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Prikaz slučaja
Case report

ARTERIAL HYPERTENSION AND KIDNEY DISEASE

MARIO PODRUG[1], NARCISSE NASRI[2], JELENA KOS[3], IVANA VUKOVIĆ-BRINAR[2,3], MARIO LAGANOVIĆ[2,4], BOJAN JELAKOVIĆ[2,3,5], SANDRA KARANOVIĆ ŠTAMBUK[2,3]

Arterial hypertension and chronic kidney disease are mutually interconnected. Uncontrolled arterial hypertension along with diabetes is one of the two most common causes of end-stage chronic kidney disease. On the other hand, chronic kidney disease, as well as renal artery stenosis, results in the development of arterial hypertension. Hypertensive crisis with target-organ damage, also known as a hypertensive emergency, is a serious arterial hypertension complication which is however becoming less frequent. It requires urgent parenteral antihypertensive treatment adjusted to the values of arterial hypertension and accompanying clinical signs. Damage can manifest on numerous organ systems primarily eyes, brain, cardiovascular system, and the kidneys. This is a case report of a patient who presented with hypertensive crisis and kidney damage and was eventually diagnosed with abdominal aortic dissection expanding into the renal arteries, but without typical clinical presentation of stabbing pain. MSCT aortography, despite the risk of progression of renal damage due to the effect of contrast agent, allowed us to set definitive diagnosis and clarify the aetiology of kidney damage and resistant arterial hypertension.

Keywords: ARTERIAL HYPERTENSION, AORTIC DISSECTION, CHRONIC KIDNEY DISEASE, KIDNEY DISEASE, RENAL ARTERY DISSECTION, RENAL ARTERY STENOS

INTRODUCTION

When Stephen Hales first measured the arterial blood pressure of a horse in 1733, he was certainly not aware of the fact that blood pressure in humans can be elevated and that in the future this would be a significant public health issue (1). Today, arterial hypertension (AH) is the main public health problem and leading factor in global morbidity. It is the main risk factor for heart, brain, and kidney disease. Chronic kidney disease is a complication arising from uncontrolled AH but is also frequently its cause. The interaction between AH and chronic kidney disease is complicated and increases the risk of adverse cardiovascular and cerebrovascular outcomes (2, 3). In 2015, standardized prevalence of hypertension in adults aged 18 and above was 22.1% (20.1% in women and 24.1% in men) (4). Among those, only 37% of men and 51% of women with AH knew they had AH. Among diagnosed hypertensives only 33% of women and 19% of men underwent antihypertensive treatment, and only 12% of women and 6% of men with AH had blood pressure values under control (5). The incidence of cardiovascular disease is significantly higher in patients with chronic kidney disease as opposed to the general population, and cardiovascular diseases have become the leading cause of death in patients with chronic kidney disease (6-8). The likelihood of adverse cardiovascular events increases with the progression of renal insufficiency, resulting in a 40-50 times higher risk of cardiovascular diseases in patients on haemodialysis compared to the general population (9). Here we present a case with arterial hypertension and renal insufficiency of unexpected aetiology.

[1]University of Split,
University Department of Health Studies
[2]School of Medicine, University of Zagreb
[3]Department of Nephrology, Arterial Hypertension, Dialysis and Transplantation,
University Hospital Center Zagreb
[4]Department of Nephrology,
University Hospital Merkur
[5]Croatian Academy of Sciences and Arts

Corresponding author:
Assistant professor,
Sandra Karanović Štambuk, MD. PhD.
Department of Nephrology, Arterial Hypertension, Dialysis and Transplantation,
University Hospital Center Zagreb
10000 Zagreb, Kišpatićeva ul. 12
E mail: skaranov@kbc-zagreb.h

