Marijana C. Jandrić Kočić¹, Snežana B. Knežević²

ZNAČAJ/ULOGA AMBULANTE PORODIČNE MEDICINE U RANOM OTKRIVANJU SEKUNDARNE HIPERTENZIJE

Prikaz slučaja

SAŽETAK – Uvod: Sekundarna arterijska hipertenzija ima osnovni uzrok koji je moguće identifikovati. Rutinski skrining iste nije indikovano imajući u vidu nisku prevalenciju bolesti (5-10% arterijske hipertenzije), dugotrajnu i skupu dijagnostičku evaluaciju.

Prikaz slučaja: U ambulantu porodične medicine javlja se pacijentkinja starosti 34. godine zbog pogoršanja, do tada stabilne, arterijske hipertenzije. Ista joj je ustanovljena prije 12 mjeseci kojom prilikom se javila u zdravstvenu ustanovu gdje joj je uključen perindopril/amlodipin 4/5 mg, 1x1 tableta. Do sada zdrava, negira bolesti od značaja za hereditet. Pušač. 24 satni ambulantni monitoring krvnog pritiska verifikuje povišene vrijednosti dijastolnog krvnog pritiska u 59,3% mjerenja u toku dana i 59,2% mjerenja u toku noći. Ultrazvuk štitne žlijezde verifikuje nehomogenu strukturu, desni režanj veličine 40x15x16 mm, lijevi režanj 42x15x16 mm. Ultrazvuk abdomena smanjenje lijevog bubrega, bilateralno istanjen korteksa, lijevostranu umjerenu hidronefrozu. Laboratorijski nalazi utvrđuju značajno povećanje vrijednosti tirostimulirajućeg hormona kao i antitijela prema tiroidnoj peroksidazi, snižene vrijednosti slobodnog tiroksina i lagano povišene vrijednosti albumina u 24 satnom urinu. Pacijentkinja se upućuje na konsultativni pregled nefrologa i specijaliste nuklearne medicine. Isti indikuju higijensko dijetetski režim i uvođenje levotiroksin natrijum tableta 100 mcg 1x 1 ¼ tablete (125 mcg). Šest mjeseci kasnije ukinuta je antihipertenzivna terapija, dok je supstitucionu terapiju levotiroksin natrijumom smanjena na 1 x 100 mcg.

Zaključak: Rad izabranog ljekara porodične medicine u skladu sa smjernicama dobre kliničke prakse omogućava rano otkrivanje, normalizaciju

¹ Marijana C. Jandrić-Kočič, Dom zdravlja Krupa na Uni, Republika Srpska, marijanajandric-kocic@gmail.com

² Dom zdravlja Kraljevo, Republika Srbija

ili značajno smanjenje vrijednosti sekundarne hipertenzije, redukuju mogućnost nastanka ireverzibilnih promjena na krvnim sudovima i koegzistirajuće esencijalne hipertenzije.

Ključne riječi: sekundarna hipertenzija, porodična medicina

Uvod

Arterijska hipertenzija predstavlja najučestaliji i najslabije kontrolisan faktor rizika smrtnosti u svijetu. Komplikacije arterijske hipertenzije (infarkt miokarda, moždani udar, srčana isuficijencija, periferna vaskularna bolest, oštećenje bubrega i vida) dovode se u vezu sa 10,4 miliona smrtnih ishoda godišnje. Svake godine na liječenje arterijske hipertenzije u Sjedinjenim Američkim Državama se troši 47,5 milijardi dolara [1,2].

U 90-95% slučajeva arterijska hipertenzija nema poznatu etiologiju i označava se kao esencijalna, primarna ili idiopatska. Riječ je o multifaktorijskoj bolesti koja nastaje kao posljedica kombinovanog uticaja genetskih faktora, načina života, faktora okoline, poremećaja u kontrolnim neurohumoralnim mehanizmima i vaskularnoj strukturi [1,2,3].

