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Research Article

CLINICAL OVERVIEW OF HYPERTENSIVE ENCEPHALOPATHY

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A B S T R A C T

Hypertensive encephalopathy is a neurological dysfunction induced by malignant hypertension. The term "hypertensive encephalopathy" was introduced to describe this type of encephalopathy by Oppenheimer and Fishberg in 1928. Hypertensive encephalopathy refers to the transient migratory neurologic symptoms that are associated with the malignant state in a hypertensive emergency and the clinical symptoms are usually reversible with prompt initiation of therapy. Hypertensive encephalopathy occurs in eclampsia, acute nephritis and crises in essential hypertension. Symptoms of hypertensive encephalopathy include headache, restlessness, nausea, disturbances of consciousness, seizures, bleeding in the retina, and papilledema. Focal brain lesions may be associated with specific neurological symptoms. These neurological impairments may culminate in a comaif not treated properly and adequately.

INTRODUCTION

Of the all people with hypertension, less than 1% develop a hypertensive emergency. The morbidity and mortality associated with hypertensive encephalopathy are related to the degree of target-organ damage. Without treatment, the 6-month mortality for hypertensive emergencies is 50%, and the 1-year mortality approaches 90%.

Hypertensive encephalopathy mostly occurs in middle-aged individuals who have a long-standing history of hypertension. Hypertension in general is more prevalent in men than in women. The frequency of hypertensive encephalopathy in various ethnic groups corresponds to the frequency of hypertension in the general population. Hypertension is more prevalent in black people, exceeding the frequency in other ethnic minority groups. The incidence of hypertensive encephalopathy is lowest in white people.

Signs and symptoms

Hypertensive encephalopathy is most commonly encountered in young and middle-aged people who suffer from hypertension. Overall, the condition is rare even among hypertensive patients. Different clinicians reported that from 0.5 to 15% of patients with malignant hypertension developed hypertensive encephalopathy. With the development of methods for detection and treatment of hypertension, hypertensive encephalopathy has been becoming more rare.

Symptoms of hypertensive encephalopathy typically start to occur 12–48 hours after a sudden and sustained increase in blood pressure. The first manifestation of these symptoms is a severe headache. Headache occurs in more than 75% of patients. The patient becomes restless. Alterations in consciousness may follow several hours later, which include impaired judgement and memory, confusion, somnolence and stupor. If the condition is not treated, these neurological symptoms may worsen and ultimately turn into a coma. Other symptoms may include increased irritability, vomiting, diplopia, seizures, twitching and myoclonus of the limbs. Alterations in vision (vision blurring, hemivisual field defects, color blindness, cortical blindness) are common. Hemiparesis, intracerebral hemorrhage, aphasia may also occur, but they are less common. So, in the evaluation of an encephalopathic patient, it is vital to exclude systematic disorders and various cerebrovascular events that may present with a similar constellation or clinical findings.

Pathophysiology

The clinical manifestations of hypertensive encephalopathy are due to increased cerebral perfusion from the loss of blood-

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brain barrier integrity, which results in exudation of fluid into the brain. In normotensive individuals, an increase in systemic blood pressure over a certain range (ie, 60-125 mm Hg) induces cerebral arteriolar vasoconstriction, thereby preserving a constant cerebral blood flow (CBF) and an intact blood-brain barrier.

In chronically hypertensive individuals, the cerebral autoregulatory range is gradually shifted to higher pressures as an adaptation to the chronic elevation of systemic blood pressure. This adaptive response is overwhelmed during a hypertensive emergency, in which the acute rise in systemic blood pressure exceeds the individual's cerebral autoregulatory range, resulting in hydrostatic leakage across the capillaries within the central nervous system (CNS). Brain MRI scans have shown a pattern of typically posterior (occipital greater than frontal) brain edema that is reversible. This usually is termed reversible posterior leukoencephalopathy or posterior reversible encephalopathy syndrome (PRES).

With persistent elevation of the systemic blood pressure, arteriolar damage and necrosis occur. The progression of vascular pathology leads to generalized vasodilatation, cerebral edema, and papilledema, which are clinically manifested as neurologic deficits and altered mentation in hypertensive encephalopathy.

