Case Report

Malignant Hypertension - A Rare Presentation of Brain Neoplasm

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ABSTRACT

Malignant hypertension is a hypertensive emergency requiring rapid lowering of blood pressure within hours. Brain neoplasms are a rare cause of hypertensive emergency. The pathophysiological basis is neurogenic edema compressing ventral medulla and pons. Intravenous corticosteroids along with antihypertensive drugs are required not only to relieve symptoms but also to reverse systemic hypertension.

Keywords – malignant hypertension, brain neoplasm, neurogenic edema, corticosteroids

INTRODUCTION

About 1 billion adults both in economically developed (333 million) and economically developing (639 million) have hypertension highest being in Eastern Europe and Latin America.¹

Hypertensive emergency including malignant hypertension accounts for 25% of all medical emergency visits. Majority of these patients have a previous history of hypertension.²

Recently diagnosed hypertension can be a rare presentation of brain neoplasm³ and can often be missed. Till date very few cases of brain neoplasm associated with secondary hypertension have been reported.⁴⁵ We believe that this is one such rare case.

CASE REPORT

A forty year old female presented to the medical emergency with complaints of severe, diffuse headache, associated with blurring of vision and giddiness which does not vary with position.

No history of previous ischemic heart disease or hypertension, recent oral contraceptive pills use or cocaine abuse. No family history suggestive of secondary causes of hypertension including adrenal disorders.

Personal and menstrual history was insignificant

Vitals – BP was 190/120, pulse was 100/minute, hyperdynamic with no radiofemoral delay. All other vitals were normal.
Systemic Examination

CNS examination showed weakness of left lateral rectus and head impulse test was negative. Rest of the systemic examination found to be normal.

Fundoscopy

The fundus picture of the patient’s left eye shows acute, established papilledema. The disc margins are blurred and irregular with obliteration of the cup and tortuous vessels. The cup is full and hyperaemic with minimal exudation. [Fig no.1]

Otoscopic Findings

The otoscopic examination of the left ear was normal except dull tympanic membrane.

Investigation

Total count was raised (23900cells/mm³), ESR 95 mm at the end of 1st hour, blood urea 19.8 mg/dl, serum creatinine 2.4, estimated creatinine clearance 24.59 ml/minute, urine analysis showed mild proteinuria.

CT SCAN findings – extra axial, uniformly contrast enhancing lesion measuring 3.5*3*3cm noted in the right cerebropontine angle tumour with broad base towards the petrous part of right temporal bone, with bony erosion causing compression over 4th ventricle and pons.fig no 2.

ECG was normal except for tachycardia. Ultrasound abdomen was normal including kidney size and echo texture with well maintained cortico medullary junction. Doppler showed no evidence of renal artery stenosis. Echocardiogram was normal with no evidence of aortic aneurysm. Urinary VMA and metanephrine levels were not raised and thyroid profile was normal. Patient was started on intravenous labetelol and nitroglycerine infusion but showed no significant drop in mean arterial blood pressure. Later intravenous corticosteroids started to relieve neurogenic edema, improved symptoms and reversed systemic hypertension. Patient was then referred to higher center for further management.

DISCUSSION

Hypertensive emergency is severe elevation of blood pressure with evidence of acute ongoing target organ damage. It consists of accelerated malignant hypertension and hypertensive encephalopathy. [6]

Malignant hypertension is characterized by fundoscopic finding of papilloedema (grade 4 Keith-Wagner) and/or acute retinal haemorrhages and exudates with or without deteriorating renal function and proteinuria. (6-8)
There are multiple causes of hypertensive emergencies including essential hypertension, renal parenchymal diseases, renovascular diseases. Endocrine tumours, drugs like cocaine and amphetamines and CNS disorders including brain tumours are rare causes.\(^{2,8,9}\)

In 1979 Janneta and Gendell introduced the concept of neurogenic hypertension caused by brain tumours due to pulsatile compression of left lateral medulla andpons.\(^{10}\)

This was followed by reports of Camera and Diag claiming brain neoplasm causing malignant hypertension. On resection of this tumour systemic hypertension was fully reversed.\(^{4}\) Similar reports of Naderi and Acarf supports this concept.\(^{5}\)

In our case, renal parenchyma disease and renovascular hypertension were excluded by ultrasound abdomen and Doppler, showing normal echotexture and size of kidney. Echocardiogram was done primarily to exclude aortic aneurysm and valvular heart disease. Thyroid profile was normal and urinary metanephrine and vanillylmandelic levels were not raised. Hence thyroid disorders and pheochromocytoma was ruled out.

Thus, other causes of secondary hypertension excluded, a CT scan showing meningioma or cerebropontine angle tumours compressing pons and third ventricle can be taken as a rare case of brain neoplasm causing neurogenic hypertension. The dramatic response including reversal of systemic hypertension to intravenous corticosteroids supports our hypothesis.

**CONCLUSION**

Brain neoplasms are rare causes of malignant hypertension. They should be included in the differential diagnosis of patients presenting as hypertensive emergency with recent onset hypertension.

**REFERENCES**

5. Naderi S, A Carf; resolution of neurogenic arterial hypertension after sub occipital decompression of chairi 1 malformation. Journal Neurosurg 2005, 102 ; 1147-1150
7. Theodore A. Kotchen, Harrison’s principle of internal medicine; 18\(^{th}\) Edn: 247,p.2058
9. Donald G Victor, Brunwald’s diseases of the heart; 9\(^{th}\) Edn : 45, p.946