Sekundarna arterijska hipertenzija ima osnovni uzrok koji je moguće identifikovati. Rana detekcija i adekvatno kauzalno liječenje ove hipertenzije omogućavaju normalizaciju ili značajno smanjenje vrijednosti krvnog pritiska, čime se i redukuje mogućnost nastanka ireverzibilnih promjena na krvnim sudovima i trajno održavanje/zaostajanje koegzistirajuće esencijalne hipertenzije [3,4].

Prikaz slučaja

U ambulantu porodične medicine javila se pacijentkinja starosti 34. godine zbog progresivnog pogoršanja arterijske hipertenzije. Hipertenzija je otkrivena 12 mjeseci ranije i započeto je kontinuirano liječenje (perindopril/amlodipin 4/5 mg/ dnevno). Do tada je bila zdrava i negirala je bolesti od značaja za hereditet. Živi sama u uslovnom stanu, puši.

Fizikalnim pregledom nisu nađeni znaci koji bi upućivali na sekundarnu hipertenziju. Pacijentkinja je bila normalne osteomuskularne građe i uhranjenosti (BMI 22,1 kg/m²), uobičajene kosmatosti ženskog tipa, a štitasta žlijezda normalne veličine i građe. Uz normalan auskultatorni nalaz na srcu, izmeren je krvni pritisak 160/98 mmHg, srčana frekvencija 100/min, a pO₂ je bio 97% meren pulsni oksimetrom. Nije bilo otoka na nogama.

Ultrasonografija štitaste žlijezde je pokazala da su oba režnja uredne veličine, blago nehomogene i hipoeogene strukture.

Ultrasonografija abdomena je utvrdila normalan oblik i položaj oba bubrega. Lijevi bubreg je bio nešto manjih dimenzija sa hidronefrozom 2. stepena. Nadbubrežne žlijezde nisu vizualizovane.

24 satni ambulantni monitoring krvnog pritiska Srednja vrijednost krvnog pritiska tokom 24 satnog ambulantnog monitoringa iznosi 113/79, gornja granična vrijednost 24 satnog dijastolnog krvnog pritiska. Srednja vrijednost krvnog pritiska tokom dana iznosi 118/85, gornja granična vrijednost dijastolnog krvnog pritiska u toku dana. Prosječna vrijednost krvnog pritiska tokom noći iznosi 104/69, gornja granična vrijednost dijastolnog krvnog pritiska u toku noći. U 59,3% mjerenja u toku dana zabilježena je povišena vrijednost dijastolnog krvnog pritiska. Povišena vrijednost dijastolnog krvnog pritiska zabilježena je u 52,9% noćnih mjerenja. Povišena vrijednost sistolnog krvnog pritiska zabilježena je u 6,8% mjerenja u toku dana. U toku noći nije zabilježeno povećanje sistolnog krvnog pritiska. Prosječna frekvencija rada srca u toku dana je bila normalna, 85/min i, u toku noći 69/min i uredna je. Verifikovan je fiziološki pad pritiska u toku noći (sistolni 12%, dijastolni 18,8%) (slika 1).

Slika 1. 24 satni ambulantni monitoring krvnog pritiska pacijentkinje

Elektrokardiogram je bio uredan.

Laboratorijski nalazi su utvrdili povišene vrijednosti antitijela na tiroidnu peroksidazu i tiroksin stimulirajućeg hormona, kao i nižu vrijednost tiroksina. Nisu uočena odstupanja u koncentraciji glukoze, elektrolita, azotnih materija i lipidnom statusu. Klirens kreatinina je bio diskretno povišen. Vrijednosti albumina i ukupnih proteina u 24 satnom urinu su bile uredne.

Zbog nalaza tiroidnih hormona koji upućuju na hipotireozu zbog hroničnog tiro-
iditisa, pacijentkinja je upućena na konsultativni pregled kod nefrologa i specijaliste
nuklearne medicine. Uz higijensko dijetetski režim uvedena je terapija levotiroksin
125 mcg. Šest mjeseci kasnije ukinuta je antihipertenzivna terapija zbog redukcije
krvnog pritiska, dok je supstituciona terapija levotiroksinom 100 mcg.

