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Hypertension without Urinary Abnormalities in Rheumatoid Purpura. Report of 2 Cases

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Abstract

Henoch-Schönlein purpura (HSP) is an IgA vasculitis, the most common in children. It often presents with atypical manifestations, with a risk of severe short-term gastrointestinal complications and delayed renal complications. Although hypertension is a known complication of HSP-associated nephropathy, it is rarely observed in children with normal renal function and urinalysis. Here we report two cases of isolated hypertension without signs of glomerulonephritis.

Keywords: *Rheumatoid Purpura (RA); Henoch-Schoenlein Purpura (HSP); IgA Vasculitis; Glomerulonephritis*

Introduction

Rheumatoid purpura (RA) or Henoch-Schoenlein purpura is a systemic leukocytoclastic vasculitis with IgA deposition that affects small vessels [1]. It is the most common type of vasculitis in children. The age of onset is 2 to 10 years, with a peak incidence between 4 and 7 years [2].

RA is often preceded by an infectious episode; the majority of cases are observed during colder seasons [3], confirming the triggering role of these infections, but the exact origin of the disease remains unknown.

RA is a benign condition; however, it can be serious due to the occurrence of digestive complications that affect the immediate prognosis and renal complications that jeopardize the long-term prognosis and can lead to chronic renal failure. Renal involvement determines the prognosis of the disease. It varies from 20 to 100% [4], and is expressed during the first weeks of the disease, but sometimes later [1].

In general, it is minimal, limited to transient microscopic or macroscopic hematuria, but proteinuria, nephrotic syndrome, renal failure and arterial hypertension can be observed [5].

Through our observations, we report two cases of rheumatoid purpura with isolated hypertension, without urinary abnormalities.

Case Description

Case study no. 1

This was a 2-year-8-month-old infant, a 35-week-old premature baby, being treated for spastic diplegia following ventricular leukomalacia. He had a respiratory infection prior to the onset of his symptoms. He was hospitalized for rheumatoid purpura consisting of necrotic purpuric patches on the lower limbs and face, associated with arthritis in both elbows.

The clinical examination revealed a conscious child in severe pain with a blood pressure of 13.9/10 mmHg, or the 97th percentile for systolic pressure and the 97th percentile for diastolic pressure. A negative urine dipstick test was performed. The cardiovascular examination was normal, with pulses present and symmetrical in all four limbs.

The paraclinical assessment showed: Plasma creatinine at 3.84 mmol/L, and blood urea nitrogen at 0.16 mmol/L. The serum protein level was 71.79 g/L, the serum albumin level was 38 g/L, the sodium level was 137 mmol/L, the potassium level was 3.4 mmol/L, and the calcium level was 72 mg/L. The 24-hour proteinuria was negative.

Hemoglobin level was 12.4 g/dL, platelets were 429,000 e/mm, white blood cells were 7,200 e/mm, CRP was 16 mg/L, prothrombin time was 100%, and the activated partial thromboplastin time was 26 seconds.

During the hospitalization, the child had experienced episodes of moderately heavy rectal bleeding; an abdominal ultrasound revealed a 6 mm parietal hematoma.

Regarding the investigation of hypertension, a repercussion assessment was performed; An eye examination, cardiac ultrasound, and electrocardiogram were normal.

An etiological assessment showed a 1st-hour sedimentation rate of 6 and a 2nd-hour sedimentation rate of 26, a normal renal and adrenal Doppler ultrasound, a normal abdominopelvic CT angiography, and a skin biopsy suggested leukocytoclastic vasculitis of small vessels with IgA deposits suggestive of rheumatoid purpura.

The child was placed on digestive rest, gastric protector, and IV methylprednisolone corticosteroid therapy (2 mg/kg per day) for 5 days, then switched to oral therapy followed by gradual tapering with adjuvant therapy. Antihypertensive treatment was administered with injectable nicardipine, followed by a switch to amlodipine and oral ACE inhibitors, with gradual tapering and monitoring of blood pressure and urine output. At 2 years' follow-up, the child was doing well and showed no signs of renal impairment, systemic disease, or autoimmune disorders.

Case study no. 2

A 12-year-old child with a history of flu-like symptoms a week earlier was hospitalized for rheumatoid purpura consisting of purpuric patches in the lower limbs, abdominal pain, and arthralgia with no signs of gastrointestinal bleeding.

