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HYPERTENSIVE ENCEPHALOPATHY: TO BE DIAGNOSED AND TREATED IMMEDIATELY

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Keywords: hypertensive encephalopathy, hypertensive crisis, posterior reversible encephalopathy syndrome, autoregulation of cerebral blood flow, treatment of hypertensive encephalopathy.

Summary

It is important to have a good understanding of the pathogenetic diagnosis and treatment features of the severe complication with high mortality - hypertensive encephalopathy (HE) because of the amount of people suffering from chronic arterial hypertension is constantly increasing. Thus, this article briefly reviews the mechanism of cerebral blood flow autoregulation, HE pathophysiology, and the main aspects of diagnosis and treatment. An HE episode is caused by a sudden and significant increase in arterial blood pressure, when the autoregulatory mechanism of cerebral blood flow becomes ineffective. Along with increased blood pressure, the patient gradually develops neurological symptoms ranging from intense headache to vision loss, loss of consciousness, and convulsions. The diagnosis of HE is based on the clinical signs - acute arterial hypertension and the subsequent neurological symptoms. Also, the diagnosis of HE can be determined retrospectively when the neurological symptoms rapidly disappear with effective antihypertensive treatment. Computed tomography is important in differentiating HE from intracerebral hemorrhage and ischemic stroke. In management of HE it is recommended to return to the limits of cerebral blood flow autoregulation by immediately reducing mean arterial blood pressure by 20-25%. In order to achieve the maximum rapid antihypertensive effect, it is suggested to administer the medication only intravenously. Labetalol and sodium nitroprusside are currently recommended first-line drugs for the treatment of HE. Patients with HE should be hospitalized in the intensive care unit for continuous control of decreasement of arterial blood pressure while observing the cautious dynamics of the neurological condition. The outcome of patients depends on the urgency and efficiency of treatment in returning arterial blood pressure to the limits of cerebral blood flow autoregulation, as well as on comorbidities, the severity of the condition and possible complications.

Introduction

The problem of arterial hypertension is global and constantly relevant, the total prevalence of this condition in adults is about 30-45% not depending on the development of the country (1, 2). It is predicted that by 2025 the number of people with hypertension could increase by 15-20% to nearly 1.5 billion (3). With such a rapid increase in the number of cases of arterial hypertension it is crucial to pay attention to the most serious related condition – hypertensive encephalopathy (HE). This hypertensive crisis is characterized by an acute manifestation of marked hypertension complicated by encephalopathy (neurological) symptoms and requires emergency care (1, 4).

Along with HE, other hypertensive conditions require urgent control of blood pressure, including acute ischemic stroke with systolic blood pressure >180mmHg or diastolic >120mmHg, acute hemorrhagic stroke with systolic blood pressure >180mmHg, severe preeclampsia with systolic blood pressure >160mmHg, eclampsia and other hypertensive conditions with target organ damage (1, 4). However, it is necessary to emphasize that according to the latest guidelines for the treatment of hypertensive crises, it is recommended to lower arterial blood pressure immediately and markedly only during HE, in order to return and maintain it within the physiological limits of autoregulation of cerebral blood flow (4). Treatment without following the recommended strategy could be associated with the risk of brain hypoperfusion and an unfavorable outcome (5-8). The latest European and United States guidelines for the treatment of hypertension indicate and justify the importance as well as the specific management of differential diagnosis, rapid and intensive treatment and monitoring in HE (1, 4, 7).

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The aim - with this article, we aim to emphasize the relevance of understanding the pathophysiology of HE for diagnosis and the associated selection of the optimal antihypertensive medication also, its method of administration, as it is related with the prognosis of HE.

Research material and methods

Review of scientific articles on the pathophysiology, diagnostics and treatment of hypertensive encephalopathy. The search was performed in PubMed, Elsevier Science and Wiley Online Library databases. Analyzed 1959-2021 published retrospective and prospective studies and scientific articles.

Results

Epidemiology. On average, 1-2% of the population with arterial hypertension will eventually experience at least one hypertensive emergency (9). Patients experiencing a hypertensive emergency (systolic blood pressure >220 mmHg or diastolic blood pressure >120 mmHg with target organ damage) make up about 0.5% of all admissions to emergency department, and HE is diagnosed in as many as 5-16% of cases (10,11).

Pathophysiology. Cerebral blood flow is one of the main factors human consciousness depends on (12). Human brain has an extremely intense need for oxygen, glucose and other metabolites, hence it is very important that the supply of these substances is continuous and uninterrupted, which directly depends on stable cerebral blood flow (13). The cerebrovascular system of a healthy person through regulatory mechanisms maintains the cerebral blood flow between 50 and 54 ml of blood per 100 g of brain mass per minute; in case of deviation from this norm, the symptoms of encephalopathy may develop (12).

