Cardiology in the Young

cambridge.org/cty

Ventricular tachycardia in fetus and neonate: a single centre experience

Lisa Hornberger[1], Carolina Escudero[2] and Hilal Al Riyami[3]

[1]Pediatrics/Obstetrics Gyecology, University of Alberta, Alberta, Canada; [2]Faculty of Medicine Dentistry, University of Alberta, Alberta, Canada and [3]Pediatric Cardiology, Sultan Qaboos University, Muscat, Oman

Original Article

Cite this article: Hornberger L, Escudero C, and Al Riyami H (2025) Ventricular tachycardia in fetus and neonate: a single centre experience. *Cardiology in the Young* **35**: 1184–1188. doi: 10.1017/S1047951125100814

Received: 1 June 2024
Revised: 8 February 2025
Accepted: 4 June 2025
First published online: 30 June 2025

Keywords:
Ventricular tachycardia; Neonate; Fetus

Corresponding author:
Hilal Al Riyami; Email: hilalnasser@squ.edu.om

Abstract

Background: Ventricular tachycardia is a rare condition in the fetus and neonate with a paucity of data regarding the management and outcomes. *Methods:* We reviewed the clinical histories, associated CHD and syndromes, diagnostic investigations, management strategies, and outcomes for all fetuses and neonates with ventricular tachycardia encountered between 2005 and 2020. *Results:* Five fetal and 8 neonatal cases of ventricular tachycardia were encountered. Two (40%) fetal cases, compared to 5 (62%) neonatal cases had CHD ($p = 0.59$), and only 1 fetus had cardiomyopathy with findings suggesting restriction before ventricular tachycardia onset. The median age at ventricular tachycardia presentation was 32 (26–37) weeks for fetal and 11 (1–27) days for neonatal cases. Of the fetal cases, 2 were initially treated trans-placentally with propranolol and 2 with amiodarone. Two fetuses (40%) had ventricular tachycardia suppression prenatally that recurred during the neonatal period necessitating propranolol therapy, 2 (40%) had resolution before birth with no postnatal recurrence, and the cardiomyopathy case never achieved full control, developed hydrops and demised. Of the neonatal cases, 4 received intravenous antiarrhythmics on admission: 3 amiodarone and 1 esmolol, and 2 of these were converted to propranolol and 2 to sotalol. Three other neonates initially received propranolol, with 1 discharged on propranolol, 1 on sotalol, and 1 on mexiletine after failed propranolol and sotalol treatment. The 8th neonate was discharged on no medication. *Conclusion:* Most fetal and neonatal ventricular tachycardia is manageable with pharmacologic therapy. Given its rarity, larger studies are needed to identify optimal management strategies.

Introduction

Ventricular tachycardia is a rare condition in fetuses and neonates that can lead to significant morbidity and mortality.[1,2] Ventricular tachycardia in the neonate is defined by having three or more ventricular complexes that happen in series at a rate of at least 20% more than the regular sinus rhythm.[2] Ventricular tachycardia in the fetus accounts for about 10% of fetal tachycardia and echocardiography permits definition of the diagnosis.[7] Ventricular tachycardia in fetal life is characterised by a fetal heart rate of 170–300 bpm with either A-V dissociation or association, with the ventricular rate being faster than the atrial rate in cases of A-V dissociation.[5] Fetal echocardiography has played a major role in the diagnosis of fetal ventricular tachycardia with use of M-mode and Doppler-based methods that define the relationship between atrial and ventricular contractions to allow for arrhythmia classification.[5] The maternal condition as well as fetal circulation affects the management of ventricular tachycardia in fetal compared to neonatal life.[3–5] Fetuses may develop hydrops due to ventricular tachycardia, which has been reported in up to 41% of cases.[4] Ventricular tachycardia may occur in the presence of a structurally and functionally normal heart or can be the result of myocarditis, electrical imbalance, long QT, cardiomyopathy or cardiac tumours.[5,6]

Although several past investigations have reported the clinical outcomes associated with paediatric ventricular tachycardia,[2,3] few report outcomes associated with fetal and neonatal ventricular tachycardia, with limited data regarding management strategies in these groups. In the present study, we sought to review the diagnosis, management, and clinical outcomes of fetal and neonatal ventricular tachycardia encountered in our centre.