Diskusija

Rutinski skrining sekundarne hipertenzije nije indikovani imajući u vidu nisku
prevalenciju bolesti, dugotrajnu i skupu dijagnostičku evaluaciju. Samim tim je zna-
čajno u inicijalnoj procjeni identifikovati stanja koja zahtijevaju skrining: rani početak
arterijske hipertenzije (<40 godina) u bolesnika bez drugih faktora rizika (porodične
anamneze, pretilosti itd.), hipertenzija u prepubertalne djece, otporna hipertenzija
(> 140/90 mmHg upkos primjeni tri antihipertenzivna lijeka, uključujući diuretik),
teška hipertenzija (> 180/110 mmHg) ili hipertenzivna kriza, nagli porast krvnog pri-
tiska kod ranije stabilnog pacijenta, odsustvo fiziološkog pada krvnog pritiska u toku
noći (u toku 24 satnog ambulantnog monitoringa krvnog pritiska), prisutnost oštećenja
ciljnih organa (hipertrofija lijeve komore, hipertenzivna retinopatija itd.), prisustvo
simptoma i znakova koji ukazuju na sekundarne uzroke arterijske hipertenzije [3,4].

Procjena sekundarne hipertenzije je indikovana i nakon uvođenja terapije ukoliko
je prisutno: prekomjerno smanjenje kalija s malom dozom diuretika, prekomjerno
smanjenje glomerularne filtracije s malom dozom inhibitora angiotenzin konvertu-
jućeg enzima, izrazito rezistentna arterijska hipertenzija, pretjerana labilnost krvnog
pritiska uprkos inicijalnom smanjenju [3,4].

Detekciji sekundarne hipertenzije nerijetko zahtjeva odbacivanje hipertenzije
bijelog mantila, potvrdu prisustva rezistentne hipertenzije, procjenu terapijske kompli-
janse i prisustva fiziološkog pada krvnog pritiska u toku noći, a samim tim i primjenu
24 satnog ambulantnog monitoringa krvnog pritiska [3].

U dijagnostici sekundarne hipertenzije važno je isključiti uticaj brojnih lekova
na krvni pritisak uključujući: nesteroidne antiinflamatorne lijekove, karbamazepin,
klozapin, inhibitore monoamino oksidaze, selektivne inhibitori ponovnog preuzimanja
serotonina-norepinefrina, triciklične antidepresive, oralne kontraceptive, prednizon,
metilprednizolon, dekongestive, lijekove za smanjenje tjelesne težine. Neophodno je

i ukloniti uticaj režim ishrane koji potencira hipertenziju (konzumacija natrijuma $\geq$ 2.4 grama dnevno, slatkog luka $\geq$ 3 grama dnevno, alkohola $\geq$ 300 gr nedeljno) [5].

Etiologija sekundarne hipertenzije uključuje: koarktaciju aorte (djeca uzrasta $<$ 11 godina i adolescenti), aterosklerotsku stenožu bubrežne arterije (uzrast 65 i više godina), bubrežnu parenhimsku bolest (uzrast 19-39 godina), feohromocitom (uzrast 40-46 godina), hiperaldosteronizam, Kušingov sindrom, disfunkciju štitne žlijezde, fibromuskularnu displaziju, opstruktivnu apneju za vrijeme spavanja [3,5].

Simptomi navedenih oboljenja mogu se utvrditi pravilno uzetom anamnezom, fizikalnim i ultrazvučnim pregledom i/ili standardizovanim upitnicima (Epworth Sleepiness Scale, Billewicz diagnostic index). [7,8,9,10].