Cerebral blood flow depends on two factors: cerebral vascular resistance and cerebral perfusion pressure (CPP), which equals to the difference between the mean arterial pressure (MAP) and the intracranial pressure (ICP). Most often, MAP and cerebral perfusion change in the same direction (11,12). As the MAP increases, so does the CPP, and in order for the cerebral blood flow to remain normal, immediately increases the cerebral vascular resistance, that is, the vasoconstriction increases the resistance of the cerebral arteries and thereby reduces the cerebral blood flow to compensate the increased MAP. Correspondingly, as the MAP decreases, the CPP also decreases, and the resistance of the cerebral vessels also begins to decrease - thus vasodilation compensates the decreased MAP (12,14). 65-125 mmHg is considered a normal MAP, but with well-functioning autoregulation of cerebral blood flow, the brain could maintain normal cerebral blood flow when the MAP is between 50-60 and 150-160 mmHg (Fig. 1) (12,15,16). Moreover, the cerebrovascular lumen physiologically responds not only to changes in MAP, but also to blood pH, pCO_2, pO_2 (12).

This autoregulatory mechanism of cerebral blood flow works very efficiently, and the cerebral blood flow remains stable regardless of changes in MAP within the above mentioned limits. However, a very sudden or marked increase in MAP can provoke decompensation of cerebral blood flow autoregulation (11). These disorders of the autoregulatory mechanisms of cerebral blood flow can be determined by aging, pregnancy, chronic hypertension (11,13). When the MAP exceeds the normal limits, the contraction of cerebral vessels is no longer sufficient to compensate the increased CPP and MAP, so the cerebral blood flow also changes causing damage to the blood-brain barrier and increasing its permeability, thus the liquid from the cerebral vessels can leak through the capillary membranes into the brain parenchyma (12). Then brain edema develops, intracranial pressure increases and, therefore, various neurological symptoms may appear (17,18).

The regulatory mechanisms of cerebral blood flow are altered in patients with chronic arterial hypertension. The graph reflecting autoregulation of cerebral blood flow proposed by Lassen NA (15) shifts to the right (Figure 1), showing that the tolerance of cerebral blood vessels to higher MAP increases, as confirmed by other researchers (11,19). However, in case of chronic arterial hypertension, a sudden increase of MAP above the limits of autoregulation of cerebral blood flow can manifest not only as incipient brain edema, but also as cerebral ischemia due to hyperregulation of cerebral blood flow and overexpressed vasoconstriction of arterioles (20). Fibrinoid necrosis of cerebral arterioles, thrombosis of arterioles and capillaries, and parenchymal damage (microinfarcts) were found during neuropathological examinations in patients with chronic hypertension who developed HE (21).

In the presence of altered autoregulation of cerebral blood flow due to chronic hypertension, using more intensive treatment than indicated by current recommendations may result in faster development of cerebral hypoperfusion (Figure 1) (11).

In recent publications the development of HE due to increased intensity of cerebral blood flow is increasingly based on the PRES – posterior reversible encephalopathy syndrome (4,17). The PRES is an acute neurological disorder characterized by various neurological symptoms - headache, reduced visual acuity or visual field deficit, loss of consciousness, confusion, seizures and focal neurological disorders (4,18,22). The name of this syndrome in terms of "reversibility" is not quite accurate, because irreversible changes may

begin to develop in the brain if the treatment is not started immediately or if the treatment is inadequate. What is more, despite the term "posterior", brain damage may not always be located in the posterior parts of the brain, and as the condition progresses, other parts of the brain may also be affected (11,17,18). However, the findings of clinical and imaging studies confirm that the posterior regions of the cerebral hemispheres are particularly sensitive to the increase in cerebral blood flow (22,23). The worse response of the posterior parts of the brain to increased cerebral perfusion is also based on the main "vasogenic" theory of the manifestation of HE. The theory states the posterior parts of the brain are less innervated by sympathetic fibers compared to the anterior part of the brain, thus vasoconstriction is slower in the posterior parts and the risk of hyperperfusion and decompensation of cerebral blood flow autoregulation may occur faster (18-20).

Diagnostics. The diagnosis of HE is based on the clinical condition, when suddenly developed or suddenly aggravated chronic hypertension (usually as arterial blood pressure exceeds 200/120 mmHg) occurs together with neurological symptoms: lethargy, seizures, visual disturbances and coma (1,4). These are the most prominent symptoms, but in the earlier stages there may be signs of general, less often even focal brain damage – that may be extremely intense headache, nausea, agitation, temporary motor function or visual disturbances (4,11).

