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Childhood Polyarteritis Nodosa Presenting With Bilateral Panuveitis and Acute Kidney Injury: Diagnostic Value of Conventional Renal Angiography

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Nidale Hazzab [1], Yousra Elboussaadni [2], Abdallah Oulmaati [3], Hajar Elbhali [4], Amine Aazghari [5]

1. Faculty of Medicine, Abdelmalek Essaâdi University, Tangier, MAR 2. Pediatrics, University Hospital Center of Tangier-Tetouan-Al Hoceima, Tangier, MAR 3. Pediatrics and Neonatology, Mohammed VI Mother and Child University Hospital Center, Tangier, MAR 4. Vascular Surgery, University Hospital Center of Tangier (CHU Tanger), Tangier, MAR 5. Vascular Surgery, Tangier University Hospital Center (CHU Tanger), Abdelmalek Essaâdi University, Tangier, MAR

Corresponding author: Nidale Hazzab, nidale.hazzab010@gmail.com

Abstract

Polyarteritis nodosa (PAN) is a rare systemic vasculitis in children that may present with atypical manifestations, complicating diagnosis. We report the case of a previously healthy 13-year-old boy presenting with acute bilateral visual impairment, headache, and vomiting. Ophthalmologic examination revealed bilateral nongranulomatous panuveitis, while investigations identified marked systemic inflammation and acute kidney injury. Despite normal computed tomography angiography, conventional renal angiography established the diagnosis of PAN. Treatment with intravenous methylprednisolone followed by oral corticosteroids and azathioprine resulted in complete recovery of visual and renal function, with sustained remission over three years. This case highlights the importance of considering systemic vasculitis in children presenting with panuveitis and unexplained renal dysfunction and underscores the diagnostic value of conventional angiography when noninvasive vascular imaging is inconclusive.

Categories: Pediatrics
Keywords: acute kidney injury, angiography, case report, medium-vessel vasculitis, polyarteritis nodosa

Introduction

Polyarteritis nodosa (PAN) is a rare systemic necrotizing vasculitis that predominantly affects medium-sized arteries and may lead to vascular damage, aneurysm formation, and tissue ischemia [1,2]. Childhood-onset PAN is uncommon and may present with heterogeneous clinical manifestations involving the skin, kidneys, peripheral nervous system, and musculoskeletal system [2,3].

The diagnosis of PAN relies on clinical findings and evidence of vascular inflammation, supported by histopathological examination or characteristic angiographic abnormalities, when available. The EULAR/PRINTO/PRES classification criteria for childhood PAN emphasize the presence of necrotizing vasculitis affecting medium or small arteries, or angiographic abnormalities, together with at least one additional clinical criterion [1,3].

Ocular involvement in PAN is uncommon, and presentation with panuveitis may complicate the diagnostic process [1-3]. Therefore, ocular findings should be interpreted in conjunction with systemic manifestations and appropriate investigations.

We report a case of childhood-onset PAN presenting with panuveitis, highlighting the diagnostic challenges associated with this unusual clinical presentation and the importance of considering systemic vasculitis in the evaluation of atypical ocular inflammation.

Case Presentation

A previously healthy 13-year-old boy was admitted to our department with a one-week history of progressive bilateral visual impairment associated with photophobia. His symptoms began with a gradual decrease in visual acuity, followed by severe diffuse headache described as helmet-like, photophobia, and recurrent vomiting. He remained afebrile throughout the course of the illness but complained of marked fatigue. There was no history of recent infection, trauma, medication use, or systemic disease.

On admission, the patient appeared alert and in good general condition. His vital signs were stable, with a body temperature of 37 °C and a heart rate of 90 beats/min. He weighed 64 kg and measured 184 cm in height. Blood pressure measurements revealed a discrepancy between the upper and lower limbs, with values of 110/60 mmHg in the right arm, 120/60 mmHg in the left arm, 140/80 mmHg in the left leg, and

160/90 mmHg in the right leg.

Neurological examination was unremarkable, showing no focal deficits, meningeal signs, or sensory abnormalities. Cutaneous examination did not reveal livedo reticularis, purpura, subcutaneous nodules, or any other skin lesions suggestive of systemic vasculitis.

Ophthalmologic assessment demonstrated bilateral nongranulomatous, nonhypertensive panuveitis. Optical coherence tomography (OCT) revealed bilateral cystoid macular edema. Fluorescein angiography showed a retrofoveal serous retinal detachment associated with cystoid macular edema in the right eye, with a best-corrected visual acuity of 5/10, whereas the left eye demonstrated isolated cystoid macular edema with a visual acuity of 1/10.

