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A case report on acute non hemorrhagic infarct in midbrain

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Abstract

Introduction: Non-hemorrhagic areas of dead tissue, in any case called ischemic areas of dead tissue, are the aftereffect of the intense interference of blood stream to a space inside the mind. The typical reason for a no hemorrhagic dead tissue is the impediment of an intracranial corridor by a thromboembolism. Non hemorrhagic areas of localized necrosis contrast from hemorrhagic areas of localized necrosis that are related with intra parenchymal mind discharge. Ischemic strokes are by a long shot the most well-known classification, addressing almost 90% of all strokes, and around 10% of all ischemic strokes occur in the cerebrum stem, as per the National Stroke Association. Strokes are the main source of long haul inability in the United States, and almost 800,000 U.S people experience a stroke each year.

Patients History: An 80 years old male was admitted in neuro ICU. A.V.B.R.H. Sawangi(m) Wardha on date 17/07/2021. Patient's complaint of fall at home around 8.00am, 07/07/2021. On date 08/07/2021 in afternoon patient developed left side weakness and slurred speech, fatigue. After CT scan patient diagnosed with acute non hemorrhage infarct in midbrain. No history of fever, cough, breathlessness and no history of Hypertension, Diabetes, and TB etc. **Clinical findings:** The patient had done all necessary investigation by physician order. **Medical Management:** Patient treated with IV. Fluids RL, NS, RT Feed 200ml/2hourly, antacids, antibiotics, analgesic, antipyretic. **Nursing Management:** Administered IV fluid , RT Feed 200ml/2hourly, monitor vital sign 2 hourly. Administered medication doctor's order **Conclusion:** Timely treatment and management of acute on non hemorrhage infarct in midbrain

Keywords: Hemorrhages, Infarct in midbrain, hematoma, stroke

Introduction:

The brainstem is made out of the midbrain, the pons, and the medulla oblongata, arranged in the back piece of the mind. It is an association between the frontal cortex, the cerebellum, and the spinal line. Embryo logically, it creates from the mesencephalon and part of the rhomb encephalon, all of which start from the neural ectoderm. The brainstem is coordinated inside in three laminae: tectum, tegmentum, and premise. Dim matter in the brainstem is found in bunches up and down the brainstem to framing generally the cranial nerve cores, the pontine cores, and the reticular development. White matter as different rising and slipping plots can be discovered fundamentally in the premise lamina,

which is the most front part.[1]In intense stroke, the differential determination of drain distinguished on registered tomography (CT) filter goes from hemorrhagic infarct (HI), essential intracerebral discharge (ICH) to drain from venous dead tissue. Hello, or hemorrhagic change of an infarct, happens in around 33% of instances of ischemic stroke. At the point when an infarct is promptly trailed by the event of petechial discharge in a similar blood vessel domain, the analysis of HI is effectively made. Notwithstanding, when mind imaging is deferred after the beginning of the patient's stroke manifestations, a mistaken finding of ICH might be made if the discharge seems blended on CT.[2] The rising reticular initiating framework (ARAS) is known to assume a fundamental part in keeping up with excitement and cognizance. The relationship between ARAS injury and cognition impediment has been explained in clients with many mind pathologies such as stroke and terrible cerebrum injury. And Cerebral hypoxic-ischemic injury. The examinations for assessment of the ARAS incorporate positron discharge tomography, electroencephalography, transcranial attractive incitement, and dissemination tensor tractography (DTT). Among these, DTT, got from dispersion tensor imaging (DTI), has been recommended better than different modalities. DTT empowers three-dimensional representation of white matter neural lots and can remake the ARAS exhaustively by division (for example the lower dorsal ARAS, associating the pontine reticular arrangement to the intralaminar thalamic cores; the lower ventral ARAS, interfacing the pontine reticular development to the nerve center; and the upper ARAS, interfacing the intralaminar thalamic cores to the cerebral cortex).[3]The climbing reticular enacting framework (ARAS) is known to assume a fundamental part in keeping up with excitement and cognizance. The relationship between injury of the ARAS and disability of awareness has been accounted for in patients with different mind pathologies including stroke, awful cerebrum injury, and hypoxic-ischemic mind injury. The examinations for assessment of the ARAS incorporate positron emanation tomography, electroencephalography, transcranial attractive incitement, and dispersion tensor tractography (DTT). Among these, DTT, got from dispersion tensor imaging (DTI), has been recommended better than different modalities. DTT empowers three-dimensional perception of white matter neural plots and can recreate the ARAS exhaustively by division (for example the lower dorsal ARAS, interfacing the pontine reticular arrangement to the intralaminar thalamic cores; the lower ventral ARAS, associating the pontine reticular development to the nerve center; and the upper ARAS, associating the intralaminar thalamic cores to the cerebral cortex).[4]

Patient specific information: An 80 years old male was admitted in neuro ICU. A.V.B.R.H. Sawangi (M) Wardha on date 17/07/2021 . Patient’s complaint of fall at home around 8.00am, 07/07/2021, on date 08/07/2021 in afternoon patient developed left side weakness and slurred speech, fatigue. After CT scan patient diagnosed with acute non hemorrhage infarct in midbrain. No history of fever, cough, breathlessness and no history of Hypertension, Diabetes, TB etc.