The importance of imaging studies in confirming the diagnosis of HE is rather low. The most common imaging test performed after the onset of acute neurological symptoms is computed tomography (CT), which is significant only for differentiation from other hypertensive conditions or complications of HE (4). Specific radiological changes

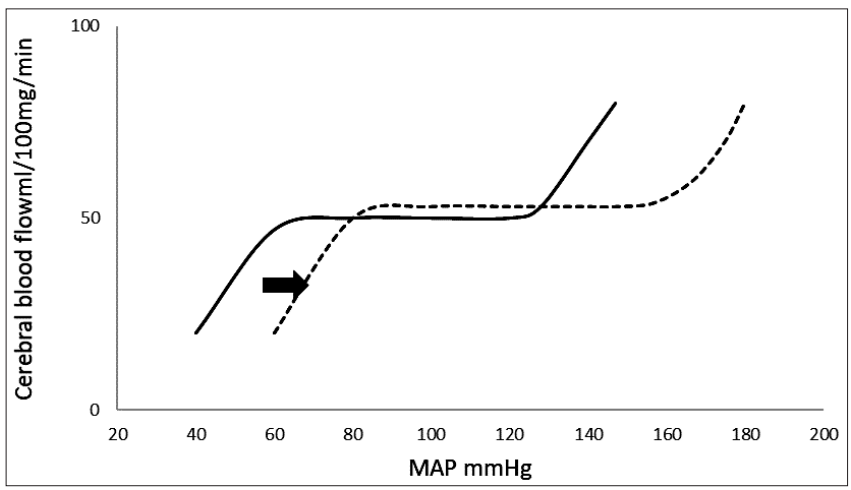

Figure 1. Normal limits of cerebral blood flow autoregulation and changes in cerebral autoregulation caused by chronic hypertension

Table 1. Characteristics of recommended antihypertensive drugs

Drug	Start of response	Duration of effect	Dose	Contraindications	Adverse reaction
Labetalol	5-10 minutes	3-6 hours	0.25–0.5 mg/kg intravenous bolus; 2–4 mg/min continuous infusion therapy until target blood pressure is reached, then 5–20 mg/h	Former second or third degree atrioventricular block, systolic heart failure, asthma and bradycardia	Brochoconstriction and bradycardia
Sodium nitroprusside	Immediately	1-2 minutes	0.3–10 µg/kg/min, increase by 0.5 µg/kg/min every 5 min until target blood pressure is reached	Liver and kidney failure (conditional contraindications)	Theoretically, toxic effects of cyanides or thiocyanates could occur in large doses over a long period of time

of HE can be seen only after significant cerebral edema has occurred (11). In HE, magnetic resonance imaging (MRI) (T2 and FLAIR modes) may present specific changes in white matter as vasogenic cerebral edema involving the posterior regions of the brain, especially the parieto-occipital lobes (18,23). Observation of these radiological changes on MRI together with the clinical picture of the neurological lesion leads to the confirmation of HE (24). In practice, performing MRI in such cases is extremely inconvenient: this examination takes a long time; due to encephalopathy it is difficult to maintain a stable position of the patient's body during the examination, what could be related with many artefacts; in the early stage of the disease the expected changes are not necessarily detectable. Thus, in general, in the critical condition of patients, MRI is not relevant as an option examination for the diagnosis of HE (4,11).

The diagnosis of HE can also be determined by the effectiveness of antihypertensive treatment. The rapid effect of immediate treatment with antihypertensive drugs in reducing the symptoms of HE is a clear criterion confirming the diagnosis of HE (11,20).

Differential diagnosis. The differential diagnosis of HE is important, since imaging tests are of little significance for confirming the diagnosis, and its practical justification is mostly based on the clinical picture (4). In order to choose the right immediate treatment, differential diagnosis is recommended to be performed as soon as possible (3). To that end, it is important to determine the sequence of symptoms: HE is characterized by an acute increase in blood pressure and subsequent neurological symptoms (11,22,25). If, on the contrary, hypertension accompanies the primary neurological damage, HE is less likely (11). This is an excellent rational way to distinguish HE from secondary hypertension, when, due to its compensatory nature, the strategy of blood pressure correction is completely different.

In the case of sudden onset of neurological symptoms, CT is the most common imaging test performed as a matter of urgency, and it is also useful in differentiating HE from intracerebral hemorrhage and ischemic stroke (4,11).

Treatment. Patients experiencing a hypertensive crisis are recommended to be hospitalized in the intensive care unit with continuous hemodynamic and neurological monitoring (1,8,26). Patients suffering from HE may experience complications of severe hypertension or its treatment – epileptic condition, cerebral ischemia, intracerebral hemorrhage (22).

HE is a hypertensive crisis, and it is strongly recommended to be treated immediately to reach the target blood pressure (returning to the limits of cerebral blood flow autoregulation). Therefore, the drugs administered should have a rapid response (within minutes) (1,4). Thus, in the case of HE, antihypertensive medications should be administered only intravenously, as this type of administration provides the fastest response (1,4,8,26,27).

