МЕДИЦИНА

DOI https://doi.org/10.32689/2786-7161-2026-2-1
UDC 616.12-008.331.1:616.831-005.1/.12]-06:616.441-008.61

THE ROLE OF ARTERIAL HYPERTENSION IN THE FORMATION OF CEREBRAL HEMODYNAMIC DISORDERS IN PATIENTS WITH THYROTOXICOSIS

Tetiana Bentsa, Oksana Pastukhova, Iryna Tsymbaliuk

Abstract

Background. *This article examines the characteristics of cerebral haemodynamics in patients with thyrotoxicosis (TT), both with and without concomitant arterial hypertension (AH), and aims to determine the role of these factors in the development of haemodynamic disturbances.* ***Materials and Methods.*** *A total of 100 patients with TT, AH, and TT combined with AH, as well as 27 essentially healthy individuals, were examined. Using the transcranial duplex scanning method, we measured and analysed the linear blood flow velocity (LBFV) in intracranial vessels and the extracranial segments of the brachiocephalic vessels, the pulsatility index (PI), the resistance index (RI) and the total cerebral blood flow as the sum of the volumetric blood flow through both internal carotid arteries and the volumetric blood flow through both vertebral arteries.* ***Results.*** *In patients with TT, a statistically significant increase in LBFV, IR and IP were observed in the extra- and intracranial segments of the major arteries of the head and neck, which was more pronounced in patients with concomitant AH; this may indicate greater strain on haemodynamic compensatory mechanisms, marked dysregulation of vascular tone, and may complicate the course of the underlying disease in these patients. In the group of patients with TT and AH, a significant increase in total cerebral blood flow was also observed, suggesting that AH acts as an additional factor placing strain on the mechanisms compensating for cerebral blood flow, which may lead to their premature exhaustion.* ***Conclusions.*** *The results obtained suggest that AH should be regarded as an additional factor that significantly impairs cerebral haemodynamics, increases the risk of cerebrovascular complications and may lead to the rapid development of metabolic cerebral disorders in patients with TT.*

Key words: *arterial hypertension, thyrotoxicosis, cerebral hemodynamic, ultrasound duplex scan.*

1. Introduction

Arterial hypertension (AH) is the most common cardiovascular condition in the world. In 2024, experts estimated that 1.4 billion people aged between 30 and 79 were living with AH worldwide [6]. Undiagnosed and uncontrolled AH remains one of the leading preventable causes of heart attack, stroke, chronic kidney disease and dementia [2, 7]. The thyroid gland (TG) is one of the main regulatory organs of haemostasis in the human body, controlling the functioning of numerous systems. Its hormones have a modulating effect, in particular on cardiac function and the peripheral vascular system, ensuring optimal functioning within the normal range [5, 13]. Disorders of thyroid function are associated with an increased risk of cardiovascular disease (CVD) through alterations in cardiac contractility, stroke volume, heart rate, peripheral vascular resistance and electrical activity [19]. According to recent studies, even moderate changes in thyroid hormone levels increase mortality from CVD by between 20% and 80% [14].

AH and thyrotoxicosis (TT) are closely interrelated conditions. TT, which is a combination of signs and symptoms arising from an excess of circulating thyroid hormones, leads to an increase in cardiac output, systolic and diastolic endothelial and left ventricular dysfunction, and a reduction in systemic vascular resistance, thereby increasing circulating blood volume and causing systolic hypertension [3, 15]. The global prevalence of TT is 0.2–1.3% [10, 23]. Cardiovascular complications in the long-term course of TT are among the earliest and most clinically significant, and they influence the subsequent prognosis and quality of life of these patients. Despite fluctuations in systemic blood pressure (BP), cerebral autoregulation maintains a constant cerebral blood flow [8]. AH affects cerebrovascular autoregulation and increases the risk of cerebrovascular events and dementia [1, 18]. In particular, it is associated with cerebrovascular remodelling and a reduction in cerebral blood flow at rest [17]. The results of a systematic review and meta-analysis demonstrated a strong association between elevated BP and reduced cerebral blood flow, with AH reducing cerebral blood flow by increasing cerebrovascular resistance [24]. As

