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Unexplained Systemic Hypertension After Closure of Ductus Arteriosus

Piroze Davierwala, MCh, Nityanand Thakur, MS, Packriswamy Babu, MS, Sreedhar Reddy, MS, Pawan Kumar, MS, Raj Menon, MCh, Anil G Tendolkar, MS

Department of Cardiovascular and Thoracic Surgery
Lokmanya Tilak Municipal Medical College and General Hospital
Mumbai, Maharashtra, India



ABSTRACT

Immediately after surgical closure of a patent ductus arteriosus, a 12-year-old boy developed severe systemic arterial hypertension refractory to medication. The cause of hypertension could not be found, but it came under control 3 weeks postoperatively with a combination of angiotensin-converting enzyme inhibitor and chlorothiazide.

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INTRODUCTION

Postoperative hypertension following surgical closure of a patent ductus arteriosus is very rare. Only a few instances have been reported.^{1–3} A case of severe hypertension arising after division and suturing of the ductus is described.

CASE REPORT

A 12-year-old boy was admitted with the clinical diagnosis of patent ductus arteriosus. His upper limb blood pressure was 110/70 mm Hg, and his lower limb pressure was 130/80 mm Hg. The diagnosis was confirmed by an electrocardiogram, a chest radiograph, and echocardiography. There was no evidence of coarctation of the aorta. Surgical closure of the patent ductus arteriosus was undertaken after routine blood investigations and a thorough anesthesia check-up. Blood pressure was monitored by a right radial arterial line. There was no rise in blood pressure during the surgical approach. Systolic blood pressure was lowered to approximately 80 mm Hg just prior to dissection of the ductus, by continuous infusion of sodium nitroprusside. The ductus was divided

and sutured, and the blood pressure was allowed to normalize by reducing the dose of sodium nitroprusside. Perfect hemostasis was achieved; there was no evidence of bleeding from the suture lines on the aorta or the pulmonary artery. The chest was closed, and the patient was extubated. After 15 minutes, there was a sudden increase in blood pressure to 220/110 mm Hg, with a gush of bright red blood in the intercostal drainage tube. Arterial blood gas analysis was within normal limits. As the patient had lost 250 mL of blood in 15 to 20 minutes, he was returned to the operating room for reexploration. To reduce the blood pressure, sodium nitroprusside dosage was increased to maximum, and an infusion of nitroglycerin and diltiazem was started. The blood pressure decreased but remained above 150/90 mm Hg. On reexploration, there was bleeding from the needle holes along the entire aortic suture line, but no loose sutures were detected, and there was no visible narrowing of the aorta. The aorta was clamped with a side-biting clamp that included the entire suture line. The suture line was reinforced with continuous vertical mattress stitches to prevent any further cutting through of the suture. On

For reprint information contact:

Anil G Tendolkar, MS Tel: 91 22 409 0802 Fax: 91 22 407 6100 email: atendolkar@yahoo.com

Department of Cardiovascular and Thoracic Surgery, Lokmanya Tilak Municipal Medical College and General Hospital, Sion, Mumbai, Maharashtra 400022, India.

releasing the clamp, there was no evidence of bleeding from the suture line. After routine closure of the chest, both sodium nitroprusside and diltiazem were administered at their maximum pharmacological dosages, but the blood pressure did not reduce. Sublingual nifedipine was also administered repeatedly, but the hypertension persisted.

The patient's blood pressure was continuously monitored postoperatively, it dropped gradually to 160/100 mm Hg over a period of 72 hours, and remained at that level; the pressure was the same in all 4 limbs. Investigations of the cause of hypertension included an abdominal ultrasound scan to rule out a renal disorder or pheochromocytoma (24-hour urinary vanillylmandelic acid was normal), and funduscopy which was normal. On the 4th postoperative day, the antihypertensive medications were changed to oral nifedipine 10 mg three times daily and captopril 25 mg twice daily, but the blood pressure remained above 140/100 mm Hg. The medications were changed after one week to a combination of enalapril 2.5 mg and chlorothiazide 25 mg twice daily. The patient was discharged on the 13th postoperative day on this medication. During follow-up, his blood pressure decreased to 110/70 mm Hg within 10 days of discharge. At the last follow-up (5 weeks after surgery), it was 120/80 mm Hg on the same medication.

DISCUSSION

Postoperative hypertension, defined as systolic blood pressure exceeding 140 mm Hg, occurs in 40% to 60% of patients.⁴ It is more common in those with a history of chronic hypertension preoperatively, prior beta-blocker therapy, and well-preserved left ventricular function. In prepubertal children, chronic hypertension is most likely caused by congenital or acquired renal parenchymal or vascular disease, coarctation of the aorta and, rarely, pheochromocytoma.⁵ Postoperative hypertension is most commonly encountered after coronary artery bypass grafting or surgical correction of various types of left ventricular outflow tract obstruction (aortic valve replacement, correction of coarctation of aorta, and repair of aortic aneurysm).⁶ The mechanism of idiopathic postoperative hypertension differs from patient to patient, but usually involves excessive sympathetic nervous system activity with elevated levels of circulating catecholamines, or pressor reflexes originating in the heart, great vessels,

or coronary arteries.^{7,8} Complications of elevated systemic pressure include an increased risk of postoperative bleeding, suture line disruption, and aortic dissection.

This patient had no history of preoperative chronic hypertension, remained normotensive during surgery, but developed severe systemic hypertension in the immediate postoperative period. The cause of his systemic hypertension appeared to be unrelated to the kidneys, adrenals, or coarctation of the aorta. Transient increases in blood pressure are quite common after closure of a patent ductus arteriosus with a large shunt, but persistent hypertension above 160/110 mm Hg is rare. It always regresses but may sometimes cause complications, particularly bleeding problems. Hyperactivity of the sympathetic nervous system resulting in a massive surge of catecholamines might have been the cause.¹⁻³

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