Given the severity of the visual symptoms, cerebral magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) were performed, both yielding normal findings. Lumbar puncture was also performed, with normal cerebrospinal fluid (CSF) analysis and opening pressure.

Laboratory investigations at admission revealed a marked inflammatory response, with elevated C-reactive protein (CRP) levels and erythrocyte sedimentation rate (ESR). Renal function tests demonstrated acute kidney injury, with elevated blood urea and serum creatinine levels and a markedly decreased estimated glomerular filtration rate (eGFR), calculated using serum creatinine according to the bedside Schwartz equation. Cystatin C was not measured because this test was not available at our hospital. Twenty-four-hour urinary protein excretion was low, indicating the absence of significant proteinuria (Table *1*). Renal Doppler ultrasonography showed normal kidney size and preserved renal blood flow without evidence of renal artery stenosis.

An extensive etiological workup was performed to exclude infectious and noninfectious inflammatory conditions. The infectious workup, including viral serologies for hepatitis B, hepatitis C, human immunodeficiency virus, cytomegalovirus, and Epstein-Barr virus, was negative. Tuberculosis screening was also negative.

Regarding noninfectious inflammatory conditions, autoimmune investigations, including antinuclear antibodies (ANA), anti-double-stranded DNA antibodies, and antineutrophil cytoplasmic antibodies (ANCA), were negative, with complement fractions C3 and C4 within normal limits. Serum angiotensin-converting enzyme levels and the pathergy test were also negative, making sarcoidosis and Behçet disease less likely.

Because of the sight-threatening ocular involvement, treatment was initiated promptly with three consecutive daily pulses of intravenous methylprednisolone (30 mg/kg/day, maximum dose 1 g/day), followed by oral prednisone at 1 mg/kg/day. This therapeutic regimen is consistent with the 2021 ACR/Vasculitis Foundation recommendations for the management of severe childhood-onset PAN. The patient showed rapid clinical improvement, with progressive recovery of both visual acuity and renal function (Table *1*; Figure *1*). Blood pressure also progressively normalized under symptomatic antihypertensive treatment with amlodipine, alongside disease-directed therapy consisting of corticosteroids and immunosuppressive treatment. Follow-up ophthalmologic examination demonstrated partial resolution of retinal lesions, regression of macular edema, and retinal remodeling. Visual acuity improved to 7/10 in the right eye and 2/10 in the left eye. Serum creatinine decreased substantially, indicating significant recovery of renal function (Table *1*; Figure *1*).

Parameter	Day 1 (admission)	Day 5	Day 10	Day 13	Day 17 (discharge)	One-year follow-up	Reference range
Blood urea (g/L)	1.14 ↑	1.13 ↑	1.17 ↑	0.99 ↑	1.07 ↑	0.43	0.18-0.50
Serum creatinine (mg/L)	38 ↑	27 ↑	19 ↑	17 ↑	16.7 ↑	12	6-12
eGFR (mL/min/1.73 m²)	19 ↓	27↓	39↓	43↓	44↓	62↓	Age-appropriate reference required
Erythrocyte sedimentation rate (mm/h)	56 ↑	Not performed	Not performed	Not performed	15	10	<20
C-reactive protein (mg/L)	40 ↑	Not performed	27↑	Not performed	6	0.6	<6
24-hour urinary protein excretion (mg/kg/day)	4	Not performed	Not performed	Not performed	Not performed	3	<5

TABLE 1: Serial laboratory parameters and renal function during hospitalization and follow-up.