Methods

This was a retrospective cohort study of all fetuses and neonates with ventricular tachycardia encountered at the Stollery Children's Hospital between January 2005 and December of 2020. After receiving approval from the University of Alberta Health Research Ethics Board, we reviewed our University of Alberta Fetal & Neonatal Cardiology database to identify all cases of fetal ventricular tachycardia diagnosed prenatally. The Alberta Health System's electronic medical records were also reviewed to identify all neonates with a coded diagnosis of ventricular

Table 1. Fetal and neonatal VT presentation, management and outcome

	Neonate	Fetal
Age at VT presentation Median (range)	11 days (1–27)	32 weeks (26–37)
Age at last VT episode Median (range)	22 days (2–90)	6 days (3–10)
Duration of postnatal treatment Median (range)	7 months (2–12)	10 months (8–12)
Length of initial hospital stay Median (range)	43 days (5–120)	12 days (3–22)
Length of follow-up Median (range)	33 months (7–36)	19 months* (12–36)
Structural heart/ Myocardial disease	5/8 patients 2 with VSD, 1 with ASD, 1 with aortic coarctation and infantile HCM, 1 with D-TGA	2/5 patients One with cardiomyopathy and 1 with aortic coarctation

For fetal age, this represents gestational age. VT = ventricular tachycardia, VSD = ventricular septal defect, ASD = atrial septal defect, HCM = hypertrophic cardiomyopathy, D-TGA = D-transposition of the great arteries *1 with fetal demise.

tachycardia through 28 days of postnatal age. We excluded cases with incomplete follow-up and those in whom ventricular tachycardia only occurred intraoperatively or postoperatively.

Data collection included clinical variables at the time of diagnosis (age, presenting symptoms, associated syndromes, presence of CHD, presence of other medical diseases, family history of CHD, or arrhythmia), the results of diagnostic investigations (ECG, echocardiogram, and Holter), management, and clinical outcomes (length of hospital stay, duration of anti-arrhythmic therapy, age at last ventricular tachycardia recurrence, and mortality). Patients were divided into fetal and neonatal groups based on the age of initial presentation. We diagnosed ventricular tachycardia in the neonatal period if four or more consecutive wide QRS complexes with atrioventricular dissociation were observed with a rate 20% greater than that of the preceding sinus rhythm, whereas in fetal cases it was defined as a fetal heart rate of 170–300 bpm with A-V dissociation with a faster ventricular rate. Fetal or neonatal ventricular tachycardia could occur with A-V association and was diagnosed when the cardiologist had determined the arrhythmia to be ventricular tachycardia once other causes of wide complex tachycardia were ruled out.

Statistical analysis

Data were collected using REDCap (Research Electronic Data Capture) hosted by the University of Alberta. Baseline characteristics for the fetal and neonatal groups are summarised using percentages for proportions and the median (range) for continuous variables. Statistical analysis was performed using Chi-square or Fisher exact analysis for differences in proportions between groups. Statistical significance was defined as $p \leq 0.05$.

Results

A total of 5 fetal and 8 neonatal cases were diagnosed with ventricular tachycardia from 2005 to 2020. Two (40%) of fetal and 5 (62%) of neonatal cases had structural heart disease ($p = 0.59$). The types of structural heart disease are provided in Table 1. Only 1 fetal case had a cardiomyopathy with findings suggesting restrictive myocardial physiology before the onset of ventricular tachycardia. Comparable functional findings were observed in a previous pregnancy. In all other fetal and all neonatal cases, tachycardia was the presenting feature. None presented with hydrops (fetal) or symptoms of heart failure (neonatal). Three fetal cases had normal left ventricular (Lfunction, while two had moderate systolic dysfunction with shortening fractions of 22 and 23%.

