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Case Report

Severe hypertension as an unusual presentation of intussusception in a 6-month-old infant: a case report

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ABSTRACT

Intussusception is a common pediatric gastrointestinal emergency that typically presents with abdominal pain, vomiting, and blood-stained stools. Severe hypertension is an uncommon and potentially overlooked manifestation of intussusception. We report a 6-month-old female infant who presented with lethargy, poor feeding, blood-stained stools, and severe hypertension of 130/80 mmHg, exceeding the 95th percentile for age, sex, and length. Serial blood pressure measurements were obtained during quiet, non-distressed periods using an appropriately sized cuff to minimize the possibility of pain- or agitation-related elevation. Initial management included oral nifedipine, followed by intravenous labetalol infusion because of persistent hypertension. Abdominal ultrasonography demonstrated the characteristic target sign of intussusception, which was successfully treated with fluoroscopy-guided pneumatic reduction. Following successful reduction, blood pressure progressively normalized. Evaluation for secondary causes of hypertension, including serum electrolytes, renal function tests, urinalysis, screening echocardiography, and dedicated renal Doppler ultrasonography, revealed no evidence of renal parenchymal disease, renovascular pathology, including renal artery stenosis, or structural cardiovascular abnormalities. Antihypertensive therapy was subsequently tapered and completely discontinued within 5 days after discharge. At 3-month follow-up, the infant remained asymptomatic and normotensive, with no evidence of recurrence. Intussusception may therefore be considered in the differential diagnosis of acute severe hypertension in infants, particularly when gastrointestinal manifestations are present.

Keywords: Intussusception, Severe hypertension, Pediatric emergency, Pneumatic reduction

INTRODUCTION

Intussusception is defined as the invagination of a proximal segment of intestine into an adjacent distal segment and represents a leading cause of acute intestinal obstruction in infants, with the highest incidence occurring between 4 and 10 months of age.[1] The classical clinical triad of intermittent abdominal pain, a palpable sausage-shaped abdominal mass, and currant jelly stools is present in fewer than 15% of affected children at presentation.[2] Severe hypertension in infancy is uncommon and is most frequently associated with secondary causes, including renal parenchymal disease, renovascular abnormalities, coarctation of the aorta, and endocrine disorders.[3]

Hypertension associated with pediatric intussusception is an uncommon clinical phenomenon, with only a limited number of cases reported in the literature.[4] Earlier reports described transient elevations in BP that resolved spontaneously following reduction of the intussusception, without the need for specific antihypertensive therapy.[5]

Similar transient responses have subsequently been reported.[6] Additional observations have supported a temporal association between successful reduction and normalization of blood pressure.[7] However, severe hypertension requiring pharmacological stabilization before definitive reduction remains exceptionally rare.[8] We report a 6-month-old infant with severe hypertension associated with ileocolic intussusception, in whom successful pneumatic reduction was followed by sustained normalization of blood pressure.

CASE REPORT

A 6-month-old female infant, born to a non-consanguineous couple, with age-appropriate developmental milestones, was brought to the pediatric emergency department with a 12-hour history of lethargy, refusal to feed, and blood-stained stools. On examination, the infant was lethargic but adequately hydrated, with normal skin turgor and no central or peripheral cyanosis. Initial vital signs revealed a heart rate of 130 beats per minute and a blood pressure of 130/80 mmHg, exceeding the 95th percentile for age, sex, and length. To minimize the possibility of spurious elevation related to crying, agitation, or acute distress, blood pressure was measured using an oscillometric device with an appropriately sized cuff and was manually verified during quiet, resting periods. Abdominal examination revealed a firm, non-tender mass in the right hypochondrium without guarding or rigidity. Bowel sounds were present. Per-rectal examination demonstrated blood-stained mucus on the examining glove. Respiratory and cardiovascular examinations were otherwise unremarkable, with equal bilateral air entry and normal heart sounds without audible murmurs.

