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MALIGNANT MCA INFRACT: A RARE CASE REPORT

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ABSTRACT
Stroke is the third leading cause of death, ranking lower only to cardiac disease and cancer. Ischemic stroke accounts for 87% of strokes and occurs when a clot or a thrombus blocks a blood vessel, cutting off blood flow to a part of the brain. If large areas of brain are affected, space-occupying oedema may result, leading to rapid neurological deterioration, coma and death. Malignant middle cerebral artery infarction (MMCAI) is a life-threating ischaemic stroke involving the whole middle cerebral artery (MCA) territory and comprises up to 10% of MCA infarction. Here we report a case of patient with weakness of right UL and LL with slurred speech , it was later diagnosed as a case of malignant right MCA infract with the complication of accelerated hypertension , which when treated with anti hypertensives lead to hyponatremia induced seizures and development of left MCA infract.

KEYWORDS: MCA infracts, hyponatremia seizures, leviteracetam, aspirational pneumonia.

INTRODUCTION
Middle cerebral artery (MCA) stroke describes the sudden onset of focal neurologic deficit resulting from brain infarction or ischemia in the territory supplied by the MCA. Generally, Malignant MCA infarction is the term used to describe rapid neurological deterioration due to the effects of space occupying cerebral oedema following middle cerebral artery stroke .The MCA is by far the largest cerebral artery and is the vessel most commonly affected by cerebrovascular accident. The MCA supplies most of the outer convex brain surface, nearly all the basal ganglia, and the posterior and anterior internal capsules.

CASE REPORT
An 89 years old female patient was admitted to the neurology department with weakness of right UL and LL with slurred speech. No past history of headache, vomiting and fever. The patient is neither hypertensive nor diabetic. No past medical history of CAD and COPD. The patient has a history of decreased vision since 5 years.

Clinical findings, Diagnostic assessment and Therapeutic intervention
MRI brain (DWI) report: showed acute infracts in left capsule ganglionic region, para ventricular area, old infracts in bilateral capsule ganglionic region and right corona radiate. Ischemic changes in pons and periventricular white matter.

CT Report: Atheromatous vascular calcifications in the bilateral cavernous ICA's.

Age related diffuse cerebral and cerebellar atrophy.

Diffuse ventriculo megaly out of proportion to adjacent sulcal spaces, this shows inappropriate clinical conditions NPH could be a possibility.

Small vessel ischemic changes in bilateral peri ventricular and deep frontoparietal white matter. Chronic lacunar infracts in bilateral corona radiate, capsule ganglionic regions and right thalamus.

Cardiology report
Sclerotic aortic valve, grade 1 diastolic dysfunction, concentric LVH.

CT brain report
Alternate days showed sun acute infracts in left capsule ganglionic region and para ventricular area. Right atlanto axial degenerative arthritis.

Lipid Profile

Cholesterol	Method	Result	Biological reference range
LDL cholesterol	Polymer detergent	132 mg/dl	100-129 mg/dl
Cholesterol/HDL		5.1	4.1-4.5

Renal Function Tests

Electrolytes	Method	Result	Biological reference range
Serum sodium	Ion selective electrode	127 mmol/l	135-155mmol/l
Serum chloride	Ion selective electrode	3.3 mmol/l	3.5-5.5 mmol/l
Serum potassium	Ion selective electrode	94 mmol/l	98-106 mmol/l

Blood Reports

Hemogram	Method	Result	Biological reference range
TLC	Cell Counter Pendra ES60	25000 cells/cumm	4000-11000 cells/cumm
ESR	West Gern	90 MM/HR	5-20 MM/HR
MCHC	Cell Counter Pendra ES60	31%	32%-36%

Biochemistry

Liver function tests	Method	Result	Biological reference range
Total serum bilirubin	Dizazotized Sulphanilic Acid	1.4 mg/dl	0.3-1 mg/dl
Serum direct bilirubin	Dizazotized Sulphanilic Acid	0.4 mg/dl	0.1-0.3 mg/dl
Serum indirect bilirubin	-	1.0 mg/dl	0.2-0.7 mg/dl
SGOT	International Federation Clinical Chemistry	45 units	5-40 units
Serum protein	Biuret	5.4 gm/dl	6.6-8.7 gm/dl
Serum albumin	Brom Cresol Green	2.8 gm/dl	3.5-5.3 gm/dl