Median age at diagnosis among fetal cases was 34 weeks (range 26–37 weeks, interquartile range 28–26 weeks) gestation. Among neonatal cases, most were diagnosed in the first two weeks with a median age at diagnosis of 11 days (range 1–27 days, interquartile range 3–19 days). All neonatal cases who had normal cardiac function by Echocardiography.

Treatment of ventricular tachycardia

Four fetal cases were treated transplacentally with pharmacotherapy, and the fifth fetus had close surveillance. In 2 fetal cases, propranolol was used and in 2 others amiodarone was used (Figure 1). One of the fetuses initially treated with amiodarone required the addition of flecainide which was continued postnatally. Two fetuses had ventricular tachycardia resolution before birth with no postnatal recurrence, 2 had suppressed ventricular tachycardia prenatally that recurred after birth necessitating propranolol therapy in the neonatal period, and 1 (with cardiomyopathy) never achieved full control and evolved hydrops (Figure 1).

All the neonatal cases were diagnosed during the neonatal period with none having prenatal findings suggestive of ventricular tachycardia. The ventricular tachycardia rate ranged from 170–300 bpm, and all 8 had intermittent ventricular tachycardia. The ventricular tachycardia had a right bundle branch block morphology in 2 and left bundle branch block in 4 patients. The axis of the ventricular tachycardia was inferior in 3/4 with LBBB morphology, and superior in the remainder with RBBB or left bundle branch block morphology (Figure 2).

One neonate was briefly treated with sotalol but never reoccurred, whereas, the 7 others had recurrence necessitating therapy. Of the 7 neonates who were treated for ventricular tachycardia, 4 received intravenous therapy including amiodarone in 3 and esmolol in 1 (Figure 3). Of those initially receiving IV therapy, 2 were converted to oral propranolol and 2 to oral sotalol with successful therapy to discharge. Three others initially received propranolol achieving successful treatment for 1, but requiring a change in medication to sotalol in 1 and to mexiletine in the other after failed treatment with sotalol as well (Figure 3).

Ventricular tachycardia was suppressed or resolved with pharmacologic treatment in 3 of 4 (75%) of fetal cases and in 6 of 8 (75%) of neonatal cases at <1 month after birth. Postnatal ventricular tachycardia treatment lasted for a median of 10

https://doi.org/10.1017/S1047951125100814 Published online by Cambridge University Press

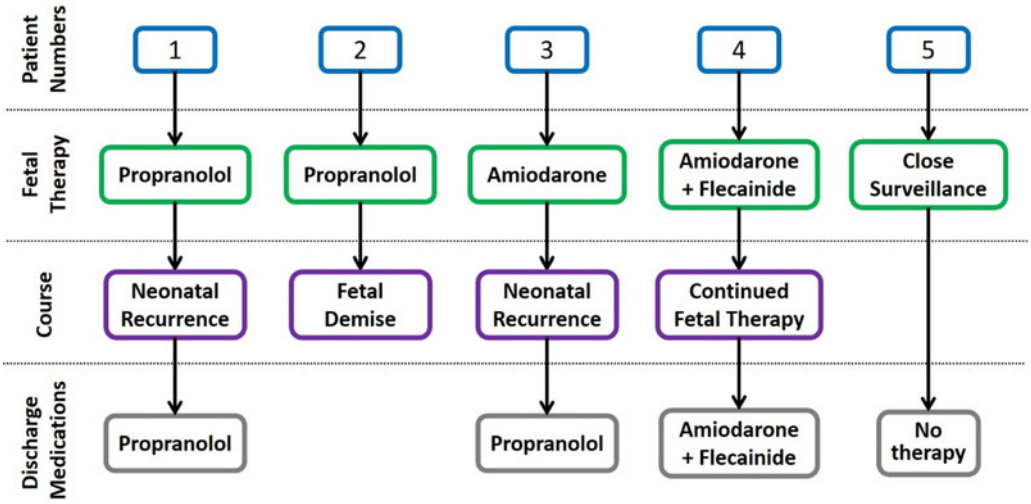

Figure 1. Management of fetal VT.