Primary concern and symptoms of patients: A 80 years old male was admitted in Neuro ICU AVBRH on date 17/07/2021 with complaint of fall at home around 8.00am, 07/07/2021, on date 08/07/2021 in afternoon patient developed left side weakness and slurred speech, fatigue. After CT scan patient diagnosed with acute non hemorrhage infarct in midbrain.

Medical, family and psycho-social History: Patient had No previous history of Hypertension, Diabetes, and TB etc. Patient belongs to nuclear family. There are 3 members are alive including patient. All family members are healthy. All family members are maintained good relationship with Doctor and Nurse.

Relevant past Interventions with outcome: No past interventions, the patient general conditions were poor.

Clinical findings:

General examination
State of health: unhealthy
General condition – not satisfactory
State of consciousness: conscious
Body built- slim
Hygiene: poor
Height: 157cm
Weight: 40 kg
Vital parameter:
Blood pressure: 150/90mhg
Temperature: 98.60^{o} F
Pulse: 76 beats/min.

Respiration: 20breath/ min.
SPO_2:90%
CVS – S_1 S_2
CNS - E2V1M4; BIL pupil RTL

Diagnostic assessment: Physical review on the basis of patient history, physical examination , CT Brain and MRI Brain with venogram, kidney and liver function test , CBC test , D-Dimer , Homocysteine ,done and other all blood investigation done .

CT Brain- Findings - acute non hemorrhage infarct in midbrain.
MRI Brain with venogram – Non hemorrhage infarct in RT. MCA is sigmoid and transverse in sinus partial signal flow.
D-Dimer value- 1098 ng/ml.

Diagnostic tests:
Blood urea = normal
Creatine – serum = slightly decrease
Serum- Potassium = increase
Sodium (Na+) = Normal
Complete blood count
Hb% = decrease

Total RBC count = decrease
Total platelet count =decrease
Total WBC count =normal
MCV (mean corpuscular volume)= normal
MCH (mean corpuscular hemoglobin) = normal
MCHC (mean corpuscular hemoglobin concentration) = normal
Physical review on the basis of patient history, physical examination , CT Brain and MRI Brain with venogram, kidney and liver function test , CBC test , D-Dimer , Homocysteine ,done and other all blood investigation done .

Therapeutic Interventions:
Medical management: IV Fluids- RL & NS, Inj. Optineuron 4ml + 100ml NS OD , Inj.pan 40 mg BD . Inj piptaz 4.5 gm TDS. Inj.neomol 100 ml TNS . . Tab . Ecosprin Av 150 OD. Tab . Emset 4 mg TDS . Tab . Dolo 650 mg TDS. Inj .ceftraxon 1 ml BD .

Follow-up and outcomes:

Significant medical follow-up and other test outcomes: Follow-up at weekly interval with necessary investigations was done.

Adherence to action and tolerability: Patient adhered well to recommended treatment.

Discussion:
Infarction associated with thalamus and midbrain due to occlusion of artery of Percheron is difficult to diagnose clinically due to the variability involvement of thalamus and midbrain, and ambiguous clinical presentations. There are many differentials to keep in mind when a patient presents with bilateral lesions of the thalamus. In this case study, patient developed left side weakness and slurred speech, fatigue[5]. A number of studies on brain related conditions due to traumatic injuries were reported[6-10]. Articles from Global Burden of Disease study were reviewed[11-12]. After CT scan patient diagnosed with acute non hemorrhage infarct in midbrain that needed urgent medical attention. Some cases patient may present with hemiplegia, ataxia and oculomotor disturbance suggesting bilateral thalamic infarction (thalamo-peduncular syndrome) . A variety of clinical signs including dysarthria, impaired convergence, retraction of eyelids, amnesia, ataxia, involuntary movement of limbs have been described . In this case, CT Brain- Findings - acute non hemorrhage infarct in midbrain. MRI Brain with venogram – Non hemorrhage infarct in RT. MCA is sigmoid and transverse in sinus partial signal flow. Patient took treatment regularly and his outcome was moderate.[5]

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