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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,

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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.

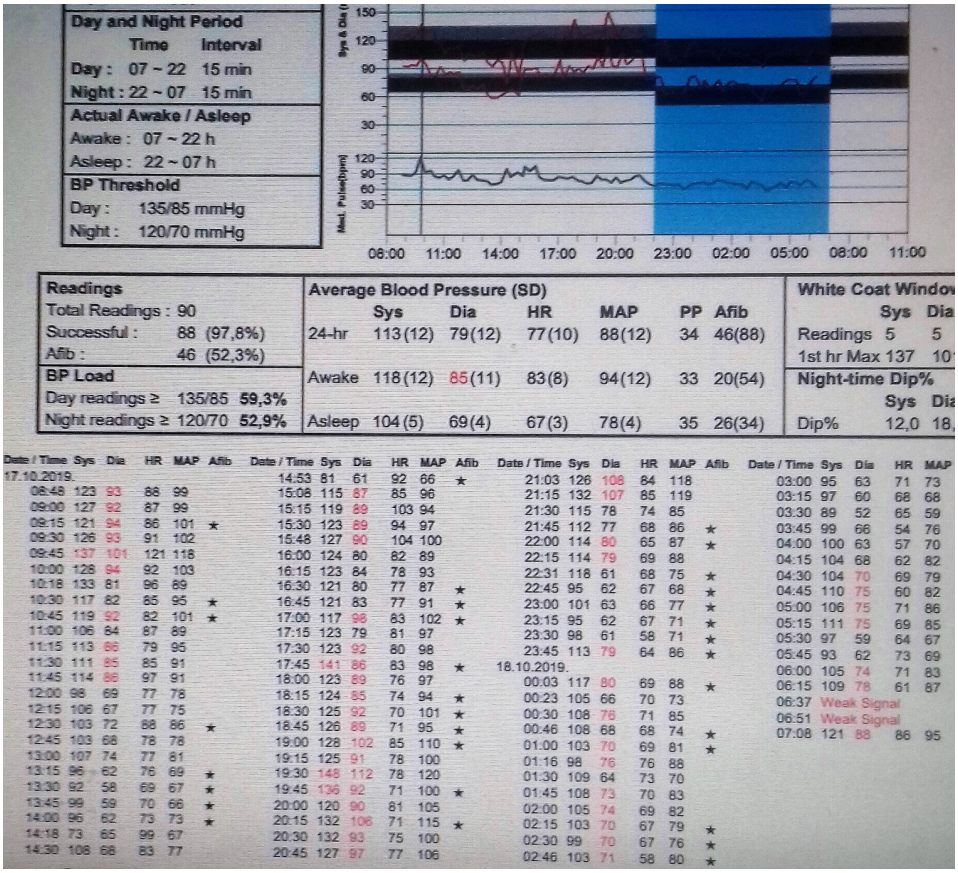
Physical examination revealed no signs of secondary hypertension. The patient had normal osteomuscular build and nutrition (BMI 22.1 kg/m2), normal female hairiness, and a thyroid gland of normal size and build. With a normal auscultatory finding on the heart, blood pressure was measured 160/98 mmHg, 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



Day and Night Period

	Time	Interval
Day :	07 ~ 22	15 min
Night :	22 ~ 07	15 min

Actual Awake / Asleep

Awake : 07 ~ 22 h
Asleep : 22 ~ 07 h

BP Threshold

Day : 135/85 mmHg
Night : 120/70 mmHg

Readings

Total Readings :	90	
Successful :	88	(97,8%)
Afib :	46	(52,3%)

BP Load

Day readings ≥	135/85	59,3%
Night readings ≥	120/70	52,9%

Average Blood Pressure (SD)

	Sys	Dia	HR	MAP	PP	Afib
24-hr	113 (12)	79 (12)	77 (10)	88 (12)	34	46(88)
Awake	118 (12)	85 (11)	83 (8)	94 (12)	33	20(54)
Asleep	104 (5)	69 (4)	67 (3)	78 (4)	35	26(34)

White Coat Windo

	Sys	Dia
Readings	5	5
1st hr Max	137	10

Night-time Dip%

	Sys	Dia
Dip%	12,0	18,

Date / Time	Sys	Dia	HR	MAP	Afib
17.10.2019.					
08:48	123	93	88	99	
09:00	127	92	87	99	
09:15	121	94	86	101	★
09:30	126	93	91	102	
09:45	137	101	121	118	
10:00	128	94	92	103	
10:18	133	81	96	89	
10:30	117	82	85	95	★
10:45	119	92	82	101	★
11:00	106	64	87	89	
11:15	113	86	79	95	
11:30	111	85	85	91	
11:45	114	86	97	91	
12:00	98	69	77	78	
12:15	106	67	77	75	
12:30	103	72	88	86	★
12:45	103	68	78	78	
13:00	107	74	77	81	
13:15	96	62	76	69	★
13:30	92	58	69	67	★
13:45	99	59	70	66	★
14:00	96	62	73	73	★
14:18	73	65	99	67	
14:30	108	68	83	77	
14:53	81	61	92	66	★
15:08	115	87	85	96	
15:15	119	89	103	94	
15:30	123	89	94	97	
15:48	127	90	104	100	
16:00	124	80	82	89	
16:15	123	84	78	93	
16:30	121	80	77	87	★
16:45	121	83	77	91	★
17:00	117	98	83	102	★
17:15	123	79	81	97	
17:30	123	92	80	98	
17:45	141	86	83	98	★
18:00	123	89	76	97	
18:15	124	85	74	94	★
18:30	125	92	70	101	★
18:45	126	89	71	95	★
19:00	128	102	85	110	★
19:15	125	91	78	100	
19:30	148	112	78	120	
19:45	136	92	71	100	★
20:00	120	90	81	105	
20:15	132	106	71	115	★
20:30	132	93	75	100	
20:45	127	97	77	106	
21:03	126	108	84	118	
21:15	132	107	85	119	
21:30	115	78	74	85	
21:45	112	77	68	86	★
22:00	114	80	65	87	★
22:15	114	79	69	88	
22:31	118	61	68	75	★
22:45	95	62	67	68	★
23:00	101	63	66	77	★
23:15	95	62	67	71	★
23:30	98	61	58	71	★
23:45	113	79	64	86	★
18.10.2019.					
00:03	117	80	69	88	★
00:23	105	66	70	73	
00:30	108	76	71	85	
00:46	108	68	68	74	★
01:00	103	70	69	81	★
01:16	98	76	76	88	
01:30	109	64	73	70	
01:45	108	73	70	83	
02:00	105	74	69	82	
02:15	103	70	67	79	★
02:30	99	70	67	76	★
02:46	103	71	58	80	★
03:00	95	63	71	73	
03:15	97	60	68	68	
03:30	89	52	65	59	
03:45	99	66	54	76	
04:00	100	63	57	70	
04:15	104	68	62	82	
04:30	104	70	69	79	
04:45	110	75	60	82	
05:00	106	75	71	86	
05:15	111	75	69	85	
05:30	97	59	64	67	
05:45	93	62	73	69	
06:00	105	74	71	83	
06:15	109	78	61	87	
06:37	Weak Signal				
06:51	Weak Signal				
07:08	121	88	86	95	

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.

Disscusion

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 diagnosic 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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