Etiology

The most common cause of hypertensive encephalopathy is abrupt blood pressure elevation in a chronically hypertensive patient. Other conditions that can predispose a patient to elevated blood pressure and cause the same clinical situation include the following:

- Chronic renal parenchymal disease
- Acute glomerulonephritis
- Renovascular hypertension
- Sudden withdrawal of hypertensive agents (eg, clonidine)
- Encephalitis, meningitis
- Pheochromocytoma, renin-secreting tumors
- Sympathomimetic agents (eg, cocaine, amphetamines, phencyclidine [PCP], and lysergic acid diethylamide [LSD])
- Eclampsia and preeclampsia
- Head trauma, cerebral infarction
- Collagen-vascular disease
- Autonomic hyperactivity
- Vasculitis
- Ingestion of tyramine-containing foods or tricyclic antidepressants in combination with monoamine oxidase inhibitors (MAOIs)

Treatment Approach Considerations

In patients without hypertension, cerebral autoregulation preserves a relatively constant cerebral blood flow (CBF) at a mean arterial pressure (MAP) range of 60-90 mm Hg. In chronically hypertensive patients, autoregulation is altered and shifted upward to maintain a relatively constant CBF at a higher MAP range.

When therapy is initiated, it is important to consider the baseline blood pressure in order to avoid excessive blood pressure reduction and prevent cerebral ischemia. It is usually safe to reduce MAP by 25% and to lower the diastolic blood pressure to 100-110 mm Hg. This level of BP control will allow gradual healing of the necrotizing vascular lesions. More aggressive hypotensive therapy is both unnecessary and may reduce the blood pressure below the autoregulatory range, possibly leading to ischemic events (such as stroke or coronary artery disease).

Acute monitoring in an intensive care unit (ICU) with arterial blood pressure monitoring is required for adequate titration of pharmacologic agents and monitoring of end-organ function. Potential complications of medical therapy (e.g, overzealous reduction in blood pressure and adverse effects or toxicity of pharmacologic therapy) must be watched for.

Deterioration of clinical status despite therapy warrants immediate and further investigation into other possible etiologies or reevaluation of therapy for worsening hypertensive encephalopathy.

Pharmacological Therapy

Pharmacologic agents selected for use in hypertensive encephalopathy should have few or no adverse effects on the central nervous system (CNS). Avoid agents such as clonidine, reserpine, and methyldopa. Although the clinical impact of diazoxide has not been determined, this agent is avoided because of the impact of decreased CBF. An increasing number of authorities are considering labetalol, nicardipine, and esmolol as preferred initial agents.

Labetalol provides a steady consistent drop in blood pressure without compromising CBF. It is frequently used as initial therapy as intravenous bolus or infusion. Because of its nonselective beta-blocking properties, labetalol should be avoided in severe reactive airway disease and cardiogenic shock.

Nicardipine is a second-generation dihydropyridine-derivative calcium channel blocker, which has high vascular selectivity and strong cerebral and coronary vasodilatory activity. It has been shown to increase stroke volume and coronary blood flow.

Clevidipine, a short acting dihydropyridine calcium blocker. It reduces blood pressure without affecting cardiac filling pressures or causing reflex tachycardia.

Nitroglycerin has been used to provide a rapid reduction in elevated blood pressure complicating myocardial ischemia. The reduction in blood pressure may be severe and can cause further complications due to venodilatory effects in volume-contracted individuals.

Nitroprusside sodium and hydralazine pose a theoretical risk of intracranial shunting of blood. Accordingly, these agents should be avoided in patients suspected of having increased intracranial pressure (ICP), because the potential intracerebral shunting of blood can increase the ICP. Hydralazine has a limited role in this setting, owing to reflex tachycardia, and it should not be used in patients with suspected coronary artery disease (CAD). Diuretics should also not be used in these patients unless there is clear evidence of volume overload. This is due to pressure natriuresis that occurs and leaves these patients volume depleted. Volume repletion by itself can sometimes lower the blood pressure.