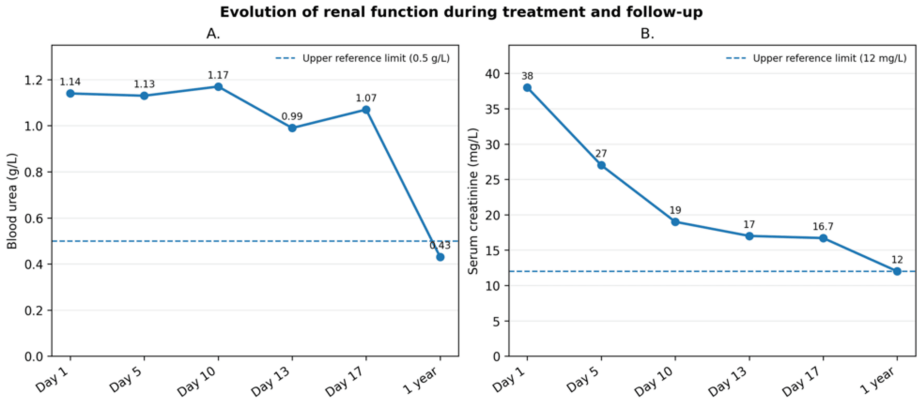


FIGURE 1: Serial changes in (A) blood urea and (B) serum creatinine levels over the course of hospitalization, illustrating the evolution of renal function in the patient.

Although thoracoabdominal CT angiography did not reveal any vascular abnormalities, the combination of severe bilateral panuveitis, persistent inflammatory syndrome, and acute kidney injury maintained a strong suspicion of medium-vessel vasculitis. Conventional selective renal angiography was therefore performed. It demonstrated multiple bilateral renal microaneurysms, segmental irregularities involving the intrarenal arterial branches, and an absence of vascularization of the upper pole of the left kidney, findings highly suggestive of PAN (Figures *2*-*3*).



FIGURE 2: Conventional angiography of the right kidney demonstrating multiple microaneurysms and segmental irregularities of the intrarenal arterial branches, consistent with renal vascular involvement in polyarteritis nodosa.



FIGURE 3: Conventional renal angiography showing absent vascularization of the upper pole of the left kidney, indicating a regional perfusion defect.

Based on the clinical presentation, laboratory findings, and characteristic angiographic abnormalities, a diagnosis of childhood-onset PAN was established. Oral corticosteroids were progressively tapered, and azathioprine (2 mg/kg/day) was introduced as maintenance therapy.

During three years of follow-up, the patient remained in sustained clinical remission, with complete recovery of renal function, sustained normalization of blood pressure, no recurrence of ocular inflammation, and no evidence of disease relapse.

Discussion

Childhood PAN is an uncommon necrotizing vasculitis involving medium-sized arteries and remains difficult to diagnose because of its broad clinical spectrum and the absence of disease-specific laboratory markers [1-4]. Although PAN represents only a small proportion of pediatric vasculitides, its rarity contributes to diagnostic challenges and limited clinical experience, particularly in children [2]. Clinical manifestations

vary considerably according to the organs involved, with constitutional symptoms, skin lesions, renal involvement, and musculoskeletal or neurological manifestations being the most frequently reported in pediatric patients [4-6]. Diagnosis may be particularly challenging when the disease presents with atypical manifestations.

In the present case, bilateral nongranulomatous panuveitis was the initial manifestation and preceded the diagnosis of PAN. Ocular involvement is uncommon in childhood PAN and has been reported mainly as episcleritis, scleritis, retinal vasculitis, retinal vascular occlusion, optic neuropathy, or, more rarely, uveitis [7,8]. The association of bilateral panuveitis, cystoid macular edema, serous retinal detachment, and acute kidney injury is exceptionally rare. This unusual presentation highlights the importance of considering systemic vasculitis in children with severe bilateral uveitis accompanied by persistent systemic inflammation and renal dysfunction, particularly after infectious and autoimmune etiologies have been excluded.

Renal involvement is a major determinant of disease severity and prognosis in PAN. Unlike immune-mediated glomerular diseases, renal impairment results primarily from inflammation of medium-sized renal arteries, leading to ischemic lesions rather than glomerular injury [4,9,10]. Consequently, acute kidney injury may occur despite a bland urine sediment and subnephrotic proteinuria, as observed in our patient. Recognition of this clinicobiological pattern is essential because it should prompt further evaluation for renovascular involvement.

The diagnosis in our patient was established according to the EULAR/PRINTO/PRES classification criteria, with conventional renal angiography providing the decisive diagnostic evidence [3]. Despite persistent clinical suspicion, thoracoabdominal CT angiography failed to detect vascular abnormalities, whereas selective renal angiography demonstrated bilateral microaneurysms, segmental arterial irregularities, and focal areas of renal hypoperfusion, findings highly suggestive of PAN. Renal biopsy was not performed because of the increased risk of hemorrhagic complications associated with the underlying renal vascular involvement. This discrepancy illustrates an important diagnostic limitation of noninvasive vascular imaging. Previous studies have shown that conventional angiography remains more sensitive for detecting distal medium-vessel lesions, particularly microaneurysms and segmental arterial abnormalities that may be overlooked on CT angiography [9]. Our case supports these observations and emphasizes that a normal CT angiogram should not preclude further vascular investigation when clinical suspicion of PAN remains high.

