 20 hours later the baby was noted to have a heart rate of 190 per minute, which was irregular, with frequent, very rapid bursts of tachycardia. The liver was enlarged to two fingers below the costal margin; there were no other abnormal findings. ECG (Fig. 1a) showed atrial flutter, with an atrial rate of 420 per minute and a ventricular rate of 240 per minute. Chest x-ray showed slight increase in the cardiac diameter. The baby was immediately digitalized, and the next day the heart rate was 130 per minute and regular. A repeat ECG on the 6th day (Fig. 1b) showed normal sinus rhythm, and digoxin was accordingly discontinued. Routine viral studies, including mumps, carried out on both mother and baby suggested that the infant’s antibodies appeared to be of maternal origin and that the mother had a recent infection with influenza A virus. When last seen on follow-up at the age of 10 weeks the baby was normal.

Case 2. The mother, gravida 3, was admitted to the Liverpool Maternity Hospital in March 1968, for induction of labour because of postmaturity. The pregnancy had been normal and she had taken promazine theobromine right through the pregnancy for nausea. At 34 weeks’ gestation she had an upper respiratory tract illness, and there had recently been cases of mumps in the area where she lived. Labour was induced by artificial rupture of the membranes, and 4 hours later the fetal heart was noted to be irregular, with a rate varying between 120 and 160 per minute. Caesarean section was considered, but as labour was progressing satisfactorily and the fetal heart, though irregular, remained about 140, it was decided to wait. She was delivered normally of a male infant weighing 4110 g., who was scored 5 on the Apgar scale at 1 minute and 10 at 10 minutes. The infant’s heart rate was noted to be irregular after delivery. The following day, he had a fixed tachycardia at about 180 per minute. There were no other abnormal findings. ECG (Fig. 2a) showed atrial flutter with an atrial rate of 400 per minute and a ventricular rate of 190 per minute. The baby was digitalized, and the rate remained irregular until 5 days later when a faint systolic murmur was detected. Repeat ECG on the 8th day (Fig. 2b) was within normal limits and showed variable nodal rhythm. Viral studies, including mumps, carried out on both mother and baby did not indicate any recent infection. The baby was discharged on digoxin, and though the systolic murmur was still present on the first follow-up visit, it had disappeared when last seen at the age of 7 weeks.

Discussion

Congenital atrial flutter indicates that the flutter is believed to have been present before birth and has been documented in the immediate neonatal period. Both cases above fulfil these criteria. In
a review of 17 cases from the literature and one case of their own, Caddell and White more (1963) reported that the atrial rate ranged from 270 to 460 per minute and that constant atrioventricular block occurred as often as variable block. 9 cases were thought to be idiopathic in origin, because the patients were normal after the arrhythmia had subsided; 5 cases were known to have some type of associated congenital heart defect, and in 4 cases, the aetiology could not be established. Nadas (1963) suggests that the association of atrial flutter with congenital heart disease is of ominous significance and that only the severest lesions, usually accompanied by congestive failure and gross atrial enlargement, manifest this arrhythmia. He also notes its association with endocardial fibroelastosis, and in the case reported by Siderides, Antonius, and Richlan (1967), necropsy revealed fibroelastosis and ectopic muscular fibres invading the sino-atrial node. The association of fibroelastosis with a positive skin test to mumps antigen (Noren, Adams, and Anderson, 1963), and the history of contact with mumps in both the cases reported, have suggested the possibility of intrauterine infection with mumps. However, viral studies in both patients revealed no evidence of infection. Clinically, in the 9 cases with idiopathic flutter discussed by Caddell and Whitemore (1963), the picture indicates cardiac involvement with mild congestive failure. Mild dyspnoea and transient systolic murmurs were heard in 3 cases, and hepatic enlargement was present in 2. In those associated with cardiac malformation, the severity of symptoms is presumably that of the underlying defect.

Digitalis appears to be the drug of choice and was successful in 75% of the cases in which it was used (Caddell and Whitemore, 1963). The infant
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reported by McLean (1952) did not respond to digitalis nor to a trial of quinidine; after a repeat month’s treatment with digitalis normal rhythm was achieved. Procaine amide has also been used, but Keith et al. (1967) suggest that most cases subside spontaneously whether treated or not. Of the total group of 18 infants with congenital flutter reported by Caddell and Whitemore (1963), normal rhythm was achieved in 10, death occurred in 6, and flutter persisted in 1. The details of the remaining case are not known. Presumably the prognosis of those cases with a congenital cardiac malformation is that of the underlying defect.

Though atrial flutter is a rare condition in both the fetus and the neonate, a wider awareness of this entity and its recognition, perhaps with the help of fetal electrocardiography, could avoid an erroneous diagnosis of fetal distress and unnecessary termination of pregnancy.

Summary

Two new cases of congenital atrial flutter are described. A brief review of the literature is presented.

I am indebted to Drs. R. McL. Todd and T. McKendrick for permission to publish details of these two cases, and to Dr. Todd for his interest and advice in preparing this work.

REFERENCES


