

Short reports

Cardiovascular collapse after verapamil in supraventricular tachycardia

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SUMMARY Five babies who presented with supraventricular tachycardia were treated with verapamil intravenously. All developed severe hypotension and two died. Verapamil should not be used in the initial management of supraventricular tachycardia in neonates.

Verapamil is commonly used to treat supraventricular tachycardia in children.¹ Adverse effects of the drug in infants have been reported² but the dangers are still not widely appreciated.

Case reports

Four boys and a girl with a mean age of 2.6 weeks (range 2-4 weeks) were all born at term after uneventful pregnancies and deliveries. Their mean birth weight was 3170 g (range 2810-3830 g), and they had no neonatal problems until presentation.

CASE 1

A baby girl developed a supraventricular tachycardia of 300 beats/minute with cardiac failure. She was given verapamil 0.3 mg/kg intravenously and five minutes later reverted to sinus rhythm. One hour later she collapsed with a severe bradycardia and despite vigorous resuscitation including administration of calcium gluconate, adrenaline, and dopamine she died. At necropsy the heart was normal.

CASE 2

A baby boy developed cardiac failure secondary to a supraventricular tachycardia of 300 beats/minute. This was reversed by verapamil 0.3 mg/kg given intravenously. A bradycardia immediately developed in association with metabolic acidosis, which responded well to frusemide and sodium bicarbonate given intravenously. A further episode of supraventricular tachycardia was successfully treated with

digoxin. He subsequently made a full recovery. An electrocardiogram showed no evidence of pre-excitation.

CASE 3

A baby boy presented with cardiac failure and a supraventricular tachycardia of 300 beats/minute. He was given verapamil 0.1 mg/kg intravenously. A few minutes later he became hypotensive and developed a profound bradycardia. Atropine was given intravenously resulting in reversion to a supraventricular tachycardia of 330 beats/minute. An echocardiogram showed a dilated, poorly contracting left ventricle. The tachycardia persisted even after treatment with digoxin. Five hours after the dose of verapamil propranolol 15 µg/kg was given intravenously and the dose was repeated when he did not respond. He collapsed and developed a profound bradycardia five minutes later, but was successfully resuscitated with calcium gluconate and dopamine given intravenously. After twelve hours the supraventricular tachycardia recurred and was only temporarily terminated with synchronised direct current cardioversion. Permanent conversion to sinus rhythm was achieved with flecainide given intravenously, and he was maintained on oral flecainide. He made a complete recovery and an electrocardiogram was normal.

CASE 4

A baby boy was admitted with a supraventricular tachycardia of 280 beats/minute and cardiac failure. He received verapamil 0.2 mg/kg intravenously and this dose was repeated after 15 minutes. Despite this the tachycardia persisted, and digoxin 6 µg/kg was given intramuscularly 30 minutes later. Within 10 minutes he reverted to sinus rhythm with no evidence of pre-excitation. Two hours later he suddenly became severely hypotensive and required assisted ventilation and plasma, sodium bicarbonate, and frusemide intravenously. The blood pressure

returned to normal within six hours and he subsequently recovered.

CASE 5

A baby boy presented with cardiac failure in association with a supraventricular tachycardia of 300 beats/minute. Frusemide was given intravenously and digoxin 15 µg/kg orally, with restoration of sinus rhythm. On admission to the regional unit he was still in cardiac failure. The electrocardiogram showed signs of Wolff-Parkinson-White syndrome. Shortly afterwards he reverted to a supraventricular tachycardia of 300 beats/minute. He received 10% calcium gluconate 0.3 ml/kg before the administration of verapamil 0.1 mg/kg intravenously without effect, so the dose of verapamil was repeated an hour later. Echocardiography showed a slightly dilated but well contracting left ventricle. The tachycardia persisted and therefore after a further two hours he was given propranolol 1 mg/kg orally. Half an hour later he became hypotensive with a severe bradycardia and failed to respond to resuscitation that included calcium gluconate, adrenaline, and isoprenaline given intravenously. At necropsy an anomalous Kent bundle was found.

Discussion

The treatment of supraventricular tachycardia can be difficult because a persistent tachycardia may result in depression of myocardial function. With the exception of digoxin, most antiarrhythmics depress myocardial function and cause a further reduction in cardiac output.

Verapamil given intravenously has been advocated as the best drug for the treatment of supraventricular tachycardia.¹ Its side effects, however, (including hypotension and pronounced bradycardia) are well known,³ and have been reported in infants.² Our experience includes two babies who died in the neonatal period and three who made stormy recoveries after developing severe hypotension. Two of these babies received only a small dose of verapamil (0.1 mg/kg).

Calcium gluconate is recommended for the treatment of the haemodynamic complications of verapamil administration.³ The response to calcium gluconate treatment, however, varies and it is not always successful.⁴ Calcium chloride may be more

effective than calcium gluconate and this should be available when verapamil is given.⁵

The combination of verapamil and propranolol has unpredictable effects on the myocardium. Studies in adults have shown that it is safe if the left ventricle is contracting well but its use in those with impaired contractility is not recommended.⁶ These drugs have been used sequentially in the management of resistant supraventricular tachycardia in childhood. Our experience in case 3 shows that even when the interval between their administration is greater than the half life of verapamil, severe myocardial depression can still occur. Case 5 also illustrates that this combination can have disastrous consequences even if there is no impairment of myocardial contractility.

We recommend that verapamil should not be used in the initial treatment of supraventricular tachycardia in neonates and emphasise the dangers of the combination of propranolol and verapamil even if the myocardium is normal and there is a prolonged interval between doses. Synchronised or unsynchronised direct current cardioversion is perhaps the safest method of restoring sinus rhythm, which can then be maintained by digoxin.

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References

- 1 Wilkinson JL. Management of paroxysmal tachycardia. *Arch Dis Child* 1983;**58**:945-6.
- 2 Epstein ML, Kiel EA, Victorica BE. Cardiac decompensation following verapamil therapy in infants with supraventricular tachycardia. *Pediatrics* 1985;**75**:737-40.
- 3 Association of the British Pharmaceutical Industry. *ABPI data sheet compendium*. London: Datapharm Publications Ltd, 1986-7:1-2.
- 4 Orr GM, Bodansky HJ, Dymond DS, Taylor M. Fatal verapamil overdose. *Lancet* 1982;**ii**:1218-9.
- 5 Worthley LIG. Treating adverse effects of verapamil. *JAMA* 1984;**252**:1129.
- 6 Kieval J, Kirsten EB, Kessler KM, Mallon S, Myerburg RJ. The effects of intravenous verapamil on hemodynamic status of patients with coronary artery disease receiving propranolol. *Circulation* 1982;**65**:653-9.

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