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arterial stiffness and autonomic dysfunction increase, BP variability rises; this is an independent predictor of the risk of ischaemic stroke and intracerebral haemorrhage, and may potentially be modulated independently of mean BP control [22]. However, the interaction between AH and cerebral blood flow control remains poorly understood.Very few scientific studies have been devoted to abnormalities in cerebral haemodynamics in patients with TT, with or without concomitant AH. Therefore, investigating the characteristics of cerebral haemodynamic parameters in patients with TT and AH is a promising area of research that could form the basis for assessing the prognosis and optimising treatment for these patients.**The purpose of this article** was to investigate the characteristics of cerebral haemodynamics in patients with TT, both with and without concomitant AH, and to determine the role of these factors in the development of haemodynamic disturbances.

2. Materials and Methods

The study included 100 patients who were receiving inpatient treatment in the Endocrinology Department for comorbid cardiovascular disease and in the Department of Thyroid Disorders and Endocrine Dysfunctions at the Endocrinology Centre of the Kyiv City Clinical Hospital No. 18 and 27 essentially healthy individuals, who formed the control group.Inclusion criteria for the study: patients with verified (documented) TT, patients with verified (documented) AH, patients with verified (documented) AH associated with TT, and those who have signed an informed consent form to participate in the study.Exclusion criteria: patients with stage III AH, grade 3 AH, malignant AH, secondary AH of other aetiologies, other endocrine disorders, a history of myocardial infarction or stroke, or with heart failure of class IIB–III (NYHA functional class III–IV), severe cardiac arrhythmias and conduction disturbances, heart defects, hepatic or renal failure, bronchopulmonary, oncological and infectious diseases, pregnant women and women who are breastfeeding, as well as patients who did not sign the informed consent form or did not adhere to the study protocol.The diagnosis of AH is established on the basis of general clinical findings (patients' complaints, medical history, physical examination findings) and diagnostic tests (office blood pressure measurement, electrocardiography (ECG), Doppler echocardiography (EchoCG), 24-hour blood pressure monitoring (ABPM)) in accordance with the requirements of Order No. 1581 of the Ministry of Health of Ukraine dated 12 September 2024 'On the Approval of the Standardised Clinical Protocol for Primary and Specialised Medical Care: "Hypertensive Disease (Arterial Hypertension)".' Stage II, Grade 1 AH was diagnosed in 19 (29.2 %) patients, and Stage II, Grade 2 AH in 48 (73.8 %) patients. The diagnosis of TT was confirmed on the basis of laboratory and diagnostic imaging tests. The laboratory criteria were defined as a reduced level of thyroid-stimulating hormone (TSH) <0.1 mIU/L (reference range 0.3–4.2 mIU/L), elevated levels of serum free T3 (reference range 2.1–4.4 pg/ml) and free T4 (reference range 0.9–1.7 ng/dl), which were associated with increased levels of anti-TSH receptor antibodies (TSHR-Ab) >1.75 IU/l (reference range <1.5 IU/l) and elevated levels of antibodies to thyroid peroxidase (TPO-Ab) (reference range <34.0 IU/ml). Serum hormone levels were determined by electrochemiluminescence immunoassay (ECLIA) using a standard kit from Roche. Severe TT was observed in 29 (44.6%) patients, and moderate TT in 36 (55.4%).All patients underwent the thyroid ultrasonography using a Sonoline Sienna scanner with a 3.5–7.5 Hz transducer (Siemens, Germany) to establish a diagnosis of TT, with a volume of up to 10 cm^3 in each lobe considered normal. The presence of diffuse enlargement of the thyroid gland, nodules, and increased overall blood flow in the TG tissue was assessed.In accordance with the aim of the study, four groups were formed:Group I (main group, n=32) – patients with TT and concomitant AH, of whom 27 (84.4%) were women and 5 (15.6%) were men, with a median age of 29 (27; 47);Group II (comparison, n=33) – patients with TT without hypertension, of whom 29 (87.9%) were women and 4 (12.1%) were men, with a median age of 27 (25; 48);Group III (comparison, n=35) – patients with AH, of whom 27 (77.1%) were women and 8 (22.9%) were men, with a median age of 29 (28; 49);Group VI (control group, n=27) – essentially healthy individuals, comprising 21 (77.8%) women and 6 (22.2%) men, with a median age of 28 (27; 46).

