



Sodium Nitroprusside Treatment for Control of Decompensated Heart Failure

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Abstract

Hypertensive crisis is thought to be initiated by an abrupt increase in systemic vascular resistance, likely related to humoral vasoconstrictors. Left ventricular dilatation and failure may develop as a result of increased afterload due to hypertension. Left heart failure may result in secondary pulmonary hypertension by increasing pulmonary venous pressure. Here, we report the effects of sodium nitroprusside for treatment of hypertensive crisis complicated with acute left ventricular dilatation, heart failure, and pulmonary hypertension secondary to vesicoureteral reflux in a 10-year-old female patient. This article presents a complex clinical case treated by simple medication.

Key Words: Hypertensive crisis, dilated cardiomyopathy, pulmonary hypertension, sodium nitroprusside, heart failure, vesicoureteral reflux

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Introduction

Hypertensive crisis is thought to be initiated by an abrupt increase in systemic vascular resistance likely related to humoral vasoconstrictors. Renal diseases were the major underlying causes of first-attack hypertensive crisis in pediatric patients, and they could induce a first attack of hypertensive crisis at any age. Left ventricular dilatation and failure may develop as a result of increased afterload due to hypertension. Left heart failure may result in secondary pulmonary hypertension by increasing pulmonary venous pressure. Hypertensive crisis with cardiac complications is the most important hemodynamic prognostic marker in patients with cardiomyopathy. Vasodilator therapy is frequently needed to treat pulmonary hypertension and systemic arterial hypertension. Sodium nitroprusside (dose: 0.5–0.8 µg/kg/min) reduces afterload and preload via both arterial and venous vasodilating effects, and has been used in the control of pulmonary hypertension as well as hypertension crisis. Here, we describe the uncommon presentation of hypertensive crisis associated with vesicoureteral reflux (VUR) in a pediatric patient and report the effectiveness of sodium nitroprusside in treatment of hypertensive crisis complicated with acute left ventricular dilatation, heart failure, and pulmonary hypertension.

Case Report

A 10-year-old female patient, body weight 32 kg, presented with a 3-day history of malaise, pallor, dizziness, fatigue, and chest pain. The patient subsequently had occasional febrile episodes but no documented urinary tract infection. Physical examination showed slight peripheral edema, temperature 35.8°C, heart rate 156 beats/min, respiratory rate 46 per minute, and blood pressure 200/145 mmHg (> 99th percentile). Pulmonary auscultation revealed fine inspiratory crepitations throughout. Cardiac examination found that heart sounds I and II were audible, with an early diastolic murmur and loud pan-systolic murmur. The liver margin was palpable 4 cm below the right costal margin. Oxygen saturation was 85% in room air. The results of laboratory tests were as follows: white blood cell count $16 \times 10^9/L$, hemoglobin 11 mmol/l, thrombocytes $280 \times 10^9/L$, C-reactive protein 4 mg/L, urea 70 mg/dL (normal range 10–50 mg/dL), creatinine 0.9 mg/dL (normal range 0.5–1.2 mg/dL), sodium 136 mmol/L, and potassium 4.5 mmol/L. Chest X-ray revealed enlargement of the cardiac silhouette. Transthoracic echocardiography showed an ejection fraction of 39%, fractional shortening of 16% (Figure 1a), left ventricular enlargement, end-diastolic diameter of 43 mm

(Figure 2a), first-degree mitral regurgitation, and tricuspid regurgitation jet velocity of 3.63 m/s. The estimated pulmonary artery pressure was 52 mmHg. Electrocardiography showed sinus tachycardia with nonspecific T-wave changes.

