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CLINICAL OBSERVATIONS

Accelerated ventricular rhythm in the neonatal period: a review and two new cases in asymptomatic infants with an apparently normal heart

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Accelerated ventricular rhythm (AVR) was observed in two newborn infants. In the first case, arrhythmia was noted during the foetal period. Both neonates were asymptomatic and had no evidence of cardiac disease. The arrhythmia eventually disappeared when the infants were 4 mo and 24 d old, respectively. AVR in the neonatal period is reviewed in this report and recent information regarding appropriate diagnostic evaluation, differentiation from ventricular tachycardia and treatment is outlined.

Conclusion: Accelerated ventricular rhythm is a benign and self-limited arrhythmia in the neonatal period. However, it is important to differentiate it from other serious rhythm disorders, mainly ventricular tachycardia, in order to avoid unnecessary and potentially harmful treatment and to relieve parental anxiety.

Key words: *Accelerated ventricular rhythm, neonates, slow ventricular tachycardia*

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Accelerated ventricular rhythm (AVR) is a well-recognized arrhythmia in adults, usually associated with acute myocardial infarction, other myocardial disease or digitalis intoxication. However, it is rarely observed during the newborn period and childhood (1–4).

Accelerated ventricular rhythm has the electrocardiographic characteristics of ventricular tachycardia (VT) except for its rate, which is just slightly greater than the preceding sinus rhythm. Initial description of the condition is credited to Harris in 1950. The first paediatric series was reported 24 y ago (1).

We report two neonates with accelerated ventricular rhythm recognized during the first 24 h of life. Both neonates had structurally normal hearts. We reviewed all the reported cases of AVR in neonates emphasizing appropriate diagnostic evaluation, differentiation from ventricular tachycardia and appropriate management.

Case reports

Two full-term infant girls, delivered by caesarean section, were referred to us for evaluation of "irregular heart rate", at 1 d of age. In case 1, arrhythmia was noted by an obstetrician at 38 wk gestation using foetal echocardiography. In case 2, arrhythmia was detected at 12 h of age by electrocardiogram. Both infants were asymptomatic and cardiovascular examination disclosed no abnormalities other than the arrhythmia. The mothers had received no medication during pregnancy and there was no history of maternal drug abuse.

In each case, a 12-lead electrocardiogram during the first 24 h of life showed frequent ventricular premature contractions (VPCs), couplets and repeated episodes of accelerated ventricular rhythm with frequent fusion beats at the onset and termination of runs. These findings were verified with 24-h Holter monitoring. The sinus and ventricular rates were nearly identical in both cases: 150 to 160 beats/min and 170 to 190 beats/min, respectively (Fig. 1). The QTc and the other ECG intervals during sinus rhythm were all within normal limits. In both neonates, serum electrolytes, IgM levels, aspartate aminotransferase, alanine aminotransferase, creatine kinase, lactate dehydrogenase and pH were all within normal limits. The echocardiogram showed a structurally and functionally normal heart in both cases.

The first neonate was started on empiric treatment with propranolol, which was withdrawn 2 d later as there was no response of the arrhythmia. The second

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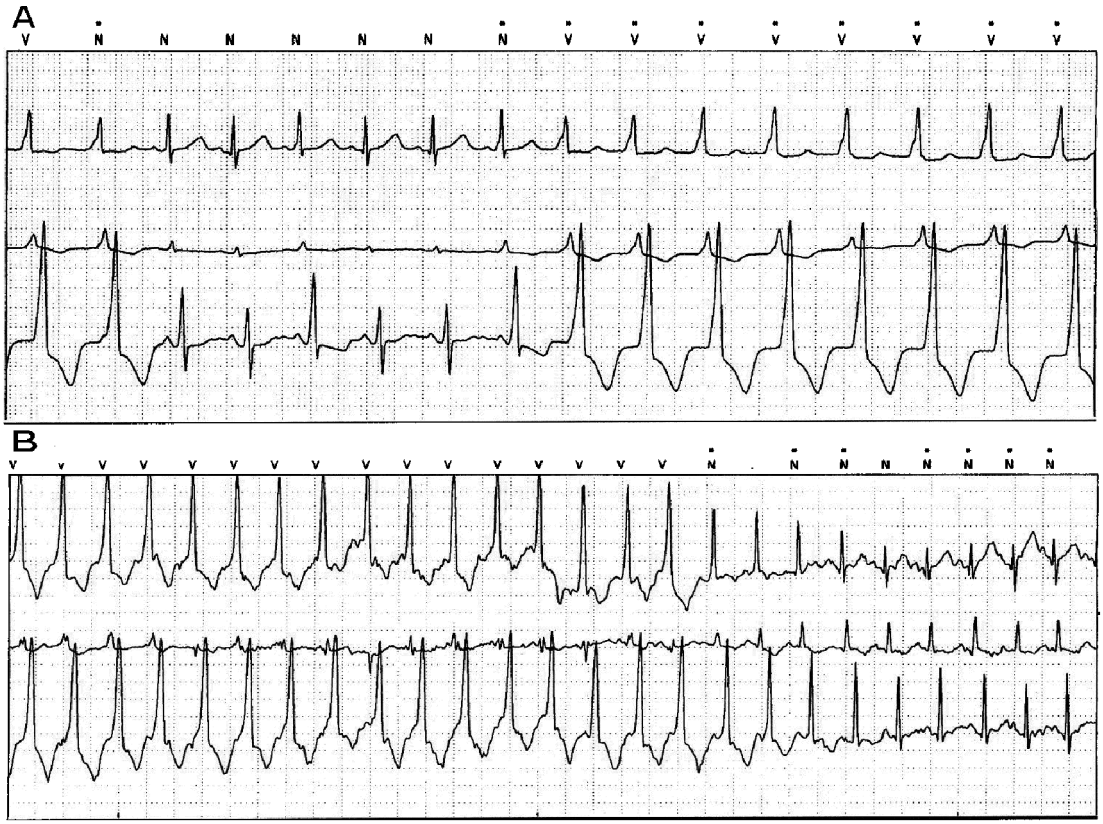

