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Case Study on A 55-Years old Male With Accelerated Hypertension With Epistaxis

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ABSTRACT: Accelerated hypertension is characterized by extreme rise of systolic blood pressure (BP) (greater than 180 mm Hg) and diastolic BP (over 130 mm Hg at the time of diagnosis) characterised by renal stenosis, narrowing of aorta, irregular usage of anti- hypertensive, pre-eclampsia, autoimmune disorders which accompanied, by bilateral retinal bleeding, nasal bleed, headache, blurred vision, seizures, confusion and irregular heartbeat. About 1-2 cases per 100,000 people have been reported annually in the United Kingdom, over the past 5 decades. And it is mainly assessed by repeated sphygmomanometer readings, ECG, MRI of brain, RFT and serum electrolytes. Accelerated hypertension is primarily treated with labetalol (beta-blocker), nicardipine (calcium channel blocker) and other class of drugs include ACE inhibitors, diuretics and angiotensin receptor blockers. We came across a case of55-years-old male presents in emergency ward with chief complaints of nasal bleed since 4am in the morning. History of excessive sneeze since 1 month.Patient blood pressure was raised by 210/110 mmHg, and provisionally diagnosed as hypertensive urgency with epistaxis. On day 2 and day 3 patient experienced epistaxis, BP was found to be 160/100 mmHg. Patient underwent 2D echo, USG of abdomen, nasoscopy, CT of head and brain, RFT and CUE. Patient was treated with beta-blockers, anti-fibrinolytic, calcium channel blockers, angiotensin-II receptor blocker and nasal decongestant.The final diagnosis was found to be Accelerated Hypertension with Epistaxis. On day 5 patient vitals was stable and got discharged in stable condition.

KEYWORDS: Accelerated Hypertension, Epistaxis, Sphygmomanometer, Labetalol, Nicardipine

I. CASE PRESENTATION

HISTORY OF PRESENT ILLNESS:

A 55-years-old male presents in emergency ward with chief complaints of nasal bleed since 4am in the morning. History of excessive sneeze since 1 month. Patient blood pressure was raised by 210/110 mmHg and GRBS was found to be 134mg/dl, based on the physical and general examination patient was provisionally diagnosed as hypertensive urgency with epistaxis. Patient was treated with Inj. Labetalol – 20mg/stat/IV followed by tab. Labetalol 200 mg /BD/PO and BP got reduced to 167/108 mmHg , Inj. TranexamicAcid – 500mg/stat/IV followed by BD, Botroclot (squeezing technique) – nasal drops – 4 drops/stat followed by 3 drops/QID, Tab. Allegra-M 1tab/HS and Tab. Sartel –ln - 40/10mg/OD/PO. On Day 2 patient experienced spontaneous nasal bleed at 6AM and BP was 140/90 mmHg , after 30 min patient experienced left sided predominantly – pulsatile bleed and BP was found to be 130/80 mmHg. PatientBotroclot drops were instilled but bleed + and right nasal cavity packed with merocel with cannula. On Day 3 patient experienced Right nostril bleed followed by epistaxis + and had fresh complaints of giddiness and fall, BP was found to be 160/100

mmHg. Patient underwent 2D echo and USG of abdomen. The final diagnosis was found to be Accelerated Hypertension with Epistaxis. On Day 4 nasal bleeding was controlled, patient underwent Nasoscopy, CT of Head and Brain, CBP, Serum Electrolytes, ECG, Thyroid profile, RFT and CUE . On Day 5 patient vitals were normal and got discharged in stable condition.

PAST MEDICAL HISTORY:

No significant past medical history.

FAMILY HISTORY:

No significant family history.

SOCIAL HISTORY:

Patient is occasionally alcoholic.

ALLERGIES:

No known medicine, food and environmental allergies.

