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Renal scarring as a cause of secondary hypertension in a 4-year-old girl

Nadciśnienie tętnicze u 4-letniej dziewczynki wtórne do blizn nerkowych

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Abstract

We present the case of a 4-year-old girl diagnosed with secondary hypertension due to renal scarring. The patient was admitted to the Nephrology Clinic following an incidental finding of significantly elevated blood pressure (max. 203/136 mm Hg). Although there was no prior record of blood pressure measurements, the patient had reported occasional nosebleeds and abdominal pain. Diagnostic workup revealed elevated renin levels. Abdominal ultrasound showed kidney asymmetry and an irregular contour of the left kidney. Computed tomography angiography revealed no renal artery stenosis, but it showed multiple parenchymal narrowings in the left kidney, indicative of post-inflammatory scarring. Static scintigraphy confirmed these findings, showing impaired left kidney function (effective renal plasma flow – 20%). Furthermore, echocardiography revealed left ventricular hypertrophy. Blood pressure was normalised and clinical improvement was observed following combined antihypertensive therapy. This case underscores the importance of early diagnosis and regular blood pressure monitoring in children to prevent organ complications.

Keywords: hypertension, renal hypertension, urinary tract infections

Streszczenie

W pracy przedstawiono przypadek czteroletniej dziewczynki z nadciśnieniem wtórnym do bliznowacenia nerek. Pacjentka została przyjęta do Kliniki Nefrologii po wykryciu znacząco podwyższonych wartości ciśnienia krwi (maks. 203/136 mm Hg). W wywiadzie nie odnotowano wcześniejszych pomiarów ciśnienia tętniczego, natomiast pacjentka okresowo zgłaszała epizody krwawień z nosa oraz bóle brzucha. W toku diagnostyki z odchyleń stwierdzono istotnie podwyższone stężenie reniny. Ultrasonografia brzucha wykazała asymetrię wielkości nerek oraz nieregularny zarys torebki lewej nerki. W angiografii tomografii komputerowej jamy brzusznej nie uwidoczniono zwężeń tętnic nerkowych, stwierdzono jednak liczne zwężenia miąższu o morfologii blizn pozapalnych. Wyniki potwierdzono w scyntygrafii statycznej, ujawniając nierównomierne gromadzenie znacznika oraz obniżony udział nerki lewej w całkowitym wydzielaniu (procentowy udział efektywnego przepływu osocza przez nerki wyniósł 20%). Stwierdzono również cechy przerostu mięśnia sercowego w badaniu echokardiograficznym. Zastosowanie skojarzonej farmakoterapii hipotensyjnej doprowadziło do normalizacji ciśnienia tętniczego. Zaobserwowano również poprawę w badaniach kontrolnych. Przypadek podkreśla znaczenie wczesnej diagnostyki i systematycznego monitorowania ciśnienia tętniczego u dzieci w celu prewencji powikłań narządowych.

Słowa kluczowe: nadciśnienie, nadciśnienie nerkopochodne, zakażenia układu moczowego

INTRODUCTION

Paediatric hypertension (HT) affects approximately 4% of the general paediatric population worldwide[1], representing a major public health concern. It may give rise to various organ complications, including left ventricular hypertrophy, retinopathy, acute kidney injury, and neurological disorders[2]. Children usually present with secondary HT, with renal parenchymal diseases considered one of its leading causes[3,4]. Therefore, confirmation of the diagnosis and identification of the underlying aetiology are crucial for initiating appropriate therapy.

CASE REPORT

A 4-year-old girl was referred from the Department of Haematology to the Department of Paediatric Nephrology and Hypertension following an incidental diagnosis of HT. Diagnostic workup for elevated haemoglobin levels found markedly increased blood pressure, reaching up to 190/122 mm Hg. It was found from medical history that the patient was born at 38 weeks of gestation via caesarean section (due to maternal ventricular septal defect). She had 1-minute Apgar score of 8 and a birth weight of 2,835 g (3^{rd}–10^{th} percentile). Transient adaptive respiratory disorders were observed, which resolved after thermal stabilisation in the Neonatal Unit. Transfontanellar ultrasound (US) performed after birth was unremarkable. Abdominal US also showed no abnormalities, with both kidneys of normal size, structure and echogenicity.