Fig. 1. Segments of initial 24-h Holter monitoring. (A) Case 1: the first beat is a fusion beat and then AVR starts at a rate similar to sinus. (B) Case 2: AVR slowly merging with sinus rhythm at a similar rate. The last 3 beats are fusion beats.

one received no therapy. Both neonates were followed with periodic 24-h Holter monitoring. At 4 mo of age in case 1 and at 24 d of age in case 2, 24-h Holter monitoring revealed neither AVR nor any other arrhythmia. The patients are now 18 and 6 mo old, respectively, and no arrhythmia has been detected by subsequent examinations.

Discussion

Accelerated ventricular rhythm is defined as three or more successive wide QRS beats at a rate close to the baseline rhythm, with a maximal difference of 10–15%. The rate is usually less than 120 beats per minute in children. In neonates who have relatively faster sinus rates, AVR rate is more rapid but usually <200 beats/min (1, 2). The QRS complex is monomorphic in all AVR runs. Because of the similarity in the two rates, capture and fusion beats are common. Furthermore, the cardiac output is not clinically compromised (1–4).

The cause of arrhythmia is unknown. An ectopic idioventricular focus, rather than a re-entry mechanism, appears to be the most likely explanation, as attempts to initiate or terminate the arrhythmia with programmed stimulation have been generally unsuccessful (1). In the immediate postnatal period, it is thought that imbalance of the autonomic nervous system, immaturity of the heart and stress during labour may be related to the development of AVR (5).

AVR's importance lies in its potential confusion with other wide-QRS arrhythmias, especially ventricular tachycardia (VT), which can be potentially dangerous. The critical difference from VT is AVR's slower rate (1). Neonates with VT deserve a careful search for causative factors, such as myocarditis, long QT syndrome, hamartomas, metabolic diseases, electrolyte disturbances and the exceedingly rare occurrence of myocardial infarction in infancy (2). Other causes of wide-QRS tachycardia include supraventricular tachycardias with aberration and atriofascicular pathways. Echocardiographic assessment of left ventricular size and function are important. In patients with normal echocardiograms, more invasive studies usually are not indicated (2, 3).

A review of the English literature on neonatal cases of AVR revealed 38 reported cases in the last 42 y (3–15), which are listed in Table 1. In all the reported cases, including the present two, neonates with AVR remained asymptomatic. Furthermore, the arrhythmia

Table 1. Cases of AVR in neonates reported in the literature.