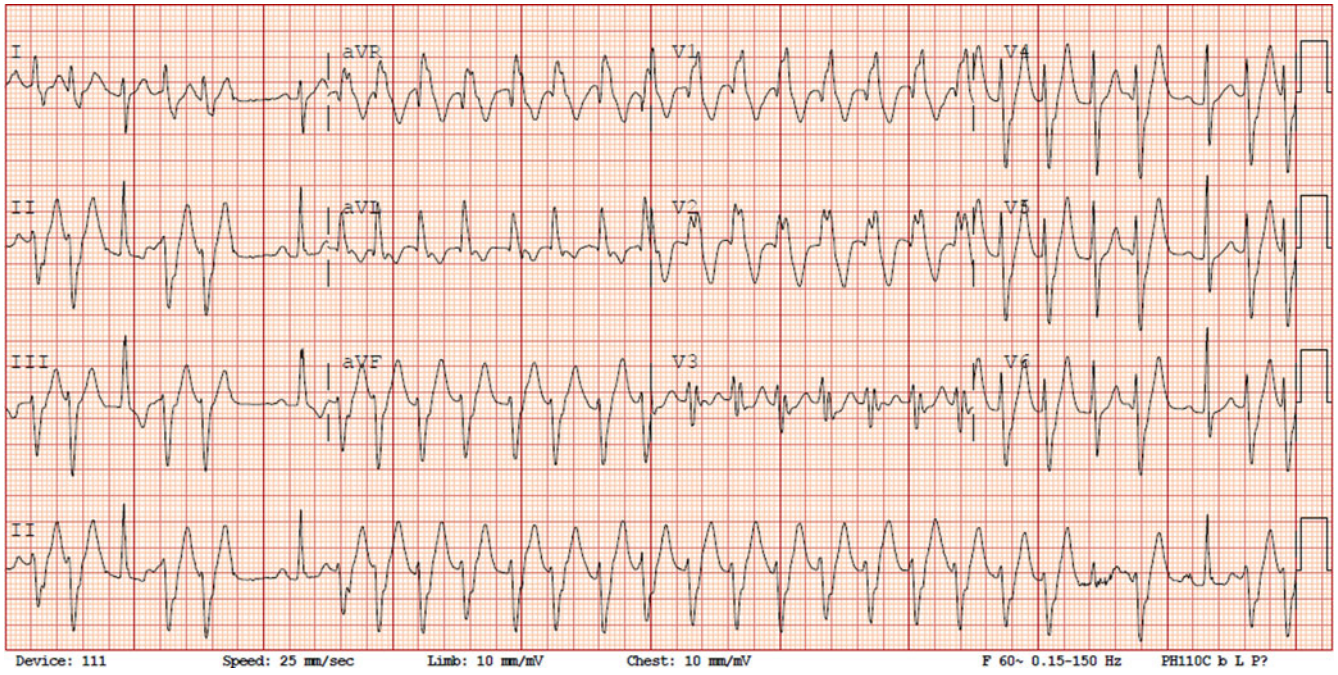

Figure 2. Neonatal VT bouts of non-sustained monomorphic ventricular tachycardia (RBBB and superior axis morphology).