This case broadens the clinical spectrum of childhood PAN by describing an unusual association of bilateral panuveitis and acute kidney injury as presenting manifestations. It also reinforces the complementary role of conventional renal angiography in patients with strong clinical suspicion despite negative CT angiography findings, thereby contributing to earlier diagnosis and appropriate treatment.

Conclusions

This case illustrates an uncommon presentation of childhood-onset PAN, characterized by the coexistence of bilateral panuveitis and acute kidney injury, and adds to the limited clinical data on ocular involvement in this condition. It also emphasizes that normal findings on CT angiography do not rule out the diagnosis when clinical suspicion remains high. In our patient, conventional renal angiography was pivotal in demonstrating characteristic vascular lesions, confirming the diagnosis, and enabling the timely initiation of appropriate immunosuppressive therapy.

Clinicians should maintain a high index of suspicion for PAN in children presenting with severe ocular inflammation associated with systemic inflammatory markers and unexplained renal dysfunction. In our patient, the diagnosis was established based on the EULAR/PRINTO/PRES classification criteria, with conventional angiography providing decisive evidence of vascular involvement when noninvasive vascular imaging was inconclusive.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Nidale Hazzab, Abdallah Oulmaati, Hajar Elbhali, Amine Aazghari

Acquisition, analysis, or interpretation of data: Nidale Hazzab, Yousra Elboussaadni, Hajar Elbhali

Drafting of the manuscript: Nidale Hazzab, Hajar Elbhali

Supervision: Nidale Hazzab, Yousra Elboussaadni, Abdallah Oulmaati, Hajar Elbhali, Amine Aazghari

Critical review of the manuscript for important intellectual content: Yousra Elboussaadni, Abdallah Oulmaati, Amine Aazghari

Disclosures

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References

1. Jennette JC, Falk RJ, Bacon PA, et al.: 2012 revised International Chapel Hill Consensus Conference nomenclature of vasculitides. Arthritis Rheum. 2013, 65:1-11. 10.1002/art.37715
2. Watts RA, Robson J: Introduction, epidemiology and classification of vasculitis. Best Pract Res Clin Rheumatol. 2018, 32:3-20. 10.1016/j.berh.2018.10.003
3. Ozen S, Pistorio A, Iusan SM, et al.: EULAR/PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara 2008. Part II: Final classification criteria. Ann Rheum Dis. 2010, 69:798-806. 10.1136/ard.2009.116657
4. Saadoun D, Terrier B, Semoun O, et al.: Hepatitis C virus-associated polyarteritis nodosa . Arthritis Care Res (Hoboken). 2011, 63:427-35. 10.1002/acr.20381
5. Huang Z, Li T, Nigrovic PA, Lee PY: Polyarteritis nodosa and deficiency of adenosine deaminase 2 - shared genealogy, generations apart. Clin Immunol. 2020, 215:108411. 10.1016/j.clim.2020.108411
6. Lee JS, Kim JG, Lee S: Clinical presentations and long term prognosis of childhood onset polyarteritis nodosa in single centre of Korea. Sci Rep. 2021, 11:8393. 10.1038/s41598-021-87718-6
7. Foster CS, Vitale AT: Diagnosis and Treatment of Uveitis . Foster CS, Vitale AT (ed): W.B. Saunders, Philadelphia; 1942.
8. Jabs DA, Nussenblatt RB, Rosenbaum JT: Standardization of Uveitis Nomenclature (SUN) Working Group. Standardization of uveitis nomenclature for reporting clinical data: results of the First International Workshop. Am J Ophthalmol. 2005, 140:509-16. 10.1016/j.ajo.2005.03.057
9. Brogan PA, Davies R, Gordon I, Dillon MJ: Renal angiography in children with polyarteritis nodosa . Pediatr Nephrol. 2002, 17:277-83. 10.1007/s00467-002-0823-4
10. Guillevin L, Mahr A, Callard P, Godmer P, Pagnoux C, Leray E, Cohen P: Hepatitis B virus-associated polyarteritis nodosa: clinical characteristics, outcome, and impact of treatment in 115 patients. Medicine (Baltimore). 2005, 84:313-22. 10.1097/01.md.0000180792.80212.5e