In view of the markedly elevated blood pressure, acute blood pressure control was initiated with oral nifedipine at 0.3 mg/kg/dose. Reassessment one hour later demonstrated persistent hypertension, with a blood pressure of 120/80 mmHg, remaining above the 95th percentile. An intravenous labetalol infusion was therefore initiated at 0.5 mg/kg, resulting in stabilization of the mean arterial pressure (MAP). Concurrent evaluation was undertaken to identify potential secondary causes of hypertension. Serum biochemistry demonstrated preserved renal function, with a blood urea nitrogen of 10 mg/dL, serum creatinine of 0.3 mg/dL, serum sodium of 138 mEq/L, and serum potassium of 4.2 mEq/L. Urinalysis showed normal specific gravity without proteinuria, glucosuria, or microscopic hematuria. Dedicated renal Doppler ultrasonography demonstrated normal renal parenchymal echogenicity, symmetrical bilateral perfusion, and normal renal arterial flow velocities, with no evidence of renal artery stenosis. Screening echocardiography showed no evidence of coarctation of aorta/ other structural cardiac abnormality.

A plain abdominal radiograph demonstrated soft-tissue mass densities in the right iliac fossa, left iliac fossa, and umbilical region, with paucity of rectal gas (Figure 1). Abdominal ultrasonography subsequently confirmed ileocolic intussusception, demonstrating the characteristic target or donut sign produced by concentric bowel loops (Figure 2). The infant underwent successful fluoroscopy-guided pneumatic reduction without procedural complications or evidence of bowel perforation (Figure 3). Following reduction, the patient was transferred to the Pediatric Intensive Care Unit for close monitoring. Continuous arterial blood pressure monitoring over the subsequent 48 hours demonstrated a progressive and sustained decline in MAP following successful reduction of the intussusception and clinical improvement (Figure 4). The intravenous labetalol infusion was gradually weaned, and the patient was subsequently transitioned to a low-dose oral antihypertensive agent before transfer to the ward. Oral antihypertensive therapy was progressively tapered and completely discontinued within 5 days after discharge. At the 3-month follow-up visit, the infant remained clinically asymptomatic, with a blood pressure of 90/55 mmHg and no sonographic evidence of recurrent intussusception.

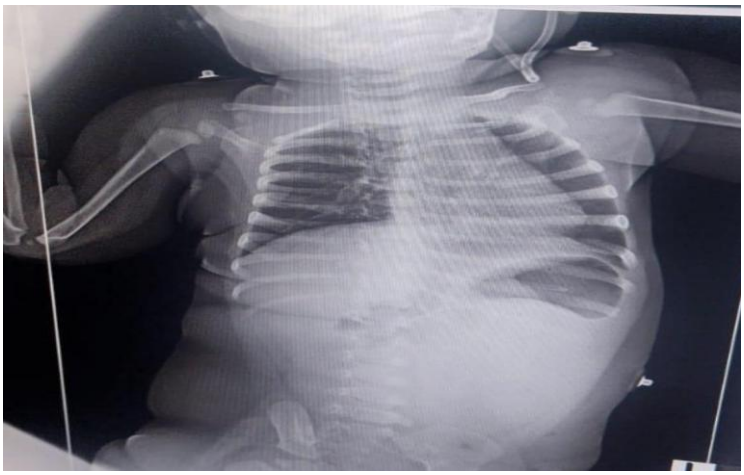


Figure 1: Plain abdominal radiograph demonstrating soft tissue mass density in the right and left iliac fossa and umbilical region with absent rectal gas.