At the age of two, the patient experienced recurrent episodes of abdominal pain, without accompanying fever or constipation. Urinalysis yielded normal results. Abdominal US performed during the diagnostic workup for abdominal pain demonstrated both kidneys of normal structure and length (approximately 60 mm), with no abnormalities in other abdominal organs. Furthermore, the mother reported periodic episodes of epistaxis for several months prior to hospital admission, without any associated infectious symptoms. Blood pressure had not been measured during previous paediatric check-ups. There was no family history of HT. Upon admission to the Department, the patient presented with significantly elevated blood pressure: 185/138 mm Hg in the left upper limb and 203/136 mm Hg in the right upper limb (average of three measurements). The patient exhibited no alarming symptoms and reported no pain. Physical examination revealed a heart murmur and overweight (body weight: 17.4 kg, 69^{th} percentile; height: 99 cm, 16^{th} percentile; body mass index: 17.75, 93^{rd} percentile). Peripheral pulses were normal and symmetrical in all four limbs. No other significant abnormalities were noted.

Peripheral blood count revealed mild thrombocytosis ($432 \times 10^3/\mu L$) with normal haemoglobin (12.7 g/dL) and leukocyte ($9.41 \times 10^3/\mu L$) levels. Inflammatory markers were not elevated (C-reactive protein: 0.12 mg/dL). Renal function parameters (creatinine: 0.33 mg/dL, urea: 17 mg/dL, cystatin C: 0.90 mg/L), serum electrolytes (Na: 137 mmol/L, K: 3.45 mmol/L, Ca: 2.60 mmol/L), triglycerides (55 mg/dL), cholesterol (179 mg/dL) and its fractions (LDL: 101 mg/dL, HDL: 67 mg/dL), glucose (85 mg/dL), uric acid (4.1 mg/dL), as well as TSH (2.549 µIU/mL) and thyroid hormone activity (FT4: 1.22 ng/dL) were within normal limits. Capillary blood gases were normal. Urinalysis and urine sediment showed no abnormalities.

Diagnostic workup was initiated to identify potential causes of secondary HT. A 24-hour urine collection excluded hypercortisolism. Methoxy-catecholamine metabolites (metanephrine: 90.9 µg/24 h, normetanephrine: 98.7 µg/24 h, 3-methoxytyramine: 41.9 µg/24 h) were not elevated. Renin and aldosterone were measured both before and after standing up. Elevated renin values were obtained both before (270.2 qIU/mL) and after (846 qIU/mL) standing up. Elevated aldosterone levels were observed in the upright posture (70.1 ng/dL).

Due to significantly elevated blood pressure in the child, the tests were run while she was on antihypertensive therapy with amlodipine, doxazosin, dihydralazine, and metoprolol. Angiotensin-converting enzyme inhibitors were not used prior to these measurements.

Abdominal US showed a size difference between the right kidney (RK) and the left kidney (LK): RK 7.7 cm (77^{th} percentile) vs. LK 6.7 cm (25^{th} percentile). The left renal capsule, with its uneven structure, was retracted in the lower pole. Renal resistive index (RI) at the level of the renal hilum could not be determined, whereas the measurements obtained from the intrarenal branches were within the reference range. The RI in the intrarenal branches was 0.61 in RK and 0.63 in LK.

A contrast-enhanced computed tomography (CT) angiography of the abdominal vessels was performed, revealing no evidence of stenosis in the thoracic or abdominal aorta or their major branches. A size discrepancy was observed between the right and left kidneys (RK: 8.0 cm – 87^{th} percentile; LK: 6.5 cm – lower limit of normal for age, 17^{th} percentile). Furthermore, a 3 mm cortical thinning was identified in the upper segment of the RK, and segmental cortical thinning were also noted in the LK, suggesting probable post-inflammatory changes.

Contrast-enhanced diffusion-weighted magnetic resonance imaging (DWI or DW-MRI) revealed segmental diffusion restriction in the cortex of the left kidney. Furthermore, both renal arteries, up to 2 mm in diameter, appeared relatively narrow yet without evidence of stenosis.

Static renal scintigraphy was also performed, revealing an irregular cortical outline of the LK. Other than that, the RK appeared normal. The effective renal plasma flow (%ERPF) was 20% for LK and 80% for RK. Voiding cystourethrography (VCUG) excluded vesicoureteral reflux and post-void urinary retention.

