International Journal of Contemporary Pediatrics
*Sankpal V et al. Int J Contemp Pediatr. 2026 Jun;13(6):1004-1007*
https://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20261548

**Case Series**

# From kidneys to cerebral vessels: diverse faces of paediatric hypertensive emergencies

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**Received:** 11 March 2026
**Revised:** 09 April 2026
**Accepted:** 17 May 2026

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## ABSTRACT

Hypertensive emergency in children is rare but potentially life-threatening, requiring urgent recognition and management. Etiologies range from renal to vascular and hematological disorders. We report a descriptive case series of six paediatric patients (ages 1.5-12 years) presenting with acute severe hypertension and target organ damage. Clinical features, underlying causes, diagnostic findings, treatment, and outcomes were analysed. Six cases were analyzed with mean age 8.5 years and male predominance (4:2). Etiologies included atypical hemolytic uremic syndrome (33%, n=2), chronic kidney disease (17%, n=1), autosomal recessive polycystic kidney disease (17%, n=1), Moyamoya disease with β-thalassemia (17%, n=1), and drug-induced post-bone marrow transplant (17%, n=1). Neurological symptoms occurred in 83% cases, with headache, seizures, and altered sensorium being predominant presentations. All patients presented with blood pressure above 99$^{th}$ percentile requiring immediate intervention. Treatment included intravenous antihypertensives (labetalol, sodium nitroprusside) combined with etiology-specific therapy including plasmapheresis, hemodialysis, peritoneal dialysis, and immunosuppressive withdrawal. Complete recovery occurred in 50% cases (n=3), partial recovery in 17% (n=1), and mortality in 33% cases (n=2). Deaths were associated with atypical HUS and ARPKD indicating poor prognosis in this subgroup. Hypertensive emergencies in children have diverse etiologies and high morbidity risk. Rapid blood pressure control combined with management of the underlying cause is essential for improving outcomes. Early diagnosis, individualized therapy, and multidisciplinary care remain crucial in reducing adverse outcomes.

**Keywords:** Hypertensive emergency, Pediatric, Atypical HUS, Moya Moya disease, Polycystic kidney disease, Chronic kidney disease, Bone marrow transplant

## INTRODUCTION

Hypertensive emergency in children is a rare but serious medical condition. As per the Indian Academy of Pediatrics (IAP), it is defined as a sudden and severe rise in blood pressure above the 95$^{th}$ percentile for age, sex, and height, along with damage to vital organs such as the brain, heart, kidneys, or eyes. This is distinguished from hypertensive urgency, where the blood pressure is high but without organ injury. High blood pressure in children is becoming increasingly common globally.[1]

Estimates suggest pediatric hypertension incidence is about 2-5%, while in India, recent studies indicate around 7% of children have hypertension and nearly 10% have prehypertension. Adolescents show even higher rates, with surveys reporting elevated blood pressure in more than one-fourth of cases. Although hypertension itself is increasingly recognized, true hypertensive emergencies are uncommon, with studies indicating around one-third of hypertensive crises in children progress to emergencies.[2] The most frequent causes are kidney-related-such as glomerulonephritis, chronic kidney disease, and renovascular disease-followed by heart

problems, endocrine disorders, and drug side effects.[2] Urgent, controlled lowering of blood pressure using intravenous medicines (labetalol, nicardipine, nitroprusside) is critical, succeeded by oral therapy for long-term control.

If untreated, complications like seizures, heart failure, stroke, or kidney injury can occur. Early recognition and intervention significantly reduce mortality and long-term disability. This study highlights the clinical features, etiologies, and outcomes of pediatric hypertensive emergencies to improve clinician awareness and guide timely intervention.

## CASE SERIES

### *Case 1*

A 10-year-old girl presented with fever, vomiting, fatigue, and giddiness. Examination revealed pallor and petechiae, with lab tests confirming microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury. Elevated anti-complement factor H antibody levels supported a diagnosis of atypical HUS. She received steroids and plasmapheresis, but developed progressive hypertension and hypertensive encephalopathy manifesting as headache, seizures, and altered sensorium. Despite oral and IV antihypertensives, only intravenous sodium nitroprusside was effective. Plasmapheresis was stopped due to instability; she was managed with oral antihypertensives (calcium channel blockers, ACE inhibitors) and immunosuppressive therapy (steroids, MMF, cyclophosphamide). BP control was maintained after discharge, with no relapse of HUS despite high antibody titers.

### *Case 2*

An 8-year-old boy with transfusion-dependent thalassemia was admitted for abdominal distension, edema, and dyspnea. On examination, severe pallor, heart failure, and hepatosplenomegaly were evident. Blood tests showed severe anemia, pancytopenia, iron overload, and abnormal liver function. After improvement with transfusions, he developed headache, vomiting, and focal seizure associated with marked hypertension. Managed initially with oral nifedipine and later IV labetalol, his BP stabilized. CT angiography revealed a left MCA aneurysm, subarachnoid hemorrhage, and puff of smoke collaterals, confirming Moyamoya angiopathy. Oral maintenance antihypertensives proved effective; the patient remained stable with neurosurgical consultation and tapering therapy over follow-up.