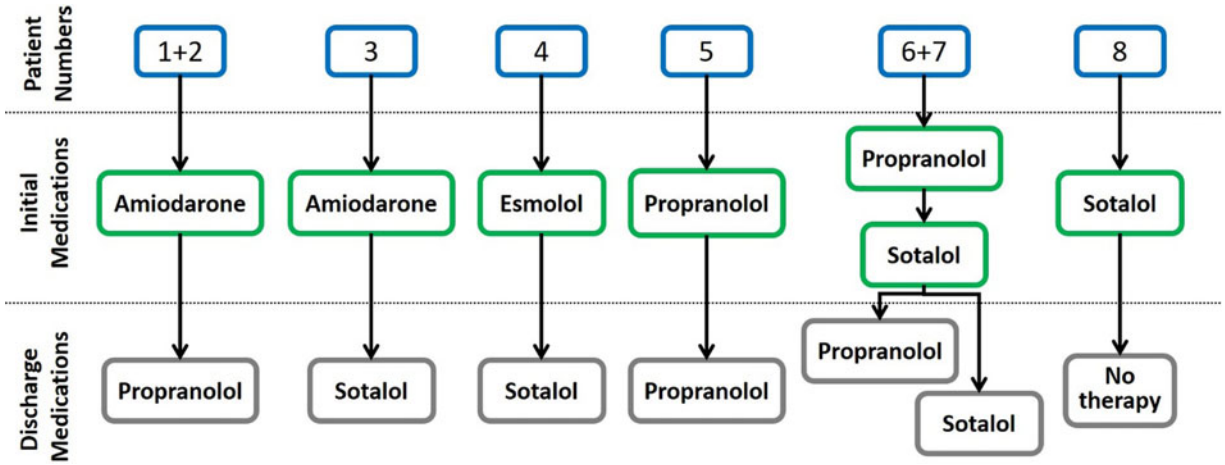

Figure 3. Management of neonatal VT.

https://doi.org/10.1017/S1047951125100814 Published online by Cambridge University Press

(range 8–12) and 7 (range 2–12) months for fetal and neonatal cases, respectively (Table 1). Demise occurred in only 1 case, which was a fetus with restrictive cardiomyopathy and progressive hydrops.

Discussion

Fetal and neonatal ventricular tachycardia is a rare diagnosis that can range in severity from benign to life-threatening.[2–4] The management varies based on the severity of symptoms, age of presentation, and haemodynamic status.[10] In our study, the clinical outcomes of both fetal and neonatal ventricular tachycardia were largely good with only a single fetal case developing hydrops related to evolving restrictive cardiomyopathy.

Ventricular tachycardia in fetuses and neonates has been reported in the presence of viral myocarditis, cardiomyopathies, cardiac tumours, anti-Ro antibodies, channelopathies, and abnormal electrolytes.[3,4,13,24] In our study, 40% of fetuses and 62% of neonates had coexistent structural heart disease and this includes one fetal case of cardiomyopathy which may have been a contributing factor to ventricular tachycardia development.[6–8]

Fetal ventricular tachycardia may become life-threatening if there is evolution of fetal hydrops.[3–5] However, proper management may reduce fetal death to less than 10%.[5] In this study, the one fetus that died had evidence of restrictive cardiomyopathy, which is known to carry a poor prognosis.[1] Neonates with ventricular tachycardia may present with poor cardiac function and in rare cases this might lead to demise.[7,8] As suggested by the present case series, the majority of neonatal ventricular tachycardia, however, is benign and if accurately diagnosed and managed, the outcome may be quite favourable.[1,8]

While neonatal ventricular tachycardia relies on well-defined electrophysiologic features on ECG, ventricular tachycardia in the fetus is indirectly inferred when there is tachycardia with faster ventricular rate relative to atrial contractions, or A-V dissociation. Occasionally, fetal ventricular tachycardia may be associated with A-V association, but differentiating the latter from supraventricular tachycardia with 1:1 ventricular-atrial or atrial-ventricular contractions may be difficult. Furthermore, differentiating fetal ventricular tachycardia from junctional tachycardia can be very challenging and should be confirmed after birth (Figure 4). The presence of intermittent ventricular ectopy can be informative in this context and lead to the suspicion of ventricular tachycardia.[5,10] Suspected fetal ventricular tachycardia should prompt exclusion of structural or functional fetal heart disease and acquisition of 12-lead ECGs in both the mother and father of the fetus given the possibility of a channelopathy, which may help tailor treatment and ensure ongoing surveillance and treatment of the affected parent.[5]