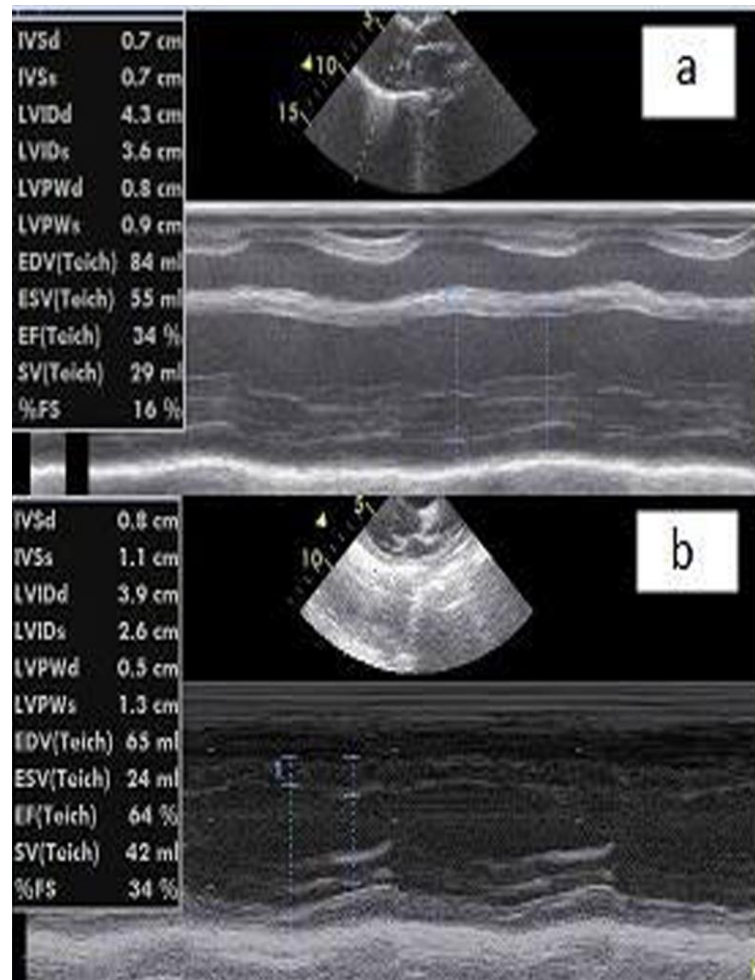


Figure 1. Left ventricular function parameters on M-Mode echocardiography (a) at admission and (b) after 1 month.

Intravenous furosemide (1 mg/kg/dose) was applied for decompensated heart failure, and intravenous infusion of sodium nitroprusside at 0.8 μ g/kg/min was initiated for systemic hypertension and pulmonary hypertension. Continuous noninvasive monitoring of blood pressure was performed. After 3 h, the heart rate was 117/min, arterial blood pressure was 140/104 mmHg, and transthoracic echocardiography revealed a left ventricular ejection fraction of 52%, fractional shortening of 28%, and tricuspid regurgitation jet velocity of 2.84 m/s; sodium nitroprusside infusion was terminated. There were no side effects of the treatment. Treatment was continued with enalapril maleate (0.2 mg•kg⁻¹•day⁻¹, per oral). Left

ventricular function parameters after 1 month were as follows: ejection fraction 64%, fractional shortening 34% (Figure 1b). Investigations were performed to determine the cause of hypertension. Ultrasound revealed small kidneys. Renal scarring was detected by technetium-99m-dimercaptosuccinic acid renal scintigraphy. Voiding cystourethrography showed grade 4–5 primary bilateral vesicoureteral reflux (VUR) (Figure 3).

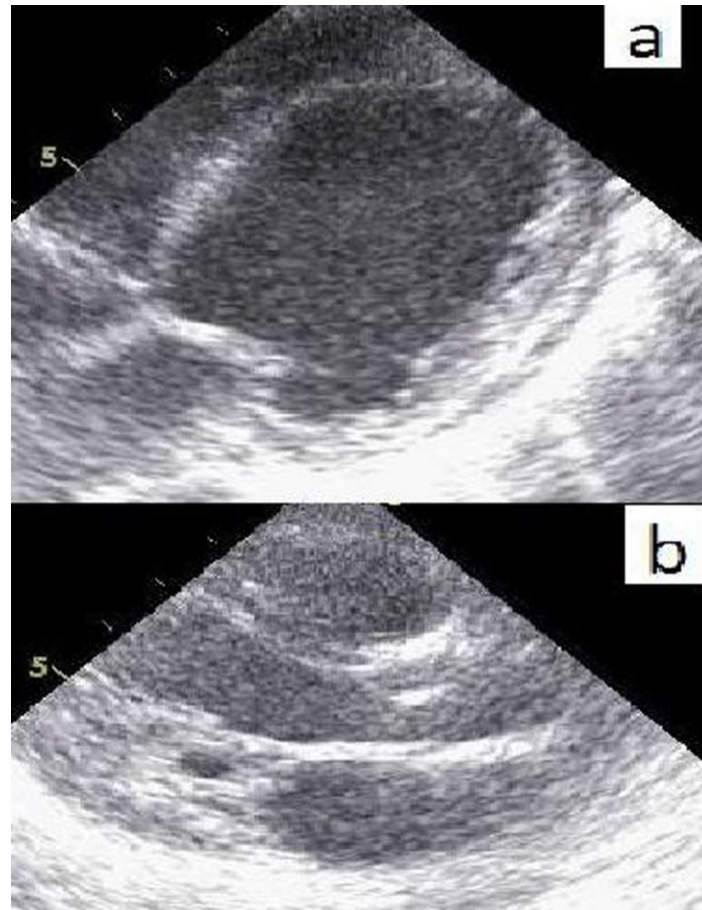


Figure 2. Two-dimensional (four-chamber) echocardiographic view (a) at admission and (b) two-dimensional (long axis) echocardiographic view after 1 month.