Izabrani ljekar porodične medicine ima značajnu ulogu u otkrivanju i lečenju sekundarne hiperetenzije. Ciljanom anamnezom, fizikalnim pregledom, ultrazvučnim pregledom uz primenu standardizovanog upitnika može se postaviti dijagnozu sekundarne hipertenzije i pacijenta uputiti u referentnu zdravstenu ustanovu. Prije upućivanja, od velike koristi je i EKG pregled koji obavlja. U zavisnosti od tehničkih mogućnosti i etiologije sekundarne hipertenzije, izabrani ljekar porodične medicine obavlja ultrasonografski pregled abdomena, štitne žlijezde, radiografiju grudnog koša (prisustvo morfoloških promjene štitne žlijezde, bubrega, nadbubrežne žlijezde i krvnih sudova), 24 satni ambulantni monitoring krvnog pritiska, ciljane diferencijalne laboratorijske analize (24 satnu natriuriju i kaliuriju, proteine u 24 satnom urinu, mikroalbuminurija, glomerularnu filtraciju, albumine, ukupne proteine, jonogram, omjer albumin/kretinin, hormone štitne žlijezde). Takođe učestvuje u adekvatnoj pripremi pacijenta za analizu metanefrina i normetanefrina u 24-satnom urinu i određivanju aldosterona i reninske aktivnosti plazme [3,6,7,11].

Prikazanoj pacijentkinji utvrđena je hipotireoza koja predstavlja značajan i često neprepoznat uzrok sekundarne arterijske hipertenzije (predominantno sekundarne dijastolne hipertenzije) kao posljedice povećanja arterijskog otpora, smanjenja kontraktilnosti srca, smanjenja brzine glomerularne filtracije, abnormalnog metabolizma natrijuma, aktivacije simpatičkog nervnog sistema i endotelne disfunkcije [4]. Rad izabranog ljekara porodične medicine u skladu sa smjernicama dobre kliničke doveo je do ranog otkrivanja i uspješnog liječenja sekundarne hipertenzije kojim se minimiziraju/spriječavaju nepovratne promjene u sistemske vaskulaturi i trajna hipertenzija s nepovoljnim dugoročnim ishodom.

Zaključak

Rana detekcija i kvalitetan menadžment sekundarne hipertenzije u ambulantama porodične medicine značajno bi smanjili funkcionalnu onesposobljenost, troškove zdravstvene zaštite i gubitak prihoda zbog izgubljene produktivnosti.

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Marijana C. Jandrić Kočić¹, Snežana B. Knežević²

SIGNIFICANCE/ROLE OF FAMILY MEDICINE PHYSICIANS IN EARLY DETECTION OF SECONDARY HYPERTENSION

Case report

SUMMARY – Introduction: Secondary arterial hypertension has an identifiable underlying cause. Routine screening is not indicated given the low prevalence of the disease (5-10% arterial hypertension), long-term and costly diagnostic evaluation.

Case report: An outpatient family medication presents a 34-year-old patient due to worsening, by then stable, arterial hypertension. She was found 12 months ago when reported to a private healthcare facility where she was allowed perindopril / amlodipine 4/5 mg, 1x1 tablet. So far healthy, it negates diseases of relevance to inheritance. Smoker. 24-hour outpatient blood pressure monitoring checks for elevated diastolic blood pressure levels in 59,3% of measurements during the day and 59,2% of measurements during the night. Thyroid ultrasound checks for inhomogeneous structure, right flap 40x15x16 mm, left flap 42x15x16 mm. Abdominal ultrasound reduces left kidney, bilateral thinning cortex, left ventricular moderate hydronephrosis. The laboratory contains large amounts of tyrosimulating hormone as well as antibodies to thyroid peroxidase, decreased levels of free thyroxine and a slight increase in albumin in 24 hours of urine. The patient is referred for a consultative examination by a nephrologist and a nuclear medicine specialist. Same indicative hygiene dietary regimen and introduction of levothyroxine sodium tablets 100 mcg 1x 1 ¼ tablets (125 mcg). Antihypertensive therapy was discontinued at most months later, while levothyroxine sodium replacement therapy was reduced to 1 x 100 mcg.