Pt no./Ref	Age (d)/sex	Sinus rate (bpm)	AVR rate (bpm)	Age at last episode (d)	Age at last follow-up (mo)	Medication/successful	Heart disease/symptoms
1/[6] [a]	13/M [a]	145	180	13	2.5		No
2/[7]	1/F		166	75	7	Procainamide/no	No
3/[8]	4/M		142	4	4		No
4/[9]	3/M		194	3	5	Lidocaine, quinidine/yes	No
5/[9]	21/M		180	21	20		
6/[4]	1/M	140	140	3	6	Lidocaine, Propranolol/yes	No
7/[4]	1/F	160	160	1	24	No	No
8/[4]	1/M	140	140	1	6	No	No
9/[10] [b]	1/F [b]	170	188	1	4.5	No	No
10–17/[11]	1–2		120–175	2–14	1–120	Lidocaine, propranolol/no	No
18/[12]	1	150	162	1	12	No	No
19/[13]	1/M	130	175	60	36	No	Myocarditis/cyanotic episodes
20–31/[3]	1–20	125–181 (mean 154)	136–200 (mean 168,9)	1–150	1–132[c]	Digoxin/Propranolol (success in 4)	No
32 [14]	1	150	160	30	3	No	VSD, ASD/no
33[14]	7	170	170	30	2	No	Ebstein's anomaly, VSD, pulmonary atresia, PDA/cyanosis
34[14]	7			22	2	Digoxin/no	VSD/tachypnoea
35,36/[5]	1	150	160	18–45	24	No	VSD (1)/no
37,38/[15]	1–7	128–136	136–142		31–56	No	No

VSD: ventricular septal defect; ASD: atrial septal defect; PDA: patent ductus arteriosis.
[a] Associated with hyperkalemia.
[b] Associated with maternal cocaine use.
[c] Died at this age due to unrelated conditions.

was transient and disappeared between 24 h and 5 mo of age. On long-term follow-up, there was no documented recurrence. The majority of reported cases had no evidence of underlying heart disease. There are only three reported cases of infants with congenital heart disease who developed AVR. In these cases, the arrhythmia did not alter the clinical features of the congenital heart disease and also disappeared during the aforementioned period (14).

The benign nature of AVR was confirmed in all the reported cases. None of the 38 neonates had clinically adverse outcomes due to AVR, and there is no reported case of AVR degenerating into ventricular tachycardia or ventricular fibrillation. Although many anti-arrhythmic drugs have been given in attempted treatment of AVR, the arrhythmia is usually resistant to drug treatment (Table 1). Given the excellent long-term outcome and the lack of haemodynamic compromise during the arrhythmia, treatment is not indicated in this age group (1, 3, 4).

In case 1, presented here, it is noteworthy that arrhythmia was observed in the foetal period. There is only one similar case reported in the literature. It has been hypothesized that cases of AVR may have been missed during the foetal period due to the similarity of the ventricular and sinus rates (5).

In conclusion, there is well-established evidence that AVR is a benign and self-limited arrhythmia in the neonatal period. The diagnostic challenge of this condition is to differentiate it from ventricular tachycardia in order to avoid unnecessary treatment and invasive diagnostic evaluation. However, we believe that careful follow-up with occasional Holter monitoring is indicated to ascertain that the child remains free of arrhythmia.

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Cushing's syndrome in pregnancy and neonatal hypertrophic obstructive cardiomyopathy

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Cushing's syndrome is rare in pregnancy but can cause spontaneous abortion, stillbirth or premature birth. We report a case of transient hypertrophic obstructive cardiomyopathy in a newborn whose mother had hypercortisolism due to a primary adrenal lesion. There was no family history of hypertrophic obstructive cardiomyopathy. Follow-up revealed complete resolution of the cardiac abnormalities in the infant. Cushing's syndrome in the mother resolved after delivery. Although maternal hypercortisolism seldom results in symptomatic hypercortisolism in the newborn, hypertrophic obstructive cardiomyopathy can occur.

Key words: *Hypertrophic obstructive cardiomyopathy, Cushing's syndrome, pregnancy, infant, newborn*

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Pregnancy rarely occurs in women with active Cushing's syndrome, in-keeping with the high incidence of anovulation and amenorrhoea in this condition (1). Primary adrenal hypercortisolism is a major cause of Cushing's syndrome during pregnancy, whereas pituitary-dependent Cushing's syndrome predominates in non-pregnant patients (2). Cushing's syndrome induced by pregnancy and independent from adrenocorticotropic hormone (ACTH) was described recently (3, 4). Left ventricular hypertrophy can occur in adults with Cushing's syndrome (5). Furthermore, hypertrophic obstructive cardiomyopathy is a well-known side effect of glucocorticoid therapy, particularly in newborn infants exposed to multiple antenatal courses of glucocorticoids and in premature infants given dexamethasone to treat bronchopulmonary dysplasia (6–8). We report a case of transient hypertrophic cardiomyopathy in a newborn infant whose mother had clinical and biochemical hypercortisolism related to a primary adrenal lesion.

Case report

A 2500-g male infant with a gestational age of 35 wk was born to a 30-y-old Caucasian primagravida by caesarean section due to breech presentation. The mother was unaware of her pregnancy until delivery. She had a history of myotonic dystrophy (Steinert disease). Purple abdominal striae had developed during

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