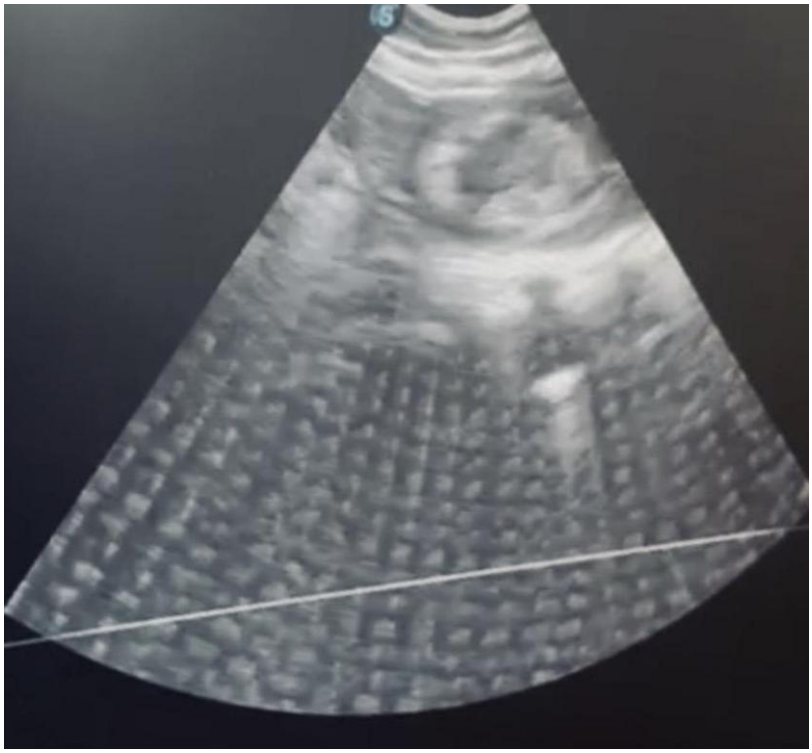


Figure 2: Abdominal ultrasound demonstrating a swirled pattern of alternating hyperechoic and hypoechoic bowel loops, representing the pathognomonic target sign of intussusception.



Figure 3: Fluoroscopic image demonstrating successful fluoroscopy-guided pneumatic reduction of the ileocolic intussusception.

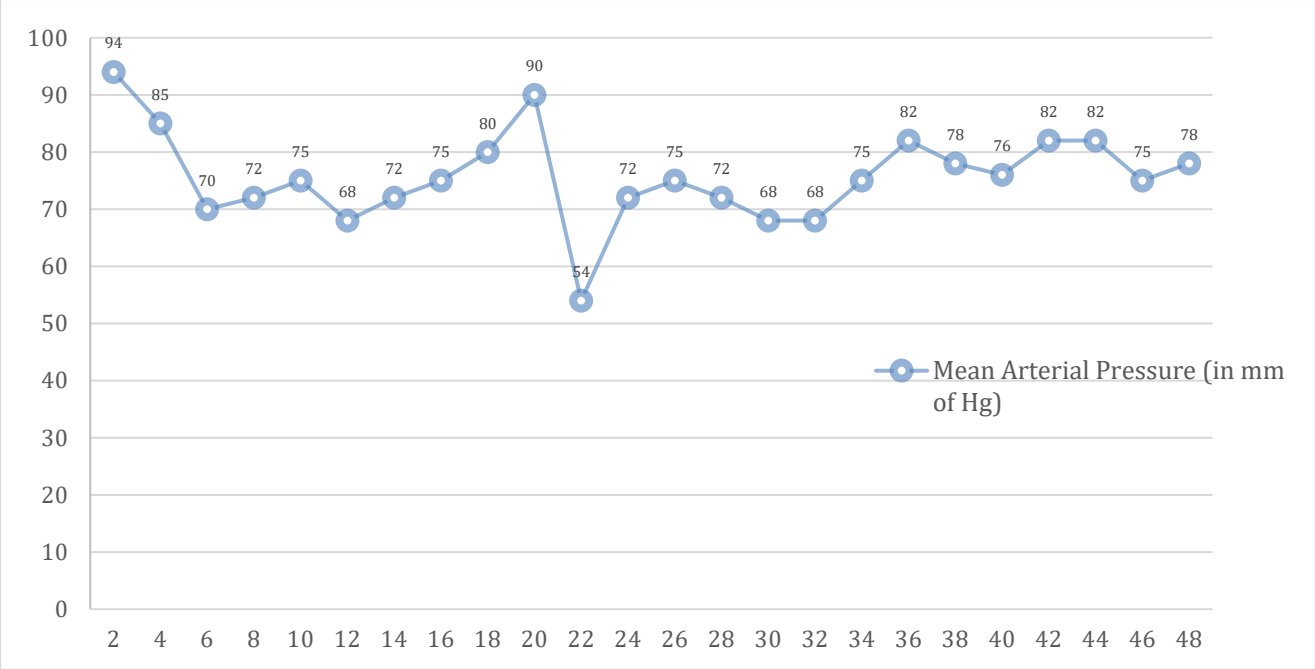


Figure 4: MAP changes over a 48-hour time duration.