Conclusion: The work of a selected family physician in accordance with good clinical practice guidelines allows for the early detection,

¹ Marijana C. Jandrić Kočić, Primary Health care Center Krupa na Uni, Republic of Srpska, marijanajandrickocic@gmail.com

² Primary Health care Center Kraljevo, Republic of Serbia

normalization or increase in the number of secondary hypertension, the reduction of the possibility of accommodation of irreversible changes in blood vessels, and coexisting essential hypertension.

Keywords: secondary hypertension, family medicine

Introduction

Arterial hypertension is the most common and the least controlled risk factor for mortality in the world. Complications of arterial hypertension (myocardial infarction, stroke, heart failure, peripheral vascular disease, kidney and vision impairment) have been linked to 10.4 million deaths annually. Every year, \$ 47.5 billion is spent on the treatment of arterial hypertension in the United States [1,2].

In 90-95% of cases, arterial hypertension has no known etiology and is marked as essential, primary or idiopathic. It is a multifactorial disease that occurs as a result of the combined influence of genetic factors, lifestyle, environmental factors, disorders in the control neurohumoral mechanisms and vascular structure [1,2,3].

Secondary arterial hypertension has an identifiable underlying cause. Early detection and adequate causal treatment of this hypertension enable normalization or significant reduction of blood pressure, which reduces the possibility of irreversible changes in blood vessels and permanent maintenance / delay of coexisting essential hypertension [3,4].

Case report

A 34-year-old female patient came to the family medicine clinic due to the progressive worsening of arterial hypertension. Hypertension was detected 12 months earlier and continuous treatment was started (perindopril/amlodipine 4/5 mg/day). Until then, she was healthy and denied diseases of importance for heredity. She lives alone in a conditional apartment, she smokes.

Physical examination revealed no signs of secondary hypertension. The patient had normal osteomuscular build and nutrition (BMI 22.1 kg/m²), normal female hairiness, and a thyroid gland of normal size and build. With a normal auscultatory finding on the heart, blood pressure was measured 160/98 mmHg, heart rate 100/min, and pO₂ was 97% measured with a pulse oximeter. There was no swelling on his feet.

Ultrasonography of the thyroid gland showed that both lobes were of orderly size, slightly inhomogeneous and hypoechoic in structure.

Abdominal ultrasonography determined the normal shape and position of both kidneys. The left kidney was somewhat smaller with grade two hydronephrosis. The adrenal glands are not visualized.

24-hour ambulatory blood pressure monitoring: The mean value of blood pressure during 24-hour ambulatory monitoring was 113/79 mm Hg, the upper limit of 24-hour diastolic blood pressure. The mean value of blood pressure during the day was 118/85 mm Hg, the upper limit of diastolic blood pressure during the day. The average value of blood pressure during the night was 104/69 mm Hg, the upper limit of diastolic blood pressure during the night. Elevated diastolic blood pressure was noted in 59.3% of measurements during the day. Elevated diastolic blood pressure was noted in 52.9% of nocturnal measurements. Elevated systolic blood pressure was recorded in 6.8% of measurements during the day. No increase in systolic blood pressure was observed during the night. The average heart rate during the day was normal, 85/min and, during the night 69/min and was normal. Physiological pressure drop during the night was verified (systolic 12%, diastolic 18.8%) (Figure 1).

Figure 1. 24-hour ambulatory monitoring of the patient's blood pressure

The electrocardiogram was in order.

Laboratory findings revealed elevated levels of antibodies to thyroid peroxidase and thyroxine-stimulating hormone, as well as lower levels of thyroxine. No deviations in glucose, electrolyte, nitrogen, and lipid status were observed. Creatinine clearance was discretely elevated. Albumin and total protein values in 24-hour urine were normal. Due to the findings of thyroid hormones that indicated hypothyroidism due to chronic thyroiditis, the patient was referred for a consultative examination by a nephrologist and a specialist in nuclear medicine. In addition to the hygienic diet regime, levothyroxine 125 mcg per day therapy was introduced. Six months later, antihypertensive therapy was discontinued to reduce blood pressure, while levothyroxine replacement therapy was 100 mcg.