The patient recovered from decompensated heart failure and no complications related to the treatment were observed. Monthly follow-ups revealed normal arterial blood pressure and pulmonary artery pressure, and echocardiographic evaluation showed normal values for left heart dimensions (Figure 2b). Cefixime (4 mg /kg/dose) prophylaxis was initiated, and bilateral Cohen's antireflux surgery procedure was planned.



Figure 3. Voiding cystourethrogram showed grade 4–5 primary bilateral vesicoureteral reflux.

Discussion

Hypertensive crisis in children is a relatively rare condition presenting with elevated blood pressure. Hypertensive crisis is defined as elevation in SBP/DBP >99th percentile +5 mmHg. The episode is considered a hypertensive emergency when there is an immediate threat to the integrity of the cardiovascular system, kidneys, or central nervous system [1-3]. Our patient presented with systemic arterial pressure >200 mmHg and end organ damage consisting of acute left ventricular dilatation with congestive heart failure.

Functional voiding disorders are common in childhood but are usually not accompanied by upper urinary tract deterioration. Nevertheless, a small group of children remain at risk of developing chronic renal insufficiency (CRI). In a previous report, three of six patients with a diagnosis of VUR developed hypertension, and one patient developed hypertensive crisis [4].

Systemic arterial hypertension has been shown to be an important etiological risk factor for all types of heart failure. Left ventricular dilatation and failure may occur as a result of increased afterload related to rapidly progressive hypertension [5]. Our patient also experienced decreased left ventricular function due to hypertensive crisis, and developed left ventricular dilatation, and ultimately, decompensated heart failure syndrome. Pulmonary hypertension is defined as mean pulmonary artery pressure >25 mmHg. Increased left ventricular end-diastolic pressure and a subsequent increase in pulmonary venous pressure have been shown to be among the causes of pulmonary hypertension. In our case, pulmonary hypertension was thought to be a result of reduced left ventricular function and increased venous pressure related to ventricular dilatation.

Mean systemic pressure and mean pulmonary arterial pressure are the most important baseline hemodynamic prognostic factors in patients with cardiomyopathy due to a wide range of ischemic and non-ischemic causes. Medications commonly used to treat hypertensive crisis in children include nicardipine, labetalol, and sodium nitroprusside [2]. Sodium nitroprusside has a rapid onset of action and reduces pre-load and after-load; consequently, it is beneficial in congestive heart failure induced by hypertensive crisis. This drug has been used for many years in the treatment of hypertensive crisis in children [6]. Vasodilator therapy is frequently needed to treat pulmonary hypertension. Furthermore, patients with myocarditis and pulmonary hypertension are at particularly high-risk. These findings may aid in the interpretation of hemodynamic data and suggest that patients with pulmonary hypertension and myocarditis should be targeted for aggressive medical therapy.

Sodium nitroprusside activates guanylate cyclase directly through the release of nitric oxide, independent of the vascular endothelium, and the guanylate cyclase provides vascular relaxation by increasing cyclic adenosine monophosphate. Sodium nitroprusside has been shown to be effective in the control of pulmonary hypertension as well as hypertension crisis. In 10 patients undergoing ventricular septal defect repair, the operation process was shown to be successful by reduction of pulmonary artery pressure and systolic pressure using

intravenous sodium nitroprusside [7]. In a study of patients with end-stage heart failure and severe pulmonary hypertension, 33 patients were given inotropes (dopamine or dobutamine), 22 patients were given nonselective vasodilators (nitroglycerin or sodium nitroprusside), and 24 patients were given prostacyclin. There was a significant decrease in pulmonary pressure, with increased cardiac output compared to baseline hemodynamics in all groups. No significant differences were observed between agents [8]. In our case, the blood pressure decreased to 140/100 mmHg at the end of the third hour after giving sodium nitroprusside for hypertensive crisis and pulmonary hypertension. The pulmonary artery pressure was reduced from 46 to 37 mmHg. Sodium nitroprusside treatment was discontinued at this stage, and enalapril maleate treatment was continued. Positive changes in cardiac function were observed in a short time. Thiocyanate poisoning can cause methemoglobinemia, metabolic acidosis, altered mental status, and seizures. Sodium nitroprusside can increase intracranial pressure [2]. The patient recovered from decompensated heart failure, and no complications related to the treatment were observed.

Conclusion

Hypertensive crisis complicated with acute left ventricular dilatation, heart failure, and pulmonary hypertension secondary to VUR with reflux nephropathy is uncommon in pediatric patients. Treatment with sodium nitroprusside infusion is effective to control the blood pressure during hypertensive crisis in pediatric patients. Here, we presented a complex clinical case, which was treated by simple medication.

Conflict of interests

The authors have no conflicts of interest or funding to disclose.

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