GENERAL EXAMINATION:

Pt – c/c

Temp – Afebrile

BP – 210/110 mmHg → 167/108 mmHg

PR – 79 bpm

RR – 20 /min

CVS – S1+S2+

RESP – BAE +

CNS – NAD

SPO2 – 96%

P/A – Soft

GRBS – 134 mg/dl

CBG – 160 mg/dl ↑

LABORATORY INVESTIGATIONS:

CBP

Hb – 13.2 gm% (12-14gm %)

RBC – 4.8 million/cmm(4-6M/cmm)

WBC – 9,100cells/cmm (4,000 – 11,000)

PLT – 2.51 lakhs/cells (1.4 lakhs – 4.4lakhs)

BT – 2 min 15 sec

CT –4 min 10 sec

RENAL FUNCTION TESTS:

Serum creatinine – 0.8 mg/dl (0.7 – 1.2 mg/dl)

Blood urea – 28 mg/dl (7-30 mg/dl)

THYROID PROFILE - Normal

CUE:

Pus cells – 6-8 /hpf

Epithelial cells – 2-4 /hpf

ANALYSIS OF SERUM ELECTROLYTES:

Sodium - 146 mmol/l

Potassium- 4.2mmol/l

Chloride - 108 mmol/l

SCANS:-

2D ECHO

Grade - 1 diastolic dysfunction

Good LV/RV (EF – 66%)

USG ABDOMEN

Cholelithiasis (target 5mm).

NASOSCOPY

Nasal cavity – normal

Nasoscope was inserted via the right middle nasal meatus

Impression - No abnormality was detected

CT OF HEAD AND BRAIN

Ventricular systemic and sulcal spaces are prominent – mild diffuse cerebral atrophy

Small vessel ischemic changes noted in B/L peri ventricular white matter

B/L maxillary, right ethmoid, right sphenoid sinusitis.

ECG

Sinus rhythm

Abnormal inferior Q wave.

ASSESSMENT:

Based on the Physical, General examination, Laboratory findings and Scan evidences, the patient was diagnosed with Accelerated Hypertension with Epistaxis.

TREATMENT:

S.No	DRUGS	DOSE	FRQY	ROA	D1	D2	D3	D4	D5
1	INJ. LABETALOL ↓ TAB. LABETALOL	20 MG ↓ 200 MG	STAT ↓ BID	IV ↓ PO	✓	✓	✓	×	×
2	INJ. TRANEXAMIC ACID	500MG	STAT ↓ BD	IV	✓	✓	✓	✓	✓
3	TAB.FEXOFENADINE + MONTELUKAST	120/10 MG	HS	PO	✓	✓	✓	✓	✓
4	HEMOCOAGULASE NASAL DROPS	4 DROPS ↓ 3 DROPS	STAT ↓ QID	PN	✓	✓	✓	✓	✓
5	TAB. TELMISARTAN + CILNIDIPINE	40/10 MG	OD	PO	✓	✓	✓	×	×
6	TAB.AMOXYCILLIN + CLAVULANIC ACID	500 MG/ 125 MG	BD	PO	×	×	✓	✓	✓
7	INJ. ETAMSYLATE	1 AMP	STAT	IV	×	×	✓	✓	✓
8	SYP. SUCRALFATE + OXETACAINE	10ML	TID	PO	×	×	✓	✓	✓
9	TAB. ALPRAZOLAM	0.5 MG	HS	PO	×	×	✓	✓	✓
10	INJ. OPTINEURON	1 AMP IN 100 ML NS	BD	IV	×	×	✓	✓	✓
11	TAB. FERROUS ASCORBATE + FOLIC ACID	100/1.5 MG	BD	PO	×	×	✓	✓	✓
12	TAB. TELMISARTAN + METOPROLOL	40/80 MG	OD	PO	×	×	×	✓	✓

II. DISCUSSION:

DEFINITION:

Malignant or so-called accelerated hypertension is characterized by extreme rise of systolic blood pressure (BP) (greater than 180 mm Hg) and diastolic BP (over 130 mm Hg at the time of diagnosis) accompanied, either with or without papilledema, by bilateral retinal bleeding and nasal bleed. [1]

EPIDEMIOLOGY:

Malignant hypertension is particularly uncommon in the general population. About 1-2 cases per 100,000 people have been reported annually in the United Kingdom, over the past 5 decades. [1]

A recent study revealed that between 2006 and 2013, the expected amount of visits to the emergency department (ED) for this disease increased by over twice the rate per million of adult ED visits. [2]

ETIOLOGY: [2]

Malignant hypertension, often known as hypertensive crisis, has various causes, such as the following:

Noncompliance with medication regimen

Renal artery stenosis, Takayasu arteritis, and polyarteritisnodosa are examples of vascular disorders.

Hemolytic-uremic syndrome, systemic sclerosis, glomerulonephritis, tubulointerstitial nephritis, and systemic lupus erythematosus are examples of renal parenchymal diseases.