Discussion

Routine screening for secondary hypertension is not indicated according to the low prevalence of the disease, lengthy and costly diagnostic evaluation. Therefore, it is important in the initial assessment to identify conditions that require screening: early onset of arterial hypertension (<40 years) in patients without other risk factors (family history, obesity, etc.), hypertension in prepubertal children, resistant hypertension (>140/90 mm Hg despite the use of three antihypertensive drugs, including diuretics), severe hypertension (>180/110 mm Hg) or hypertensive crisis, sudden increase in blood pressure in a previously stable patient, absence of physiological drop in blood pressure during the night (during 24-hour ambulatory blood pressure monitoring), the presence of damage to target organs (left ventricular hypertrophy, hypertensive retinopathy, etc.), and the presence of symptoms and signs indicating secondary causes of arterial hypertension [3,4].

Assessment of secondary hypertension is indicated after initiation of therapy if present: excessive potassium reduction with low dose diuretics, excessive decrease in glomerular filtration with low dose angiotensin converting enzyme inhibitors, extremely resistant arterial hypertension and excessive lability and blood pressure reduction [3,4].

Detection of secondary hypertension often requires rejection of white coat hypertension, confirmation of the presence of resistant hypertension, assessment of therapeutic compliance and the presence of physiological drop in blood pressure during the night, and thus the application of 24-hour ambulatory blood pressure monitoring [3].

In the diagnosis of secondary hypertension, it is important to exclude the effect of a number of drugs on blood pressure including: non-steroidal anti-inflammatory drugs, carbamazepine, clozapine, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, tricyclic antidepressants, antidepressants, oral contraceptives, drugs for reducing body weight. It is also necessary to eliminate the influence of a

diet that potentiates hypertension (sodium consumption ≥ 2.4 grams per day, sweet onions ≥ 3 grams per day, alcohol ≥ 300 g per week) [5].

The etiology of secondary hypertension includes: aortic coarctation (children < 11 age and adolescents), atherosclerotic renal artery stenosis (age 65 and older), renal parenchymal disease (age 19-39), pheochromocytoma (ages 40-46), hyperaldosteronism, Cushing's syndrome, thyroid dysfunction, fibromuscular dysplasia and obstructive sleep apnea [3,5]. Symptoms of these diseases can be determined by a proper medical history, physical and ultrasound examination and/or standardized questionnaires (Epworth Sleepiness Scale, Billewicz diagnostic index) [7,8,9,10].

The family doctor has a significant role in the detection and treatment of secondary hypertension. Targeted anamnesis, physical examination, ultrasound examination with the use of a standardized questionnaire can diagnose secondary hypertension and refer the patient to a secondary health institution. Before the referral, an electrocardiogram is useful. Depending on the technical possibilities and etiology of secondary hypertension, the family doctor performs ultrasonographic examination of the abdomen, thyroid gland, chest radiography (presence of morphological changes of the thyroid gland, kidneys, adrenal glands and blood vessels), 24-hour ambulatory blood pressure monitoring, laboratory analyzes (the 24 hour urin sodium and potassium, 24-hour urine protein test, microalbuminuria, glomerular albumin filtration, total proteins rage, blood ionogram, albumin/creatinine ratio, thyroid hormones). Family physician also participates in adequate patient preparation for analysis 24-hour urine metanephrine and normetanephrine and determination of aldosterone and plasma renin activity [3,6,7,11].

The presented patient was diagnosed hypothyroidism, which is a significant and often unrecognized cause of secondary arterial hypertension (predominantly secondary diastolic hypertension) as a consequence of increased atrial resistance, decreased cardiac contractility, decreased glomerular filtration rate, abnormal sympathetic metabolism and endothelial system [4]. The work of family physician in accordance with good clinical guidelines has led to early detection and successful treatment of secondary hypertension which minimizes/prevents irreversible changes in vasculare system and persistent hypertension with unfavorable long-term outcome.

Conclusion

Early detection and quality management of secondary hypertension in family medicine significantly reduce functional disability, health care costs, and loss of income due to lost productivity.

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