### *Case 3*

An 11-year-old girl with thalassemia underwent bone marrow transplant 10 months prior and was on immunosuppressive therapy (prednisolone, cyclosporine). She presented with headache, generalized seizure, and altered sensorium; prior hypertension had resolved. At admission, a generalized seizure was treated with IV midazolam. Blood pressure exceeded the 99$^{th}$ percentile and was stabilized with IV labetalol; immunosuppressives were temporarily withheld. Subsequent recovery involved normal ECG, imaging, and labs. Oral amlodipine-controlled BP and she was discharged, though later lost to follow-up.

### *Case 4*

A 12-year-old boy, previously operated for rectovesical fistula and diagnosed with unilateral renal agenesis, presented with fever, urinary incontinence, and acidotic breathing. He was found to have oliguria, stage 5 CKD, and culture positive UTI with severely elevated BUN and creatinine. Hemodialysis improved renal parameters and daily dialysis was maintained. Supportive CKD management included antibiotics, salt restriction, diuretics, sodium bicarbonate, erythropoietin, and calcium-phosphate supplementation. BP was uncontrolled, necessitating IV labetalol and sustained hemodialysis. He achieved partial recovery, was discharged on oral antihypertensives, and scheduled for regular dialysis.

### *Case 5*

A 1.5-year-old boy with autosomal recessive polycystic kidney disease (ARPKD) and hepatic cysts, on CKD treatment and UTI prophylaxis, presented with decreased activity, poor feeding, reduced urine output, and fever. Examination revealed irritability, acidotic breathing, grade II hypertension, and seizure-like episodes. Pyuria and deranged renal tests indicated stage IV CKD; sepsis was confirmed by culture. IV labetalol failed to improve his clinical state and he deteriorated further despite antibiotics and supportive care. Peritoneal dialysis was initiated but had to be withheld due to hemodynamic instability in septic shock, leading to progressive renal failure and death.

### *Case 6*

An 8-year-old boy with a history of atypical hemolytic uremic syndrome (aHUS), previously treated with plasma exchange and immunosuppressive therapy, presented with vomiting, pallor, Grade II hypertension, tachycardia, pedal edema, and hepatomegaly during his second relapse. Laboratory tests revealed schistocytes, thrombocytopenia, metabolic acidosis, and high anti-complement factor H antibody titers (6000 AU). He received seven cycles of plasma exchange and five cycles of hemodialysis. Persistent, refractory hypertension was unresponsive to oral agents (prazosin, nicardipine, clonidine), requiring escalation to intravenous labetalol and sodium nitroprusside. The patient developed worsening edema, progressive abdominal distension, and clinical ascites. Despite plans for further plasmapheresis, his condition deteriorated with the onset of pulmonary

edema, which did not improve despite intensive diuretic therapy and ventilatory support. Ultimately, he succumbed to complications of progressive multi-organ dysfunction and pulmonary edema after aggressive treatment.

**Table 1: Clinical profile, investigations, management and outcome of all patient.**

| Parameters | | N (%) |
|---|---|---|
| **Age group (in years)** | <5 | 1 (16.7) |
| | 5-10 | 3 (50) |
| | >10 | 2 (33.3) |
| **Sex** | Male | 4 (66.7) |
| | Female | 2 (33.3) |
| **Presenting features** | Headache | 3 (50) |
| | Vomiting | 2 (33.3) |
| | Seizures | 5 (83.3) |
| | Altered sensorium | 3 (50) |
| | Edema | 1 (16.7) |
| **Etiology** | Renal causes | 4 (66.7) |
| | Neurological causes | 1 (16.7) |
| | Drug induced | 1 (16.7) |
| **Clinical findings** | Neurological signs | 6 (100) |
| | Cardiovascular signs | 1 (16.7) |
| | Oligouria | 3 (50) |
| | Fundoscopy abnormality | 1 (16.7) |
| **Investigations** | Renal dysfunction | 4 (66.7) |
| | Abnormal neuroimaging | 1 (16.7) |
| **Management** | Labetelol use | 6 (100) |
| | Sodium nitroprusside use | 2 (33.3) |
| | Dialysis required | 2 (33.3) |
| **Outcome** | Complete recovery | 2 (33.3) |
| | Partial recovery | 2 (33.3) |
| | Death | 2 (33.3) |

## DISCUSSION

Our case series findings demonstrate diverse etiological patterns and outcomes in pediatric hypertensive emergencies that align with established literature while revealing unique institutional patterns. The mean age of presentation was approximately 8.5 years, consistent with Vijai et al who reported higher prevalence in school-going children (36%) and adolescents (43%), though our series included a broader age range from 1.5 to 12 years.[3] The male predominance (4:2 ratio) differs from Vijai et al reported male-to-female ratio of 2.5:1, likely reflecting the small sample size effect in our series.[3]