The girl was also evaluated for HT-induced target organ damage (TOD). Although electrocardiogram (ECG) showed no cardiac arrhythmias, signs of left ventricular

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hypertrophy were present. Echocardiography confirmed left ventricular hypertrophy, with a left ventricular mass (LVM) of 55.65 g and a left ventricular mass index (LVMI) of 57.18 g/m^2 (>95th percentile for age). Other cardiac imaging findings were unremarkable. Ophthalmologic fundus examination revealed no signs of retinopathy. Daily urine collections demonstrated mildly increased protein excretion (154 mg/24 h and 199.8 mg/24 h) in the absence of microalbuminuria. Brain MRI found no vascular malformations or occlusions of the major intracranial arterial trunks. A 4.5 × 2 mm non-contrast-enhancing lesion consistent with a malacic cavitation was visualised in the left genu of the corpus callosum. The findings were discussed with a neurologist, who recommended periodic follow-up MRI. A continuous infusion of hydralazine at 1 μg/kg/min was temporarily administered during hospital stay to normalise blood pressure. Long-term antihypertensive therapy was initiated with amlodipine (5 mg/day), doxazosin (2 mg/day), ramipril (2.5 mg/day), metoprolol tartrate (50 mg/day), and hydrochlorothiazide (12.5 mg/day), resulting in normalisation of home blood pressure measurements.

Adequate blood pressure control was confirmed during follow-up hospital admissions. No proteinuria or microalbuminuria was detected in 24-hour urine collections. A follow-up echocardiogram performed six months after treatment onset showed normalised cardiac parameters: LVM of 40.744 g, LVMI of 41.86 g/m^2 (<75th percentile for age). The patient remains under regular follow-up at the Nephrology Clinic.

DISCUSSION

Literature data indicate that secondary HT, which may arise from both congenital and acquired diseases of the renal parenchyma or urinary tract, is the predominant form of HT in younger children, particularly those under 6 years of age[3,5]. An analysis of 1,025 Polish children with HT showed that renal parenchymal diseases were the cause of secondary HT in 68% of patients, with significant renal scarring diagnosed in 50% of these cases[6]. Another study involving 57 patients with secondary HT identified renal scarring as the underlying cause in up to 78.9% of participants[7].

According to the recommendations of the Polish Society of Paediatric Nephrology, urinary tract infection (UTI) should be suspected in any child under 5 years of age who presents with a fever of unknown origin, as well as in children exhibiting symptoms suggestive of UTI. Such symptoms are often nonspecific and may vary by age. Fever may be the only manifestation of UTI particularly in the youngest patients. Other, less obvious signs that may suggest UTI include abnormal weight gain, decreased fluid intake, vomiting, or excessive sleepiness. If UTI is suspected, a urine sample should be collected for both urinalysis and culture prior to treatment initiation. Urine dipstick testing may be used as a preliminary diagnostic tool; however, any positive or equivocal result should be followed by further evaluation, including urine sediment analysis[8].

Renal parenchymal scarring is commonly observed in patients with recurrent UTIs, both in the context of congenital abnormalities and in the course of febrile UTIs[9]. Scars have also been identified in patients with atypical or subclinical UTIs, in whom symptoms were nonspecific and not consistently associated with fever[10,11]. A relationship has also been demonstrated between delayed antimicrobial therapy and renal scarring[12]. Early diagnosis of febrile episodes and/or abdominal pain in paediatric patients is crucial for preventing subsequent complications.

Vesicoureteral reflux (VUR) is a common urinary tract anomaly associated with renal scarring[9]. VUR may remain asymptomatic until UTI occurs. Studies have shown that low-grade reflux, particularly when diagnosed in early childhood, often resolves spontaneously[13]. By the time complications such as renal scarring are identified, VUR may no longer be detectable.

Paediatric HT can lead to multiple organ complications early in life. Adverse consequences of severe or untreated HT include alterations in the density and structure of retinal vessels (angiopathy), left ventricular hypertrophy (based on LVMI), and microalbuminuria[14]. At the time of diagnosis with stage II HT, our patient exhibited left ventricular hypertrophy, highlighting the need for regular cardiac monitoring and an interdisciplinary approach to diagnosis and management. Primary paediatric care should include routine blood pressure measurements in children from 3 years of age, particularly in those presenting with headaches, epistaxis, or atypical psychomotor behaviour. In addition to younger age, stage II HT, evidence of TOD, and the presence of chronic comorbidities are indications for extended diagnosis to identify secondary causes of HT[15].

CONCLUSIONS

In our patient, secondary HT related to renal parenchymal changes may have resulted from a subclinical UTI. However, the possibility of transient vesicoureteral reflux cannot be excluded. Unfortunately, despite the patient's age and presented symptoms, including epistaxis, blood pressure measurements were not performed prior to hospital admission. It is important to emphasise that early diagnosis enables prompt initiation of appropriate therapy and monitoring, which are crucial for preventing multiple complications.

Conflict of interest

The authors do not report any financial or personal associations with other persons or organisations that might affect the content of this publication of claim any rights hereto.

Author contributions

Original concept of study: PM, JZ, DON. Collection, recording and/or compilation of data; analysis and interpretation of data; writing of manuscript: PM. Critical review of manuscript; final approval of manuscript: JZ, DON.

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