In fetal ventricular tachycardia, use of transplacental antiarrhythmic medication has been recommended in cases with recurrent and prolonged tachycardia or in the presence of hemodynamic compromise.[4,5,10] However, close surveillance is mandated for both treated and untreated fetuses.[10]

In the current cohort, we were able to manage 4 of the 5 fetal cases with no need for early delivery. One fetus did not have rhythm normalisation until flecainide was added to the initial amiodarone therapy. Jaeggi et al.[3] suggested that beta-blockers is the mainstay to treat fetal ventricular tachycardia; however, as propranolol has poor transplacental transfer, if not clearly reducing the ventricular tachycardia, moving to other medications, such as amiodarone, sotalol, and flecainide, after excluding long QT syndrome, may be necessary to achieve control. Further

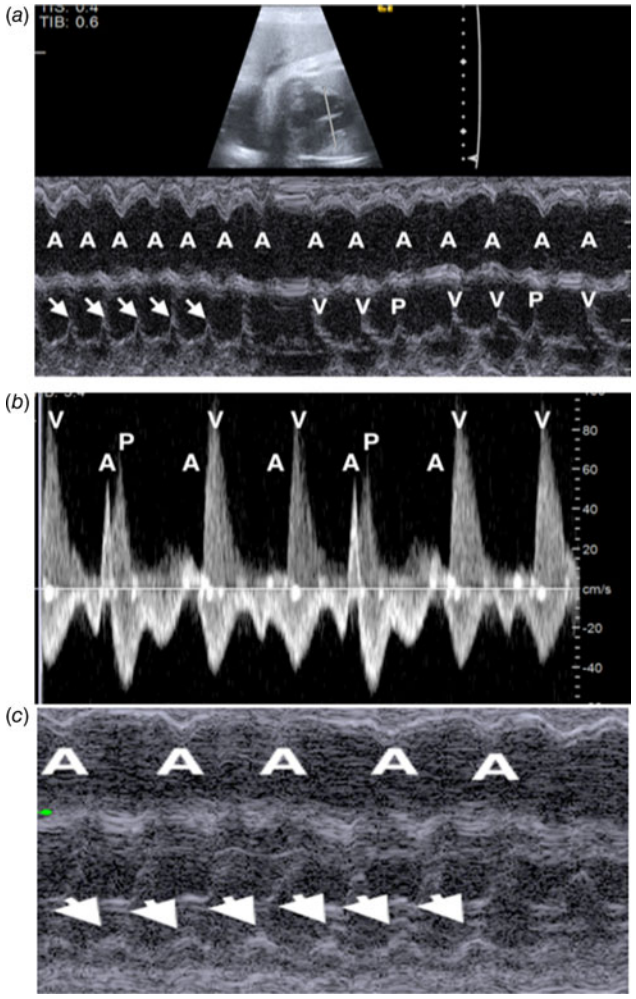

Figure 4. Differntiating VT from JET A: This 34-week fetus presented in tachycardia initially with 1:1 ventricular (arrow) to atrial (A) relationship at 180-190 bpm. When not in tachycardia, the baby was having ventricular trigeminy (P) and otherwise normal A-ventricular (V) synchrony. B: The ventricular ectopy was confirmed by the simultaneous superior vena cava (SVC)-ascending aortic Doppler interrogation with SVC a wave reversal and forward flow through the aorta are shown above the baseline and forward flow through SVC below the baseline. Normal atrial (A) and ventricular (V) contractions are shown, but with a premature ventricular contraction (P) occurring during an atrial contraction, the AV has a very high velocity in keeping with a cannon A wave (atrial contraction against a closed tricuspid valve). C: In addition to the presence of premature ventricular contractions out of tachycardia, the fetus also demonstrated ventricular (arrow)-atrial (A) dissociation at one point in tachycardia with a clearly faster ventricular rate.

