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Commentary

High-Dose Carvedilol as a Potential Key Drug for Arrhythmia in Histiocytoid Cardiomyopathy

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DESCRIPTION

Histiocytoid cardiomyopathy is a rare serious disease of infancy characterized by the development of variety of lethal ventricular arrhythmias [1-3]. In our case, the ventricular arrhythmias were managed with various antiarrhythmic drugs, including multiple beta blockers, and cardiac sympathetic nerve blockade, but it was very difficult to control her arrhythmias due to the arousal of the patient. Continued deep sedation, the only effective method, makes it impossible to maintain the life that should be lived as an infant.

The importance of sympathetic nerve activity suppression in infantile arrhythmias

Non-pharmacologic therapies such as device therapy including anti-tachycardia pacing and catheter ablation have become increasingly available for the treatment of such refractory arrhythmias. However, the mechanisms of arrhythmias in infants and young children are abnormal automaticity and are strongly influenced by sympathetic nervous activity such as emotion and excitement as modulating factors, making it difficult for them to benefit from non-pharmacological treatment. Neonatal atrial tachycardia or Atrio-Ventricular Reciprocating Tachycardia (AVRT), which develops in the fetal period, is a good example. In the case of atrial tachycardia, induction of anesthesia for ablation will stop the tachycardia. Ultimately, the effectiveness of beta blocker is keenly felt. In neonatal AVRT, the efficacy of flecainide has been clinically demonstrated, and the combination of beta blocker or sotalol was reported to be effective when Flecainide alone is not sufficient to control [4]. It can be understood that flecainide suppresses the conduction of the accessory pathway as a substrate, and beta blocker or sotalol suppresses sympathetic nerve activity as a modulating factor.

Suppression of the Store-Overload-Induced Ca^{2+} Release (SOICR)

There are a variety of conditions in which sympathetic activity as a modulating factor of an arrhythmia in infancy makes arrhythmia management difficult, and long QT syndrome and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) are frequently experienced. Inspired by the inhibitory effect of Carvedilol on arrhythmogenic store overload-induced Ca^{2+} release for the patients with CPVT reported in 2011 by Zhou, et al., [5], we administrated high-dose Carvedilol. Carvedilol is the only beta blocker that suppresses Store-Overload-Induced Ca^{2+} Release (SOICR). Although considered effective in CPVT, its beta blocking effect is weak [6], and bisoprolol and nadolol are expected to be selected and used in combination with flecainide to suppress SOICR. The SOICR is a cause of triggered activity, therefore we attempted to administer verapamil for VT in this case, but as with other beta blockers, the clinical response was poor. In addition, the side effect of verapamil is its inhibition of cardiac contraction [7], which is problematic in infants. Although further case series and some functional analysis are needed to validate this effectiveness of carvedilol, the results of freedom from prolonged sedation and successful control of fatal ventricular arrhythmias were clinically very satisfying.

CONCLUSION

Management of refractory arrhythmias in infancy is very difficult due to their altered emotional state and high sensitivity to sympathetic nervous activity. Application of non-pharmacologic therapies is difficult due to their body size and the suppression of modulating factors by anesthesia during the procedure. We believe that the clinical efficacy of our findings in this case, applied to other diseases similarly strongly affected by sympathetic nervous activity, will be of great help in the treatment of the same diseases in the future.

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REFERENCES

1. Malhotra V, Ferrans VJ, Virmani R. Infantile histiocytoid cardiomyopathy: Three cases and literature review. Am Heart J. 1994;128(5):1009-1021.
2. Finsterer J. Histiocytoid cardiomyopathy: A mitochondrial disorder. Clin Cardiol. 2008;31(5):225-227.
3. Xie H, Chen X, Chen N, Zhou Q. Sudden death in a male infant due to histiocytoid cardiomyopathy: An autopsy case and review of the literature. Am J Forensic Med Pathol. 2017;38(1):32-34.
4. Price JF, Kertesz NJ, Snyder CS, Friedman RA, Fenrich AL. Flecainide and sotalol: A new combination therapy for refractory supraventricular tachycardia in children<1 year of age. J Am Coll Cardiol. 2002;39(3):517-520.
5. Zhou Q, Xiao J, Jiang D, Wang R, Vembaiyan K, Wang A, et al. Carvedilol and its new analogs suppress arrhythmogenic store overload induced Ca^{2+} release. Nat Med. 2011;17(8):1003-1009.
6. Sumitomo N. Current topics in catecholaminergic polymorphic ventricular tachycardia. J Arrhythm. 2016;32(5):344-351.
7. Kirk CR, Gibbs JL, Thomas R, Radley-Smith R, Qureshi SA. Cardiovascular collapse after verapamil in supraventricular tachycardia. Arch Dis Child. 1987;62(12):1265-1266.