*Sequential hemodynamic monitoring demonstrates fluctuations in MAP (in mm of Hg) corresponding to pharmacological interventions and dose adjustments. Key clinical events and dosing milestones are indicated at specific timepoints: initial administration of 0.25 mg/kg at 6 hours, a dose reduction to 0.125 mg/kg at 12 hours, a subsequent increase to 0.25 mg/kg at 20 hours, complete cessation of the intervention resulting in a sharp pressure drop at 22 hours, and a final resumed administration of 0.25 mg/kg at 36 hours.

DISCUSSION

Intussusception is the most common cause of intestinal obstruction in young children.[1] Definitive treatment is generally achieved using non-operative reduction techniques, including fluoroscopy-guided pneumatic reduction and ultrasound-guided hydrostatic reduction. Ultrasound-guided reduction has the additional advantage of avoiding ionizing radiation exposure.[2]

The pathophysiological association between acute intussusception and hypertension is likely to involve activation of the sympathetic nervous system in response to visceral nociceptive and ischemic stimuli. Invagination of the proximal bowel results in mesenteric traction, progressive luminal distension, and varying degrees of localized ischemia. These stimuli activate visceral afferent pathways and may produce marked sympathetic discharge with increased adrenal catecholamine release, particularly epinephrine and norepinephrine.[3] Increased alpha-adrenergic stimulation results in peripheral vasoconstriction and consequent elevation of systemic vascular resistance, thereby contributing to an acute rise in blood pressure. In addition, localized splanchnic ischemia may transiently stimulate the renin-angiotensin-aldosterone system and potentially augment the hypertensive response.[4]

Historically, Prichard et al described an infant with a blood pressure of the 140/90 mmHg that normalized following reduction.[5] Barton et al subsequently reported a similar transient elevation in blood pressure.[6] Paget et al also documented hypertension associated with intussusception that resolved following successful reduction.[7] Hida et al. reported blood pressure elevations ranging from 140/90 to 160/100 mmHg, with spontaneous or minimally assisted resolution following reduction.[8] In contrast, de Vries et al described a 10-month-old infant with malignant hypertension of 170/110 mmHg who required treatment with sodium nitroprusside and labetalol before definitive surgical management.[9]

Our patient demonstrated a similarly marked hypertensive response and required pharmacological blood pressure control with oral nifedipine followed by intravenous labetalol before pneumatic reduction could be performed. Following successful reduction, blood pressure progressively declined and remained within the normal range without the need for continued antihypertensive therapy.[4]

An important clinical consideration is distinguishing sustained hypertension from transient blood pressure elevation related to pain, crying, or agitation. In the present case, elevated blood pressure was documented repeatedly during calm, non-crying periods and included elevation of diastolic pressure. The persistence of hypertension despite initial oral therapy and the requirement for continuous intravenous labetalol further supported the presence of clinically significant hypertension. Evaluation for recognized secondary causes was unrevealing, with normal renal function, urinalysis,

echocardiographic findings, and renal Doppler parameters, providing no evidence of renal parenchymal, renovascular, or structural cardiovascular disease.[10] The temporal relationship between successful pneumatic reduction and sustained normalization of blood pressure, together with complete discontinuation of antihypertensive therapy and absence of recurrence during follow-up, supports an association between the intussusception and the acute hypertensive episode. However, given the observational nature of a single case, this temporal association should be interpreted as supportive rather than definitive evidence of causality.

CONCLUSION

Severe hypertension is an under reported and atypical presentation of intussusception in children. This case underscores the importance of considering intussusception along with other secondary causes in the differential diagnosis of unexplained acute hypertension in children. Early recognition of this atypical presentation is important so as to prevent the end organ damages that might be caused by long standing severe hypertension.

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