After five days there was increase in serum chloride levels (117 mg/dl). CT brain report Alternate days showed sun acute infracts in left capsule ganglionic region and para ventricular area. Right atlanto axial degenerative arthritis. Thyroid function tests decreased T3 (0.2ng/dl), TSH (0.3 mIU/ml). Doppler study of extra cranial carotid and vertebral arteries showed intima media thickening in the bilateral CCA and ICA, fibro fatty plaques in right ICA causing 25% stenosis, left carotid buld causing 44%stenosis in left ICA causing 18 % Stenosis. Fibro fatty, calcific plaques in right CCA causing 27% stenosis.

CASE DISCUSSION

Patients with stroke involving large vessels, including the middle cerebral artery, account for almost half of all patients with ischemic strokes and have an increased risk for poor outcomes and mortality at 6 months. Middle cerebral artery syndrome is a condition whereby the blood supply from the middle cerebral artery (MCA) is restricted, leading to a reduction of the function of the portions of the brain supplied by that vessel: the lateral aspects of frontal, temporal and parietal lobes, the corona radiata, globus pallidus, caudate and putamen.

The MCA is the most common site for the occurrence of ischemic stroke. Contralateral hemiparesis and hemisensory loss of the face, upper and lower extremities is the most common presentation of MCA syndrome. In this casr the patient is having hemiperisis and slurred speech. Hypertension is a widely recognized risk factor for stroke and Hypertension is present in up to 84% of patients presenting with acute stroke, A severe increase in blood pressure that can lead to a stroke. Extremely high blood pressure - a top number (systolic pressure) of 180 millimeters of mercury (mm Hg) or higher or a bottom number (diastolic pressure) of 120 mm Hg or higher — can damage blood vessels. Uncontrolled high blood pressure can lead to stroke by damaging and weakening your brain's blood vessels, causing them to narrow, rupture or leak. High blood pressure can also cause blood clots to form in the arteries leading to your brain, blocking blood flow and potentially causing a stroke. In this case the blood pressure of the patient is 210/100 mmHg. Which is treated with anti hypertensives like mannitol,calcium channel blokers and beta blockers the patient developed hyponatremia(121 mEq/l). hyponatremia (135 mEq/l) usually represents a relative excess of water in relation to sodium in the blood. Acute or severe electrolyte imbalance can manifest with rapidly progressive neurological symptoms or seizures. Seizures are more frequently observed in patients with sodium disorders (especially hyponatremia). Rapidly evolving electrolyte disturbances are more likely to cause seizures than those developing more gradually. Hyponatremic seizures represent an ominous sign and hence a medical emergency, as they are associated with high mortality. seizures induced by hyponatremia can be controlled by increasing the serum sodium concentration. The most common treatment for hyponatremia consists of hypertonic saline (3%), which produces a rapid reduction in brain volume and intracranial pressure. An increase in serum sodium to values of 120 mEq/L to 125mEq/L should be the target of therapy. Of note, more aggressive treatment of hyponatremia with hypertonic saline solution carries the risk of shrinkage of the brain leading

to osmotic demyelination syndrome manifesting with severe neurologic symptoms such as quadriplegia, pseudobulbar palsy, coma, and even death.

Treatment

Treatment options

Fibrinolytics: Intravenous recombinant- tissue-plasminogen activator (rtPA) is approved to treat eligible patients with acute ischemic stroke within 4.5 h of stroke onset.

Antiplatelets like aspirin, clopidogrel and anti coagulants like warfarin and new oral anti coagulants.

Antihypertensives: Hypertension is a risk factor for recurrent strokes and is often managed with thiazide diuretics, calcium-channel blockers, angiotensin-converting enzyme inhibitors (ACE inhibitors), and angiotensin receptor blockers (ARBs). A number of studies, including the Heart Outcomes Prevention Evaluation (HOPE) study and the Perindopril Protection Against Recurrent Stroke Study (PROGRESS), support the use of ACE inhibitors with or without the combination of a thiazide diuretic to reduce the reduce the risk of stroke recurrence.Beta-blockers are generally considered second-line agents but are often used for rate control.