CASE REPORT

A 45-year-old man arrived at the emergency room (ER) due to nausea, stomach-ache lasting 2 days and dull pain in the lumbar spine region. He had no prior serious illness besides occasionally elevated blood pressure levels for the last 5 years, however without treatment. He had an increased body mass with a body mass index of 28.3 kg/m2. Family history of both parents was positive for arterial hypertension (AH). Work up in the ER identified uncontrolled AH with blood pressure value of 250/150 mmHg and unremarkable status aside from mild pain in the epigastric region during stomach palpation. Furthermore, renal insufficiency of unknown duration with a creatinine level of 484 µmol/l (eGFR CKD EPI 12 ml/min/1,73 m2) and dipstick spot urine analysis positive for blood and 3+ proteins were detected. Additional findings included mild microcytic anaemia, borderline hypokalaemia, mixed liver lesion and mildly elevated inflammatory markers. Chest X ray revealed signs of enlarged heart shadow, acute stasis, inhomogeneous infiltrate of the middle right lobe and effusions in both phrenicocostal sinuses. Left ventricle hypertrophy was observed by electrocardiogram. Ophthalmological examination revealed changes of the fundus due to grade 3 arterial hypertension which included arterial narrowing with focal irregularities, small subretinal haemorrhages and both hard and soft exudates but no papilledema. Abdominal X ray was unremarkable, while the abdominal ultrasound showed normal size kidneys, but with hyperechogenic parenchyma; fatty liver with dilated hepatic veins, no ascites and the abdominal aorta was not visualized due to meteorism. Diagnosed with hypertensive crisis, the patient was hospitalized in the internal medicine department. After admission, antimicrobial therapy was initiated, AH was gradually abated with parenteral followed by per oral antihypertensive medication. Abdominal pain regressed immediately. However, diarrhoea occurred. Work up of potential secondary causes of AH and kidney disease that was perceived as chronic due to ultrasound findings and consequently resulting in no kidney biopsy, was performed simultaneously. Immunological tests came out negative. There were no signs of haemolysis. Fabry disease, dysproteinaemias, and infective aetiology (haemorrhagic fever viruses, leptospirosis) were ruled out as well. Cardiac echocardiography revealed heart failure with preserved ejection fraction most probably because of untreated AH. Doppler ultrasound of the renal arteries (RA) was performed twice, but unfortunately due to meteorism, it was unable to visualize the aorta and the ostia of the RA, intrarenal ramification was scarce while the occasionally visible intrarenal spectra had resistive indices of about 0.64. Pheochromocytoma was ruled out via negative urine metanephrines. Thyroid function was normal. There were no elements of obstructive sleep apnoea. Due to the risks of the contrast-induced nephropathy (CIN) a native multi-slice computed tomography (MSCT) of the thorax, abdomen and pelvis was performed, revealing borderline cardiomegaly, fatty liver, adenoma of the right adrenal gland, while the kidneys were of normal size but with a paler parenchyma. Given the difficulties in interpretation of hormonal findings in advanced renal damage and hypertensive crisis, an overnight dexamethasone test, along with aldosterone and renin as part of the testing for hypercortisolism and primary aldosteronism were not determined. It was concluded that the patient had long-term uncontrolled AH with target organ repercussions - primarily the kidney, heart, and the eyes and the patient was dismissed home. Nevertheless, to rule out RA stenosis and potentially improve renal function and AH control, and with as little exposure to nephrotoxic media as possible, one month later the patient was referred to a digital subtraction angiography (DSA) of the RA to perform diagnostics and intervention at the same time if needed. During the exam abdominal aortic dissection was suspected, resulting in the discontinuation of the DSA and immediate emergency MSCT aortography which confirmed the aortic dissection starting from the level of the originating point of the left subclavian artery with expansion to the proximal third of the external iliac artery on the right, and to the proximal third of the superficial femoral artery on the left, affecting the renal arteries as well, no significant signs of atherosclerosis were observed. Medical expert group including vascular and cardiac surgeons, interventional radiologists, cardiologists, and nephrologists, having in mind aortography finding and lack of any symptoms, advised conservative treatment and patient follow up. The angiology expert opinion was that that the dissection was caused by a long-term uncontrolled hypertension and that potential testing for hereditary connective tissue diseases was not necessary since patient's family medical history was negative, additionally he had no previous history of trauma, use of fluoroquinolone, and there were no typical signs of vasculitis. The patient is currently, a year after initial presentation, taking 8 antihypertensive drugs (trandolapril 4 mg, eplerenenone 2x25 mg, torasemide 20 mg, amlodipine 10 mg, moxonidine 0.6 mg, urapidil 3x60 mg, bisoprolol 10 mg, isosorbide mononitrate 3x20 mg) with a satisfactory blood pressure values and heart rate control, stable kidney function with an eGFR 15 ml/min/1,73 m2, still without need for renal replacement therapy.

DISCUSSION

Renal artery stenosis is one of the most common secondary causes of arterial hypertension, but also of renal failure of unknown origin (10-12). Its aetiology is most commonly atherosclerosis, however, it can also occur because of fibromuscular dysplasia, vasculitis, hereditary connective tissue diseases, as well as aortic dissection expanding into the renal arteries with the consequent renal hypoperfusion. The underlying mechanism is the activation of the renin-angiotensin-aldosterone system (13).