Oral agents: A slower onset of action and an inability to control the degree of BP reduction has limited the use of oral antihypertensive agents in the therapy of hypertensive crises. They may, however, be useful when there is no rapid access to the parenteral medications described above. Both sublingual nifedipine and sublingual captopril can substantially lower the BP within 10 to 30 minutes in many patients. A more rapid response is seen when liquid nifedipine is swallowed.

The major risk with oral agents is ischemic symptoms (e.g., angina pectoris, myocardial infarction, or stroke) due to an excessive and uncontrolled hypotensive response. Thus, their use should generally be **avoided** in the treatment of hypertensive crises if more controllable drugs are available.

Now despite of all efforts if neurologic deterioration worsens with therapy, it is necessary to reconsider the extent of blood pressure reduction or to consider alternate diagnoses.

Once the BP is controlled, the patient should be switched to oral therapy, with the diastolic pressure being gradually reduced to 85 to 90 mmHg over two to three months. The initial reduction to a diastolic pressure of approximately 100 mmHg is often associated with a modest worsening of renal function; this change, however, is typically transient as the vascular disease tends to resolve and renal perfusion improves over one to three months. Antihypertensive therapy should not be withheld in this setting unless there has been an excessive reduction in BP. A change in medication, however, is indicated if the decline in renal function is temporally related to therapy with an angiotensin (ACE) converting enzyme inhibitor or angiotensin II receptor blocker, which can interfere with renal autoregulation and produce acute renal failure in patients with bilateral renal artery stenosis.

Prevention

Recommend lifestyle modifications, including weight reduction to decrease the patient's body mass index (BMI) to less than 27, moderation of alcohol and sodium intake, increasing physical activity, and avoidance of tobacco products.

Discharge patients on antihypertensives that were effective in maintaining an adequate blood pressure range during hospitalization. Emphasize the importance of adhering to antihypertensive therapy and scheduling reassessment at regular intervals to modify failing regimens.

Long-Term Monitoring

Because hypertension is a chronic problem, regularly reassessment is vital. Adequate control of hypertension is essential in preventing the progression of target-organ disease. High blood pressure has been associated with a rapid rate of cognitive decline and an increased risk of cardiac and neurologic events.

To guide the formulation of a more effective treatment plan, document prior hypertensive medication regimens that have failed.

Prognosis

Patients with hypertensive encephalopathy who are promptly treated usually recover without deficit. However, if treatment is not administered, the condition can lead to permanent neurological deficit or even death.

References

1. Moyer JH, Miller SI, Tashner AB, Snyder H, Bowman RO(1953) Malignant hypertension and hypertensive encephalopathy. Am J Med 14: 175-183
2. Chester EM, Agamanolis DP, Banker BQ, Victor M (1978) Hypertensive encephalopathy: a clinicopathologic study of 20 cases. Neurology 28: 928939.
3. Clarke E, Murphy EA (1956) Neurological manifestations of malignant hypertension. Br Med J 2: 1319-1326.
4. Strandgaard S, Paulson OB (1989) Cerebral blood flow and its pathophysiology in hypertension. Am J Hypertens 2: 486-492.
5. Tamaki K, Sadoshima S, Baumbach GL, Iadecola C, Reis DJ, Heistad DD (1984) Evidence that disruption of the blood–brain barrier precedes reduction in cerebral blood flow in hypertensive encephalopathy. Hypertension 6: I75-81.
6. Kaplan, NM. Hypertensive crises. In: Kaplan's Clinical Hypertension, 9th ed, Neal, W (Ed), Lippincott, Williams and Wilkins, Baltimore 2006. p.311.
7. Vaughan CJ, Delanty N. Hypertensive emergencies. Lancet 2000; 356:411.
8. Ledingham JG, Rajagopalan B. Cerebral complications in the treatment of accelerated hypertension. Q J Med 1979; 48:25.
9. Finnerty FA Jr (1972) Hypertensive encephalopathy. Am J Med 52:672-678.
10. Ram CVS (1978) Hypertensive encephalopathy: recognition and management. Arch Int Med 138: 1851-1853.

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