The duration of AH in the groups ranged from 3 to 6 years, whilst the duration of TT ranged from 2 to 7.5 years. The groups were comparable in terms of key clinical and demographic characteristics.All patients underwent routine clinical assessment, duplex ultrasonography of the major arteries of the head and neck, 12-lead electrocardiography, office blood pressure measurement, EchoCG, ABPM, thyroid ultrasonography, and laboratory testing for TSH, FT3, FT4, TSHR-Ab and TPO-Ab.

The clinical assessment of patients included an analysis of their symptoms, medical and personal history, a physical examination carried out in accordance with standard procedures, and laboratory tests.ECG recordings were made using a YUKARD-200 electrocardiograph ('YUTAS', Ukraine) in 12 standard leads.ABPM was performed using the VAT 41–2 apparatus (IKS-TECHNO, Ukraine) according to the standard method. The assessment of cerebral haemodynamics was carried out using transcranial duplex scanning of the intracranial vessels and extracranial segments of the brachiocephalic vessels on an MX7 (Mindray, China), using an L13-3Ns linear probe with a frequency of 3–13 mHz. The examination was performed with the patient in a supine position (following a 10-minute rest period), with the chin tilted towards the side opposite to that being examined to ensure better access. Scanning of the carotid arteries was performed in three planes: two longitudinal (anterior and posterior) and one transverse. Scanning of the brachiocephalic arteries was carried out under the same conditions, in accordance with standard requirements for conducting biomedical experiments.

During the study, the mean linear blood flow velocities (LBFV) in the external carotid arteries (ECA), internal carotid arteries (ICA), vertebral arteries (VA) and middle cerebral arteries (MCA) were measured and analysed, the pulsatility index (PI) and the resistance index (RI), and the total cerebral blood flow volume (TC VBF) was calculated as the sum of the volumetric blood flow through both internal carotid arteries (VBF ICA) and the volumetric blood flow through both vertebral arteries (VBF VA).

Statistical analyses were performed using STATISTICA 6.0 and Microsoft Excel XP. Data normality was assessed using the Kolmogorov–Smirnov test. As most variables were not normally distributed, quantitative data were presented as the median and the first and third quartiles: Me (Q1, Q3).

The statistical significance of differences was assessed using the Mann–Whitney U test for independent samples and the Wilcoxon signed-rank test for paired samples. Between-group differences in categorical variables were assessed using Pearson's chi-square test with Yates' continuity correction, where appropriate. A two-sided p-value of <0.05 was considered statistically significant.

The study was conducted in accordance with the principles of the World Medical Association's Code of Ethics (the Declaration of Helsinki) regarding research involving human subjects.

3. Results and Discussion

Statistically significant differences were found among all study groups for most of the parameters examined. The most pronounced differences were observed in LBFV and the integral measures of volumetric blood flow.

The results of the analysis of the parameters demonstrated a significant increase in the mean LBFV in extracranial and intracranial vessels specifically in patients with TT combined with AH, compared with patients with TT without AH, patients with AH and the control group in the ECA (by 20.4%; 29.0% and 30.6% respectively; $p < 0.05$), in the ICA (by 10.3%; 40.3% and 18.6% respectively, $p < 0.05$), in the VA (by 8.9%; 28.9% and 22.6% respectively; $p < 0.05$) and in the MCA (by 14.5%; 34.2% and 21.0% respectively; $p < 0.05$) (Table 1).

In Group II, a statistically significant increase in LBFV values was also observed in the ECA (by 10.8% and 12.9%; $p < 0.05$), in the ICA (by 33.5% and 9.4%; $p < 0.05$), in the VA (by 22.6% and 15.8%; $p < 0.05$) and in the MCA (by 22.9% and 7.6%; $p < 0.05$), compared with Group III and the control group respectively (Table 1). Other authors also indicate that patients with TT typically exhibit an increase in regional mean LBFV and regional hyperperfusion compared with essentially healthy individuals [9].