This case report highlights the association of arterial hypertension, kidney damage and a serious complication of uncontrolled hypertension - aortic dissection. By all odds, uncontrolled hypertension in this patient has led to aortic dissection, heart failure and retinal haemorrhage. The expansion of the aortic dissection into the renal arteries caused renal hypoperfusion resulting in kidney damage and the activation of the renin-angiotensin-aldosterone system that was supporting additional blood pressure elevation. The exact moment when the dissection occurred is not clear- whether it occurred at the time when the patient arrived at the emergency room or whether the onset of the dissection occurred earlier, for which there is evidence in the form of the already chronically altered renal parenchyma, remains in question. Namely, the typical clinical presentation of an aortic dissection includes unbearable stabbing pain in the chest/back, along with a pulse deficit, the murmur of aortic regurgitation in the case when the aortic valve is affected, focal neurologic deficit, hypotension, syncope, all of which our patient did not have. However, in about 6% of the cases an atypical, painless dissection is described (14). The gold standard for diagnosis of both renal artery stenosis and dissection is angiography, or aortography to be more precise. These examinations are most performed using

iodinated contrast media. In patients with impaired renal function there is additional risk of further renal function deterioration when applying intravenous iodinated contrast media due to the possibility of developing contrast induced nephropathy. The mechanism of CIN includes acute tubular necrosis caused by renal vasoconstriction and the resulting hypoxia of the medulla, and the direct cytotoxic effect of the contrast media on the tubular cells (15). The incidence of CIN in patients with chronic kidney disease varies between 4 and 44%, depending on the risk factors (16). CIN can be prevented by avoiding intravenous contrast examinations if they are not essential, by applying the minimum required amount of contrast media, by substantial periprocedural hydration and temporary cessation of interfering medications such as diuretics. In our patient we hesitated to use intravenous contrast diagnostics due to risk of CIN and attempted non-contrast methods to evaluate primarily the renal arteries (Doppler ultrasound), however this method was limited by the patient's constitution and meteorism, and this is also the reason why abdominal aorta was not visualized. Additionally, the native MSCT examination is also not a diagnostic method for either aortic dissection or stenosis/dissection of the renal arteries, especially while the dissected aorta is still of normal calibre. When intravenous contrast examination was finally carried out, the definitive diagnosis was established and the aetiology of kidney damage and cause of the worsening of the arterial hypertension was determined, which all resulted in patient receiving appropriate treatment and instructions how to continue life while lowering the chances of further complications. Although a large amount of contrast media was used, there was no renal function deterioration after the examination, most likely due to adequate periprocedural measur.

CONCLUSION

The case reported here had one of the most severe, and fortunately rarest complication of AH which led to the atypical hypertensive kidney damage. Although rare, this condition should be kept in mind. In case of clinical suspicion of such a condition, despite the risk of CIN, an angiography should be performed without delay.

Abbreviations:

AH - arterial hypertension
CIN - contrast-induced nephropathy
DSA - digital subtraction angiography
ER - emergency room
MSCT - multi-slice computed tomography
RA - renal arty

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Sažetak

ARTERIJSKA HIPERTENZIJA I BUBREŽNA BOLEST

Mario Podrug, Narcisse Nasri, Jelena Kos, Ivana Vuković-Brinar, Mario Laganović, Bojan Jelaković, Sandra Karanović Štambuk

Arterijska hipertenzija i kronična bubrežna bolest uzročno-posljedično su povezane. Neregulirana arterijska hipertenzija uz šećernu bolest jedan je od dva najčešća uzroka završnog stadija kronične bubrežne bolesti, a s druge strane, kronična bubrežna bolest, jednako kao i bolest bubrežnih arterija, dovodi do razvoja arterijske hipertenzije. Hipertenzivna kriza s oštećenjem ciljnih organa, takozvana hipertenzivna emergencija, ozbiljna je komplikacija arterijske hipertenzije koja se ipak sve rjeđa viđa. Iziskuje hitno parenteralno antihipertenzivno liječenje prilagođeno prema vrijednostima arterijskog tlaka i pridruženim kliničkim znakovima. Oštećenja se mogu manifestirati na brojnim organskim sustavima, prvenstveno očima, mozgu, kardiovaskularnom sustavu i bubrezima. Prikazan je slučaj bolesnika s hipertenzivnom krizom uz bubrežno oštećenje u kojeg se obradom izdiferencirala disekcija abdominalne aorte sa širenjem u renalne arterije, no bez tipične kliničke slike s parajućim bolovima. Učinjenom kontrastnom pretragom, unatoč riziku, postavljena je definitivna dijagnoza i razjašnjena je etiologija bubrežnog oštećenja i uzrok pogoršanja arterijske hipertenzije.

Ključne riječi: ARTERIJSKA HIPERTENZIJA, BUBREŽNA BOLEST, DISEKCIJA AORTE, DISEKCIJA RENALNE ARTERIJE, KRONIČNA BUBREŽNA BOLEST, STENOZA RENALNE ARTERIJE

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