Regarding etiology, our series showed 66% renal causes (4/6 cases with atypical HUS, ARPKD, and chronic kidney disease), which is slightly lower than the 68% reported by Vijai et al and 75% documented by Yang et al.[3,4] This difference may be attributed to our inclusion of unusual cases like Moyamoya disease and drug-induced hypertensive emergencies, which represent less common but clinically significant etiologies. The presence of two atypical HUS cases (33% of our series) is notably higher than the 5% reported in larger studies, suggesting either selection bias toward complex hematological cases at our tertiary center or potential geographic clustering. Raina et al. emphasized that secondary causes contribute to greater incidence of hypertensive crisis in pediatrics, supporting our observation of predominantly secondary etiologies.[5]

The clinical presentation pattern in our series showed neurological symptoms in 83% of cases (5/6), which exceeds the 55% headache prevalence and 11-20% seizure incidence reported in comprehensive reviews.[5,6] This higher neurological involvement may reflect more severe presentations reaching our tertiary care center or delayed recognition leading to advanced end-organ damage. Our case of Moyamoya disease presenting with hypertensive encephalopathy is particularly rare, as Inaguma et al noted that most hypertension in Moyamoya disease remains unexplained and is often refractory to conventional management.[7]

Treatment response varied significantly in our series, with sodium nitroprusside required in severe cases, particularly both atypical HUS patients, indicating treatment-resistant hypertension. This aligns with Ba et al findings that end-organ damage represents a major risk factor for mortality in pediatric hypertensive emergencies.[8] The mortality rate in our series (33%, 2/6 cases) appears higher than the general pediatric hypertensive emergency mortality, which Yang et al suggested remains poorly defined but carries significant morbidity risk.[4] Deaths occurred in patients with atypical HUS and ARPKD, suggesting these etiologies carry particularly poor prognosis when complicated by hypertensive emergencies.

The drug-induced case following bone marrow transplantation highlights the increasing recognition of iatrogenic hypertensive emergencies, particularly with immunosuppressive agents like cyclosporine. This etiology is less commonly reported in pediatric series but represents an important consideration in the modern era of complex medical therapies.[5,9] The complete recovery achieved in this case after discontinuing the offending agent supports the importance of identifying and managing reversible causes.

## CONCLUSION

Pediatric hypertensive emergencies present diverse etiologies spanning renal, vascular, and hematological disorders. Our series demonstrates that atypical HUS constitutes a significant proportion with poor prognosis, while vascular causes like Moyamoya disease represent rare but important presentations. Drug-induced emergencies, particularly posttransplantation, emphasize the importance of monitoring immunosuppressive therapy. Neurological symptoms predominate clinical

presentations, occurring in 83% of cases. Treatment requires individualized approaches combining prompt blood pressure control with etiology-specific interventions.

The mortality rate remains concerning at 33%, primarily associated with complex hematological disorders requiring intensive therapeutic interventions. Early recognition protocols should be established for high-risk populations including chronic kidney disease and hematological disorders. Multidisciplinary management teams incorporating nephrology, neurology, and intensive care specialists enhance outcomes through coordinated care approaches.

## ACKNOWLEDGEMENTS

Authors sincerely thank Dr. Sangeeta Kumbhojkar, Head of the Department, and Dr. Shishir Mirgunde, Head of the Unit, for their guidance, support, and valuable suggestions.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

## REFERENCES

1. Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics. 2017;140(3):e20171904.
2. Tullus K. Hypertensive emergencies in children-a review. Pediatr Nephrol. 2018;33(1):49-57.
3. Vijai Anand Babu B. Clinical profile and etiological spectrum of hypertensive emergencies in children. Sch Acad J Biosci. 2018;6(1):84-9.
4. Yang WC, Zhao LL, Chen CY, Wu YK, Chang YJ, Wu HP. First-attack pediatric hypertensive crisis presenting to the pediatric emergency department. BMC Pediatr. 2012;12:200.
5. Raina R, Krishnappa V, Das A, Ronith C, Sarisha M, Sidharth KS, et al. Overview of hypertensive crisis in children. Front Pediatr. 2020;8:588911.
6. Flynn JT, Tullus K. Severe hypertension in children and adolescents: pathophysiology and treatment. Pediatr Nephrol. 2009;24(6):1101-2.
7. Inaguma D, et al. Hypertension associated with moyamoya disease: clinical characteristics and management. Clin Exp Nephrol. 2011;15(5):696-701.
8. Ba J. Clinical characteristics and outcomes of hypertensive emergencies in children. Pediatr Nephrol. 2019;34(12):2487-95.
9. Baracco R, Mattoo TK. Hypertensive crises in children and adolescents. Curr Hypertens Rep. 2014;16:456.

**Cite this article as:** Sankpal V, Awadkhan M, Jadhav M. From kidneys to cerebral vessels: diverse faces of paediatric hypertensive emergencies. Int J Contemp Pediatr 2026;13:1004-7.