Treatment Given

Here the patient is treated with aspirin (325 mg) and clopidogrel (75 mg) along with intravenous mannitol (75ml) as the patient developed a very high blood pressure of 210/110 mm of Hg. Patient is then treated with antihypertensives like telmisarton (40mg) and amlopidine (5mg) This lead to telmisarton induced hyponatremia. hyponatremia is tried to treat with 3% Nacl infusion and tolvaptan (15 mg). Despite of the treatment for hyponatremia patient developed generalized seizures. seizures are treated with anti epileptic drug - levetiracetam (250 mg in 100 ml NS). After treating with the levetiracetam the patient became drowsy and developed aspirational pneumonia leading to deterioration of Glassgow Coma Scale (GCS). Aspiration pneumonia, resultant from penetration of food, saliva, and gastric acid, has very serious ramifications, including high mortality, increased length of hospital stay, and poor functional outcome. The above treatment is continued along with Noninvasive ventilation (NIV) and later patient developed new onset of left sided hemiplegia for which the second time MRI brain was done and found new onset right MCA infract with mass effect on MRI brain. Patient has no eye opening and is suffering from quadriplegia. The patient newly developed hypotension and started on ionotropic support. GCS is even more deterioted and patient is kept on mechanical ventilation.

CONCLUSION

A complicated case report with left MCA induced hypertension which on treatment with anti hypertensives like telmisartan lead to telmisarton induced hyponetremia which lead to hyponatremia induced seizures. Treatment of seizures with anti epileptic drugs like leviteracetam lead adverse effects like drowsiness and aspirational pneumonia. later on patient developed right MCA infract leading to quadriplegia. As the patient condition is getting worse, the patient is referred to higher centers for advanced and appropriate treatment of her deteriorating condition.

REFERENCES

1. Huttner, H. B., & Schwab, S. Malig-nant middle cerebral artery infarction: clinical characteristics, treatment strategies, and fu-ture perspectives. The Lancet Neurology, 2009; 8(10): 949-958.
2. Lambrinos, A., Schaink, A. K., Dhalla, I., Krings, T., Casaubon, L. K., Sikich, N., Hill, M. D. Mechanical Thrombecto-my in Acute Ischemic Stroke: A Systematic Review. Canadian Journal of Neurological Sciences, 2016; 43(4): 455-46.
3. Bogousslavsky J, Barnett HJM, Fox AJ, Hachinski VC, Taylor W. Atherosclerotic disease of the middle cerebral artery. Stroke, 1986; 17: 1112-1120.
4. Wong KS, Li H, Lam W W, Chan YL, Kay R. Progression of middle cerebral artery occlusive disease and its relationship with further vascular events after Stroke. Stroke, 2002; 3: 3532-3536.
5. Akins PT, Pilgram TK, Cross DT, Moran CJ: Natural history of stenosis from intracranial atherosclerosis by serial sngiography. Stroke, 1998; 29: 433-438.
6. Arenillas JF, Montaner J, Abilleira S, González-Sánchez MA, Álvarez-Sabín J. Progression and clinical recurrence of symptomatic middle cerebral artery stenosis: a long-term follow-up transcranial doppler ultrasound study. Stroke, 2001; 32: 2898-2904.
7. Malferrari G, Bertolino C, Casoni F, Zini A, Sarra VM, Sanguigni S et al. For the Eligible group and SINV group: The Eligible Study: Ultrasound assessment in acute ischemic stroke within 3 Hours. Cerebrovasc Dis., 2007; 24: 469-76.
8. Shin DH, Lee PH, Bang OY. Mechanism of recurrence in subtypes of ischemic stroke: a hospital-based follow-up study. Arch Neurol, 2005; 62(8): 1232-1337.
9. Bang OY, Heo JH, Kim JY, Park JH, Huh K. Middle cerebral artery stenosis is a major clinical determinant in striatocapsular small, deep infarction. Arch Neurol, 2002; 59: 259-263.
10. Corston RN, Kendall BE, Marshall J: Prognosis in middle cerebral artery stenosis. Stroke, 1984; 15: 237-241.
11. Hinton RL, Mohr JP, Ackerman RH, Adair CB, Fisher CM. Symptomatic middle cerebral artery stenosis Ann Neurology, 1979; 5: 152-157.
12. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4828558.