assessment is needed in these cases to role out long QT by getting detailed family history and ECG for the parents and genetic testing if needed.[32]

In the neonatal period, we initially utilised intravenous medications in half and oral in the other half of our patients. The choice of antiarrhythmic was based on the stability of the neonates and the suitability of using oral or intravenous medication. Wang et al.[9] reported beta-blockers as first-line treatment for paediatric ventricular tachycardia with good outcomes, and others have reported the successful use of slow infusion amiodarone as a second-line treatment in this age group.[11,24,29] For all fetal and neonatal cases in the current report, successful therapy was achieved prior to discharge. Levin et al.[11] reported that infants with ventricular tachycardia have promising outcomes, with follow-up needed for most of the cases up to

https://doi.org/10.1017/S1047951125100814 Published online by Cambridge University Press

3 years. Similarly, our study has found that all neonatal ventricular tachycardia resolved within the first year with a median follow-up of 33 months with no recurrence up to three years from the last episodes (Table 1).

Limitations

This study was retrospective resulting in variable treatment strategies. Treatment was guided by practitioner preferences and patient characteristics. It is possible that some of the fetal and neonatal cases would have had a favourable outcome without therapy. The number of cases encountered was also limited, but represents a single-centre experience of this rare diagnosis.

Conclusion

The majority of fetal and neonatal ventricular tachycardia may be manageable with pharmacologic therapy with promising outcomes in the absence of cardiomyopathy. Given its rarity, larger multi-centre studies are needed to identify optimal management strategies for this age group to further understand the incidence of associated cardiac pathology and clinical outcomes.

Competing interests. The authors have no disclosures. No conflicts of interest have been reported by the authors.