The clinical examination found the child conscious with a BP of 15/10 mmHg, or the 97th percentile for systolic pressure and the 97th percentile for diastolic pressure. A negative urine dipstick test was performed. The cardiovascular examination was normal, and the heart rate was 90 beats/min with pulses present and symmetrical in all four limbs.

The paraclinical assessment showed: plasma creatinine at 6 mmol/L, urea at 0.27 mmol/L. The serum protein level was 83 g/L, the serum albumin level was 40 g/L, the sodium level was 138 mmol/L, the potassium level was 3.9 mmol/L, and the calcium level was 80 mg/L. The 24-hour proteinuria was negative, and the abdominal ultrasound was normal.

Regarding hypertension investigations, a funduscopy, cardiac ultrasound, electrocardiogram, and renal Doppler ultrasound were normal. The 8-hour cortisol level and urinary catecholamine levels were measured. The lipid profile was normal.

Antihypertensive treatment with oral nicardipine and ACE inhibitors was initiated with good progress, and blood pressure and urine monitoring were normal.

At 4 months' follow-up, treatment was discontinued; the child was doing well and showed no signs of renal involvement.

Discussion

RA is a small-vessel vasculitis that primarily affects the skin, joints, gastrointestinal tract, and kidneys. Worldwide, its incidence is 10 to 20 cases per 100,000 children per year [6,7].

Several factors are involved in the onset of rheumatoid purpura. These include the interaction of several genes and environmental factors, such as infections, medications, and vaccination [6].

The diagnosis of RA is primarily clinical, and additional testing is required, especially to look for complications.

Renal manifestations develop in 40 to 50% of patients [7,8]. The majority of these manifestations are seen within three months of disease onset. The most common renal involvement is microscopic or, rarely, macroscopic hematuria and acute glomerulonephritis [7-10].

Other manifestations include proteinuria, nephrotic syndrome, and acute nephritis with arterial hypertension, which are also possible [7,11,12].

Arterial hypertension is a complication described during the course of RA and is often associated with urinary abnormalities [13]. When isolated and without renal involvement, it is rare, with very few reported cases.

In the Saulsbury study [14], a series of 100 children with RA was documented, some of whom presented with transient minimal hypertension without associated urinary signs and which did not significantly influence the renal prognosis. Pillebout., *et al*. [15] reported an incidence of hypertension between 5% and 15%, which was correlated with significant proteinuria and severe glomerular involvement. In a retrospective study of 120 children, Feng., *et al*. [16] noted that hypertension was 15%, associated with a more severe progression to renal failure.

Naomi., *et al*. [17] studied 155 children. Among them, 30% presented with renal involvement during RA consisting of: hematuria or nephritis, nephrotic or nephritic syndrome. During their long-term follow-up: 7 children developed hypertension, among them: 5 had an initial nephritic or nephrotic syndrome, 1 had macroscopic hematuria, without initial hypertension, and 1 other had no renal or hypertension signs at baseline. In the case of Drummond., *et al*. [18], he reported the case of a girl with significant hypertension with minimal urinary abnormalities, whose explorations revealed one normal kidney and one almost destroyed kidney following reflux nephropathy, where the hypertension resolved after unilateral nephrectomy.

Four cases presented by Dubois., *et al*. [19], Whyte., *et al*. [20], Darteyre., *et al*. [21], and Pui Ling Thong., *et al*. [22] who presented hypertension without identifiable renal abnormalities and without secondary etiology found during exploration, as described in our cases, where the hypertension had resolved with the healing of the RA and under medical treatment.

The objective of our cases is to highlight that hypertension can occur without the classic signs of nephritis or renal failure in RA.

The question that arises is the mechanism responsible for this hypertension, after ruling out a secondary cause or vasculitis, hence the importance of a renal biopsy, which was not clinically indicated in our case.

Authors have put forward several hypotheses, such as: RA could lead to a worsening of renal pathologies, leading to severe hypertension; or the neurohormonal and vascular mechanism due to reflex vasoconstriction and activation of the renin-angiotensin-aldosterone system without glomerular lesions [23].

Hypertension in RA represents a major prognostic factor for progression to chronic renal failure, hence the importance of close monitoring.

Conclusion

Rheumatoid purpura is a benign condition; blood pressure monitoring and urine dipstick testing are essential due to the risk of kidney damage.

The discovery of hypertension in this disease requires the search for a secondary etiology and an assessment of the impact before determining essential hypertension.

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