Antihypertensive treatment should be titrated according to MAP in order to reduce it by 20-25% (1,4,7). Unduly intensive antihypertensive treatment exceeding the limits of autoregulation of cerebral blood flow (reducing MAP by >25%) can excessively reduce the cerebral blood flow and cause cerebral hypoperfusion, especially in patients with chronic arterial hypertension. If the arterial blood pressure is reduced by 50% or more, this alone could lead to the death of the patient due to cerebral ischemia, which is why the treatment, although urgent, should be cautious enough: antihypertensive drugs should be titrated according to the percentage change in MAP and clinical status should be closely monitored (28,29).

Labetalol and sodium nitroprusside are safe and effective for significant blood pressure correction even in patients with impaired cerebral autoregulation (4,30). However, compared to labetalol, sodium nitroprusside is easier to titrate due to its significantly shorter duration of action, thus allowing patient safety to be ensured by more flexible balancing of the drug's effect on blood pressure increase, when adverse changes could last only a very short time without causing iatrogenic complications (Table 1) (4,19,31). This ensures an extremely fast and highly beneficial sensitively flexible effect on the blood flow in urgent controlling of life threatening condition – hypertensive crisis.

Conclusions

1. Hypertensive encephalopathy to be diagnosed based on the clinical signs, with a sudden and significant increase in arterial blood pressure and the subsequent appearance of a wide range of neurological symptoms. The diagnosis is also established if the clinical signs of encephalopathy disappear with proper correction of hypertension.

2. Cerebral computed tomography scan is useful for differentiation of hypertensive encephalopathy with intracerebral hemorrhage and ischemic stroke.

3. Hypertensive encephalopathy treatment should be started immediately by lowering mean arterial blood pressure by 20-25%.

4. Intravenous medications with the fastest possible response should be prescribed to treat hypertensive encephalopathy, labetalol and sodium nitroprusside are currently first line medications. Titration is determined by the change in mean arterial blood pressure.

5. Although hypertensive encephalopathy is often a reversible condition, serious, irreversible changes may develop when patients are treated without following the brain

autoregulation norms and recommended treatment tactics.

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HIPERTENZINĖ ENCEFALOPATIJA: DIAGNOZUOJAMA IR GYDOMA NEDELSIANT

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Raktažodžiai: hipertenzinė encefalopatija, hipertenzinė krizė, grįžtamasis užpakalinis encefalopatijos sindromas, smegenų kraujotakos autoreguliacija, hipertenzinės encefalopatijos gydymas.

Santrauka

Pasaulyje nuolat daugėjant sergančiųjų lėtine arterine hipertenzija, svarbu gerai suvokti sunkiausios komplikacijos – hipertenzinės encefalopatijos (HE) patogenetinės diagnostikos bei gydymo ypatumus, siejamus su jo baigtimi. Straipsnyje glaustai apžvelgiami smegenų kraujotakos savireguliacijos mechanizmai, HE patofiziologija, pagrindiniai diagnostikos ir gydymo aspektai. HE epizodą sukelia staigus ir ženklus arterinio kraujospūdžio padidėjimas, kai smegenų kraujotakos intensyvumo autoreguliacinis mechanizmas tampa neveiksmingas. Pacientui kartu su padidėjusiu kraujospūdžiu laipsniškai ryškėja neurologinė simptomatika: nuo intensyvaus galvos skausmo iki regos, sąmonės sutrikimo, traukulių.

HE diagnozė grindžiama klinikiniu vaizdu – ūmine arterine hipertenzija ir jos sukelta neurologine simptomatika. HE diagnozė gali būti nustatoma retrospektyviai, kai taikant efektyvų antihipertenzinį gydymą, neurologiniai simptomai sparčiai nyksta. Kompiuterinės tomografijos tyrimas svarbus diferencijuojant HE nuo intracerebrinio kraujavimo ir išeminio insulto. Grįžti į smegenų kraujotakos autoreguliacijos ribas rekomenduojama nedelsiant sumažinant vidurinį arterinį kraujo spaudimą 20-25 procentais. Siekiant greičiausio antihipertenzinio poveikio gydant HE, medikamentus siūloma skirti tik į veną. Pirmo pasirinkimo vaistai HE gydymui šiuo metu siūlomi labetalolis ir natrio nitroprusidas. HE sergančius pacientus reikėtų stacionarizuoti į reanimacijos ir intensyviosios terapijos skyrių, nuolatos monitoruoti arterinį kraujospūdį, stebint neurologinės būklės dinamiką. Gydymo baigtis priklauso nuo jo skubumo ir efektyvumo grąžinant arterinį kraujospūdį į smegenų kraujotakos savireguliacijos ribas, gretutinių ligų, būklės sunkumo ir galimų komplikacijų.

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