References

1. Chiu SN, Wu WL, Lu CW, et al. Primary ventricular tachycardia in the pediatric population in a tertiary center. Arch Dis Child 2017; 102: 1137–1142.
2. De Rosa G, Butera G, Chessa M, et al. Outcome ofnewborns with asymptomatic monomorphic ventricular arrhythmia. Arch Dis Child Fetal Neonatal Ed 2006; 91: F419–22.
3. Jaeggi E, Ohman A. Fetal and neonatal arrhythmias. Clin Perinatol 2016; 43: 99–112.
4. Strasburger JF, Wakai RT. Fetal cardiac arrhythmia detection and in utero therapy. Nat Rev Cardiol 2010; 7: 277–290.
5. Simpson J. Fetal arrhythmias. In: Allan L, Hornberger LK, Sharland G (eds.), Textbook of Fetal Cardiology. Greenwich Medical Media, London, 2000, pp. 423–437.
6. Kilic E, Celiker A, Karagoz T, et al. Analysis of idiopathic ventricular tachycardia in childhood. Turk J Pediatr 2012; 54: 269–272.
7. Batra AS, Balaji S. Fetal arrhythmias: diagnosis and management. Indian Pacing Electrophysiol J 2019; 19: 104–109.
8. Iwamoto M. Idiopathic ventricular tachycardia in children. Circ J 2011; 75: 544–545.
9. Isik DU, Celik IH, Kavurt S, et al. A case series of neonatal arrhythmias. J Matern Fetal Neonatal Med 2016; 29: 1344–1347.
10. Wang S, Zhu W, Hamilton RM, et al. Diagnosis-specific characteristics of ventricular tachycardia in children with structurally normal hearts. Heart Rhythm 2010; 7: 1725–1731.
11. Lulic Jurjevic R, Podnar T, Vesel S. Diagnosis, clinical features, management, and post-natal follow-up of fetal tachycardias. Cardiol Young 2009; 19: 486–493.
12. Levin MD, Stephens P, Tanel RE Ventricular tachycardia in infants with structurally normal hearts: a benign disorder. Cardiol Young 2010; 20: 641–647.
13. Karpawich PP, Pettersen MD, Gupta P, et al. Infants and children with tachycardia: natural history and drug administration. Curr Pharm Des 2008; 14: 743–752.
14. Turner CJ, Wren C. The epidemiology of arrhythmia in infants: a population-based study. J Paediatr Child Health 2013; 49: 278–281.
15. Roggen A, Pavlovic M, Pfammatter JP. Frequency of spontaneous ventricular tachycardia in a pediatric population. Am J Cardiol 2008; 101: 852–854.
16. Kothari DS, Skinner JR. Neonatal tachycardias: an update. Arch Dis Child Fetal Neonatal Ed 2006; 91: F136–F144.
17. Poddar B, Basu S, Parmar VR. Neonatal arrhythmias. Indian J Pediatr 2006; 73: 131–134.
18. Boldt T, Eronen M, Andersson S. Long-term outcome in fetuses with cardiac arrhythmias. Obstet Gynecol 2003; 102: 1372–1379.
19. Dattilio L, Suddaby B. Ventricular tachycardia in a neonate. Pediatr Nurs 2002; 28: 612–613.
20. Pfammatter JP, Bauersfeld U. Idiopathic ventricular tachycardias in infants and children. Card Electrophysiol Rev 2002; 6: 88–92.
21. Hanisch D. Pediatric arrhythmias. J Pediatr Nurs 2001; 16: 351–362.
22. Kinoshita S, Kurosawa Y, Katoh T. Ventricular tachycardia induced by supraventricular extrasystoles. Pediatr Cardiol 2001; 22: 250–252.
23. Cuneo BF, Strasburger JF. Management strategy for fetal tachycardia. Obstet Gynecol 2000; 96: 575–581.
24. Sacchetti A, Moyer V, Baricella R, et al. Primary cardiac arrhythmias in children. Pediatr Emerg Care 1999; 15: 95–98.
25. Perry JC. Ventricular tachycardia in neonates. Pacing Clin Electrophysiol 1997; 20: 2061–2064.
26. Davis AM, Gow RM, McCrindle BW, et al. Clinical spectrum, therapeutic management, and follow-up of ventricular tachycardia in infants and young children. Am Heart J 1996; 131: 186–191.
27. Lopes LM, Cha SC, Scanavacca MI, et al. Fetal idiopathic ventricular tachycardia with nonimmune hydrops: benign course. Pediatr Cardiol 1996; 17: 192–193.
28. Ban JE. Neonatal arrhythmias: diagnosis, treatment, and clinical outcome. Korean J Pediatr 2017; 60: 344–352.
29. Dai Y, Yin R, Yang L, et al. Clinical and genetic spectrum of neonatal arrhythmia in a NICU. Transl Pediatr 2021; 10: 2432–2438.
30. Vaksmann G, Lucidarme S, Henriet E. Fetal ventricular tachycardia: beta-blockers should be the first-line treatment. J Gynecol Obstet Hum Reprod 2021; 50: 101946.
31. De Rosa G, Butera G, Chessa M, et al. Outcome of newborns with asymptomatic monomorphic ventricular arrhythmia. Arch Dis Child Fetal Neonatal Ed 2006; 91: F419–F422.
32. Batra AS, Silka MJ, Borquez A, et al. Pharmacological Management of Cardiac Arrhythmias in the Fetal and Neonatal Periods: A Scientific Statement From the American Heart Association: Endorsed by the Pediatric & Congenital Electrophysiology Society (PACES). Circulation 2024; 149: e937–e95.

https://doi.org/10.1017/S1047951125100814 Published online by Cambridge University Press