In contrast, Group III showed lower LBFV values in the intracranial vessels compared with healthy individuals: in the ICA (by 36.4%; $p < 0.05$) and in the MCA (by 20.0%; $p < 0.05$) (Table 1). This is confirmed by a study of cerebral perfusion in patients with untreated, controlled and uncontrolled AH, which observed a reduction in mean LBFV values in patients with AH, particularly in the MCA, compared with essentially healthy individuals [4].

The increase in total cerebral blood flow in patients with AH and TT, compared with patients with TT without AH, patients with AH and the control group (by 38.8%; 37.3% and 29.8%, respectively; $p < 0.05$) was due to an increase in the carotid component (by 27.6%; 37.9% and 32.8%, respectively; $p < 0.05$) (Table 2).

No statistically significant differences were found between the total cerebral blood flow indices in patients with TT without AH and those with AH. However, total cerebral blood flow in patients with TT without AH and in patients with AH was reduced by 12.8% and 10.6%, respectively, compared with the control group ($p < 0.05$) (Table 2).

Consequently, a reduction in total cerebral blood flow was observed in the group of patients with TT, which is a sign of impaired cerebral circulation and may subsequently contribute to the

Table 1

Measures of mean linear velocities of extracranial and intracranial blood flow in the study groups

Parameter, cm/s	Group I (main) (n=32)	Group II (comparison) (n=33)	Group III (comparison) (n=35)	Group IV (control) (n=27)
LBFV ECA	121.1 (98.0–134.0)*,#,&	96.4 (82.0–112.0)#,&	86.0 (72.0–101.0)	84.0 (76.0–110.0)
LBFV ICA	96.2 (86.6–125.5)*,#,&	86.3 (64.2–102.7)#,&	57.4 (44.9–60.7)&	78.3 (72.3–84.6)
LBFV VA	46.1 (39.3–58.5)*,#,&	42.4 (37.3–53.7)#,&	32.8 (27.7–38.9)	35.7 (39.2–42.8)
LBFV MCA	158.3 (134.0–163.8)*,#,&	135.3 (123.7–139.0)#,&	104.2 (94.0–113.5)&	125.0 (120.0–132.0)

Notes: Data are presented as median (Q1–Q3). *p < 0.05 vs Group II; #p < 0.05 vs Group III; &p < 0.05 vs Group IV.

Table 2

Integral measures of volumetric blood flow in the study groups

Parameter, L/min	Group I (main) (n=32)	Group II (comparison) (n=33)	Group III (comparison) (n=35)	Group IV (control) (n=27)
VBF ICA	0.58 (0.55–0.63)*,#,&	0.42 (0.29–0.49)	0.36 (0.31–0.37)	0.39 (0.24–0.41)
VBF VA	0.11 (0.10–0.23)	0.09 (0.08–0.22)	0.10 (0.10–0.25)	0.10 (0.07–0.19)
VBF TC	0.67 (0.62–0.80)*,#,&	0.41 (0.27–0.55)&	0.42 (0.32–0.49)&	0.47 (0.39–0.50)

Notes: Data are presented as median (Q1–Q3). *p < 0.05 vs Group II; #p < 0.05 vs Group III; &p < 0.05 vs Group IV.

development of metabolic cerebral disorders and other complications in these patients. Scientific literature frequently describes an increase in regional perfusion; however, some researchers also point to lower global cerebral perfusion in patients with TT, particularly against a background of high T4 levels [6, 9]. In some patients with AH, cerebral perfusion at rest remains preserved; however, a number of researchers note a chronic tendency towards hypoperfusion due to vascular remodelling and an increase in cerebrovascular resistance [4, 17, 24].

In fact, in the group of patients with TT and AH, a significant increase in total cerebral blood flow was observed, suggesting that AH acts as an additional factor placing strain on the mechanisms compensating for cerebral blood flow, which may lead to their premature exhaustion.

We observed statistically significant differences in RI values in the ECA between patients in the main group and those with TT without AH, patients with AH, and the control group (by 20.3%; 12.2% and 24.3% respectively; $p < 0.05$), in the ICA (by 25.9%, 17.3% and 28.4% respectively; $p < 0.05$), in the MCA (by 19.4%; 11.1% and 23.6% respectively; $p < 0.05$, and in the VA (by 20.8%; 11.1% and 27.7% respectively; $p < 0.05$) (Table 3).