Endocrine disorders, including renin-secreting tumors, Cushing disease, pheochromocytomas, and primary hyperaldosteronism

Aortic coarctation; drug use or exposure to other substances, such as cocaine, phencyclidine, sympathomimetics, cyclosporine, and erythropoietin

Withdrawal from antihypertensive medications

Amphetamine-containing substances

Diseases of the central nervous system, including brain hemorrhage, cerebral infarction, and head injury

Other etiological factors include - Collagen-vascular diseases (such periarteritisnodosa, systemic sclerosis, and systemic lupus erythematosus) and preeclampsia. [3]

CLINICAL FEATURES: [4]

Hypertension is asymptomatic, thus signs and symptoms of a hypertensive emergency would be specific to the organ systems involved.

Cerebrovascular manifestations - Hypertensive encephalopathy is most typically characterized by global symptoms such as headache, nausea, and vomiting, which proceed to visual abnormalities with or without retinal alterations, focal neurological impairments, seizures, coma, and death.

Cardiovascular symptoms include chest pain and dyspnea, especially in the presence of inadequate cardiac output, pulmonary edema, or congestive heart failure.

Renal manifestations - After diabetes mellitus, hypertension is the second leading cause of chronic renal impairment. The first indications of renovascular disease will be reduced glomerular filtration rate (GFR) and microproteinuria.

Severe renal failure can cause a variety of clinical symptoms, including general malaise, uremic delirium, and fluid overload.

PATHOPHYSIOLOGY:[5]

Blood pressure is regulated by the balance between cardiac output and systemic vascular resistance. Blood pressure regulation is a complex process which involves a variety of physiological processes, including arterial baroreceptors, the renin-angiotensin-aldosterone system, endothelins,atrial natriuretic peptide, and mineralocorticoid and glucocorticoid hormones. These complicated mechanisms work together to regulate the amount of vasodilation or vasoconstriction in the systemic circulation, as well as the retention or excretion of salt and water, in order to maintain an adequate blood volume.

Dysfunction in any of these mechanisms can result in hypertension. This could be due to increased cardiac output, systemic vascular resistance, or both.

DOES HYPERTENSION CAUSE EPITAXIS? [6]

Although there isn't always a direct connection between high blood pressure and epitaxis, several studies have shown one.

Researchers found that those with hypertension are more likely to experience nosebleeds that may require medical care, in comparison to those with normal blood pressure.

Another study found that while hypertension is not typically the cause of epitaxis, it can make nosebleeds more difficult to treat.

DIAGNOSIS: [7]

A systematic evaluation of the heart, including ECG and echocardiography, as well as a systematic brain MRI.

Repeated sphygmomanometer readings are typically used to diagnose hypertension.

Other examinations include Urine albumin-to-creatinine ratio, blood sugar and hemoglobin levels, potassium in serum, Serum creatinine (in conjunction with the predicted GFR), Calcium in serum, Panel of fasting lipids. [8]

TREATMENT:

MHT is a hypertensive emergency which demands immediate antihypertensive medication due to the increased risk of renal failure, stroke, myocardial infarction, and heart failure.

The initial aim of the treatment is to reducing diastolic blood pressure to about 110 mmHg.

The most commonly used intravenous medications were labetalol, sodium nitroprusside, nicardipine, nitrates, and furosemide. [9]

The first line treatment used are labetalol and nicardipine. Alternatively, nitroprusside and urapidil can be used as a safe and effective treatment for MHT. [10]

Labetalol (initially 20–50 mg for at least a minute. Titrate infusion starting at 2 mg/min OR repeat every 5 minutes up to a maximum of 200 mg. Next, titrate the infusion at a rate of 2 mg/min) [11]is a nonselective beta-adrenergic receptor and alpha-1 adrenergic receptor blocker. Its primary advantage is to maintain cardiac output while also reducing peripheral resistance while improving cerebral, renal, and coronary blood flow.

Nicardipine (infusion of 2.5–5 mg every hour, increase by 0.5 mg every 15 minutes, up to a maximum of 15 mg per hour, based on response)[11]is a calcium channel blocker derived from dihydropyridine that has cerebral and coronary vasodilatory effects. It enhances coronary blood flow and stroke volume.

III. CONCLUSION:

In my case report uncontrolled hypertension leads to epistaxis. And It is a rare symptom of uncontrolled hypertension, after giving the proper anti-hypertensives, blood pressure was controlled and bleeding was reduced. Patient was stable after 5 days and got discharged.

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