A statistically significant increase in RI values was also observed in patients with AH, compared with those with TT and the control group; this was most pronounced in the ICA (by 10.4% and 13.4% respectively; $p < 0.05$) and the MCA (by 10.9% and 18.8% respectively; $p < 0.05$). In patients with TT, there was a tendency towards increased RI in the vessels under investigation compared with healthy individuals, but this difference was not statistically significant ($p > 0.05$) (Table 3).

Analysis of the PI index in extra- and intracranial arteries showed a statistically significant increase in patients in the main group compared with patients with TT without AH, patients with AH, and the control group. The most significant differences were observed in the ICA (by 29.6%; 13.0% and 31.9% respectively; $p < 0.05$) and in the MCA (by 34.1%; 24.6% and 37.3% respectively; $p < 0.05$) (Table 3).

In patients with AH, the RI was significantly higher in all the vessels studied compared with patients with TT and the control group. The most significant increase in RI in these patients was observed in the carotid artery system: in the ECA (by 22.9% and 26.1% compared with groups II and IV, respectively; $p < 0.05$) and in the ICA (by 19.0% and 21.8% compared with groups II and IV, respectively; $p < 0.05$). The RI index in patients with TT showed a clear tendency to increase compared with the control group, but again this difference did not reach statistical significance (Table 3).

Table 3

RI and PI values in the study groups

Parameter, a.u.	Group I (main) (n=32)	Group II (comparison) (n=33)	Group III (comparison) (n=35)	Group IV (control) (n=27)
RI				
RI ECA	0.74 (0.62–0.83)*,#,&	0.59 (0.47–0.69)	0.65 (0.54–0.71)*,&	0.56 (0.41–0.68)
RI ICA	0.81 (0.69–0.83)*,#,&	0.60 (0.52–0.66)	0.67 (0.56–0.72)*,&	0.58 (0.49–0.61)
RI VA	0.77 (0.67–0.84)*,#,&	0.63 (0.59–0.68)	0.65 (0.60–0.74)	0.60 (0.55–0.67)
RI MCA	0.72 (0.62–0.81)*,#,&	0.57 (0.51–0.66)	0.64 (0.57–0.69)*,&	0.52 (0.51–0.63)
PI				
PI ECA	1.72 (1.79–1.86)*,&	1.24 (1.22–1.52)	1.61 (1.40–1.72)*,&	1.19 (1.07–1.34)
PI ICA	1.69 (1.64–1.83)*,#,&	1.19 (1.10–1.41)	1.47 (0.17–1.54)*,&	1.15 (1.09–1.29)
PI VA	1.53 (1.25–1.78)*,&	1.25 (1.17–1.46)	1.44 (1.21–1.62)*,&	1.21 (1.10–1.33)
PI MCA	1.26 (1.14–1.52)*,#,&	0.83 (0.70–0.94)	0.95 (0.73–1.22)*,&	0.79 (0.75–1.11)

Notes: Data are presented as median (Q1–Q3). *p < 0.05 vs Group II; #p < 0.05 vs Group III; &p < 0.05 vs Group IV.

Data from other authors indicate that an increase in RI and PI is not typical in patients with TT without concomitant pathology. However, against a background of tachycardia, hyperdynamic circulation and in the presence of vascular complications, increases in these parameters have been observed in patients with TT [6, 20]. Nevertheless, the results of a number of studies convincingly indicate a typical increase in RI and PI in patients with AH compared with essentially healthy individuals, reflecting the remodelling of small cerebral arteries and arterioles in hypertensive individuals [21, 24].

Thus, in patients with TT, compared with healthy individuals, the most characteristic findings are hyperdynamic and regional changes in perfusion: increased blood flow to specific areas of the brain, particularly those associated with cognitive activity. At the same time, a reduction in global cerebral perfusion is observed, which may reflect depletion of the vasodilatory reserve and lead to hypoxia and metabolic-haemodynamic imbalance [6, 9, 16, 20].

In patients with AH, compared with healthy individuals, a different pattern of changes predominates: an increase in cerebrovascular resistance, a rise in the pulsation index, a reduction in blood flow velocity and a chronic tendency towards hypoperfusion, indicating vascular dysadaptation, which may be associated with vascular remodelling and endothelial dysfunction, and leading to rapid disruption of autoregulatory mechanisms and premature depletion of cerebrovascular reserve [4, 12, 21, 24].

4. Conclusions

1. In patients with TT and concomitant AH, LBFV values and vascular resistance and pulsatility indices in the extracranial and intracranial segments of the major arteries of the head and neck were significantly higher, along with increased total cerebral blood flow, than in patients with TT without AH and in the control group.

2. These findings suggest that concomitant AH may contribute to marked dysregulation of vascular tone, increased haemodynamic load, and premature depletion of compensatory haemodynamic mechanisms in patients with TT. Consequently, AH may aggravate the course of the underlying disease and promote the earlier development of dysmetabolic cerebral disorders and cerebrovascular complications.

The perspective for further research lies in a more in-depth study and analysis of the pathophysiological changes in the intra- – and extracranial vessels and the state of cerebral haemodynamics, as well as their probable interrelationships with clinical and syndromic characteristics and the severity of cognitive impairment in patients with AH and TT; this will enable the refinement of diagnostic algorithms and the optimisation of treatment approaches for these patients.

Conflict of interest

The authors declare that they have no conflicts of interest in relation to this study, including those of a financial, personal, authorship or other nature that might influence the study and its results as presented in this article. The work is a fragment of the research project "Clinical and pathogenetic aspects of diagnosis and treatment of patients with combined pathology of internal organs (diseases of the cardiovascular system, digestive organs, endocrine system)", state registration No. 0119U101507.

Financing

The study was conducted without financial support.

Data availability

The de-identified data that support the findings of this study are available from the corresponding author upon reasonable request.

Use of artificial intelligence

The authors confirm that they did not use artificial intelligence technology in the creation of this work.

Authors' contribution

Tetiana Bentsa: Conceptualization, Formal analysis, Writing – Original Draft, Writing – Review & Editing; Oksana Pastukhova: Methodology, Investigation, Formal Analysis, Writing – Original Draft, Writing – Review & Editing; Iryna Tsymbaliuk: Methodology, Investigation, Writing – Review & Editing.

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ВІДОМОСТІ ПРО АВТОРІВ

Tetiana Bentsa
Doctor of Medical Sciences, Professor,
Professor at the Department of Therapy
and Rheumatology
Shupyk National Healthcare University of Ukraine
bentsa_t@i.ua
ORCID: 0000-0001-9427-2549

Oksana Pastukhova
Candidate of Medical Sciences, Associate Professor,
Associate Professor at the Department
of Family Medicine and Outpatient Care
Shupyk National Healthcare University of Ukraine
xsenon26@gmail.com
ORCID: 0000-0003-0734-8598

Iryna Tsymbaliuk
Candidate of Medical Sciences,
Head of the Diagnostics Department
Endocrinology Centre,
Kyiv City Clinical Hospital No. 18
itsymbaliuk4@gmail.com

Бенца Тетяна Михайлівна
доктор медичних наук, професор,
професор кафедри терапії та ревматології
Національний університет охорони здоров'я
України імені П. Л. Шупика
bentsa_t@i.ua
ORCID: 0000-0001-9427-2549

Пастухова Оксана Анатоліївна
кандидат медичних наук, доцент, доцент кафедри
сімейної медицини та амбулаторно-поліклінічної
допомоги
Національний університет охорони здоров'я
України імені П. Л. Шупика
xsenon26@gmail.com
ORCID: 0000-0003-0734-8598

Цимбалюк Ірина Леонідівна
кандидат медичних наук,
завідувачка діагностичним відділенням
Ендокринологічний центр КНП
Київська міська клінічна лікарня № 18
itsymbaliuk4@gmail.com

Дата надходження статті: 03.04.2026
Дата надходження виправленої версії статті: 12.05.2026
Дата прийняття статті: 29.05.2026
Дата публікації статті: 02.11.2026