Top of Basilar Artery Infarction: A Case Series from Sudan

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Case Report

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Abstract

The posterior circulation represents 20% of blood supply of the brain and its occlusion commonly by embolism cause brainstem, cerebellar and lower cerebral infarction. The clinical presentation varies from mild innocent symptoms leading to sever neurological deficit or death. Time of intervention is vital commonly with antithrombatic drugs or through intervention. Here, we report two Sudanese patients who had a complicated medical sequence over months ended as top of basilar artery occlusion received anticoagulants and supportive therapy according to their condition showed a variable recovery over weeks.

Introduction

The right and left vertebral arteries united together at the level of pontomedullary junction to form the basilar artery which joined to the brainstem via penetrating median, paramedian, short and long circumferential branches that's connected by anastomotic channels in majority of people (1)(2). As a whole Posterior circulatory arteries occlusion estimated to be the fifth of all strokes; and for basilar artery occlusion the clinical presentation is commonly of non-specific prodromal symptoms such as vertigo or headaches, followed by the hallmarks of Basilar artery occlusion, including decreased consciousness, quadriparesis, pupillary, oculomotor abnormalities, dysarthria, and dysphagia; necessitate its exclusion in any acute brain stem insult as the result may be devastating stroke (2)(3).The time from symptoms to diagnosis is crucial as early intervention to reopen the artery via either by intravenous thrombolysis, intra-arterial thrombolysis or mechanical endovascular technique may improve the outcome(3).

Case 1

A 36 years old female presented 2 days after being discharged from the hospital where she was admitted for 1 month due to snake bite complicated by DIC, compartment syndrome, sepsis and renal impairment, received FFP, Antibiotics, underwent fasciotomy of her Left arm, and discharged on good condition; with Sudden onset Left side weakness, reached maximum intensity in the same day and became even unable to walk that's not preceded by a headache or loss of consciousness; associated with Aphasia, difficulty in swallowing of her meals without nasal regurgitation and deviation of her Right eye to the Left side with no loss of her sight. There is no mouth deviation, drooling of saliva, sphincteric disturbances or sensory symptoms and no fever. Her past medical history otherwise is clear a part of irregular cycles.

On examination: we found a lady with fasciotomy wound on her left upper limb, Bp 90/60, pulse 100 beat per minute regular, with normal examination findings on her chest, pericardium and abdomen; nervous system examination revealed GCS 10/10 (Aphasic) ,on cranial nerves examination we found 6th cranial nerve palsy and bulbar palsy, no neck stiffness, upper limbs: normal tone power and reflexes on right side, within left limb there is reduction of power in a pyramidal fashion, hypotonia and hyper reflexia. Lower limbs: normal tone power and reflexes on right, reduction of power in a pyramidal fashion hypotonia and

hyper reflexia within left, no clonus, upgoing planter response and we had a difficulty in sensation and coordination tests. Funduscopic examination not done.

Investigation showed Twbcs 11.2 (neutrophils 78%),Haemoglobin 9.1(normocytic normochromic),Platelets 473,RFT :urea 9 S.Creatinine 1.3 electrolytes normal, RBG 104 mg /dl, Echo:mild LV systolic dysfunction EF 45 %, AF, no intra cardiac shunt thrombus or vegetation; Brain MRI confirm a diagnosis of top of basilar artery infarction. The patient received enoxaparin weight 1mg B.D,i.v fluids, PPI, Antibiotics and discharged on good condition 14 days later (left side power grade 4) on Rivaroxaban 20mg O.D.

Case 2

-A 85 year old male Known hypertensive for 20 years on amlodipine 10 mg,end stage renal disease for 6 months on regular haemodialysis, presented with high grade continuous fever associated with rigor, confusion and decrease level of consciousness for 2 days; at the same times his family noticed that he was unable to speak, swallow or to move his limbs there was no mouth deviation or history of seizure.

-On examination: the patient looked unwell with NG tube and permeant haemodialysis catheter, pulse 80,BP 160/100, cardiovascular system revealed tender hepatomegaly with bilateral lower limb oedema; neurological examination:GCS6/15 (Aphasic+spontinous eye opening),Bilateral cataract, no neck stiffness or carotid bruit, right side showed hypotonia with normal reflexes, left side findings include hypotonia and hyper reflexia with extensor planter reflex. Power, sensation and coordination couldn't be assessed (locked in syndrome), fundoscopy not done.

-Investigations shows: Twbcs 17, Haemoglobin 6.5 (normocytic normochromic), Platelets 131, RBG 184,RFT:urea142 mg/dl,S.creatinine 7.2 mg/dl,Na 122mmol/l,K5 mmol/l, INR 1.7. MRI brain, MRA & MRV confirm a diagnosis of top of basilar artery infarction.

-the patient received i.v antibiotics renal dose, dual antiplatelet therapy, enoxaparin 4,000 i.u O.D, blood transfusion, and frequent session of haemodialysis. Later the patient developed bilateral epistaxis and bleeding per mouth manged with fresh frozen plasma, heparin free haemodialysis session and stopping of enoxaparin and antiplatelets. Patient discharged 14 days later static on locked in with GCS 6/15.

Discussion

Herein we report two cases of top of basilar artery infarction. Two vertebral arteries form the basilar artery at the pontomedullary junction. It flows near the brainstem and ends at the ponteno-mesencephalic sulcus in the majority of people. It's divided into a superior branch and an inferior branch [4–6]. The basilar artery can be affected by different mechanisms, for example; basilar artery occlusion due to thrombosis, or thrombus embolism [7]. Top of the Basilar Artery Syndrome is due to the thromboembolic occlusion of the top of the artery. Risk factors of strokes in general are divided into modifiable; hypertension, hyperlipidaemia, diabetes mellitus and many more, or nonmodifiable; age, sex, genetics and

race [8]. Basilar artery infarction clinical presentation can range from mild symptoms such as vertigo, to more severe complications with a poor prognosis such as muscle weakness, oculomotor abnormalities and dysarthria [7]. In the first case, our 36-year-old female patient was bitten by a snake and was admitted to the hospital for a month. After two days of discharge, she was brought to the emergency room as she developed sudden left side weakness that made her unable to walk. The patient developed symptoms consistent with basilar artery occlusion as we found later on imaging. Cerebral infarction association with snake bites has been reported less frequently, and it is often due to cerebral or subarachnoid bleeding due to depletion of clotting factors [9]. Snakes' bites secrete enzymes in the body [10]. These enzymes contribute for cerebral infarction due to variant mechanisms such as:

- Thrombotic occlusion of large vessels, vasculitis and consumption coagulopathy [10].
- Direct anticoagulant effects that can lead to cerebral infarction [9].
- Hypotension due to hypovolaemia predisposing for watershed infarcts [11].
- Direct cardiotoxic effects leading to dysrhythmias that facilitate thromboembolism due to the direct cardiotoxic effects of these enzymes [11].

Interestingly, our patient did not have any of the prodromal symptoms: vertigo, nausea, and headache, which usually occur 2 weeks before the onset of stroke [12]. We assume this to be due the consequences of the toxic venom from the bite, as the patient was in a more vulnerable state to develop an acute stroke without prodromal symptoms. Also, the long admission stay associated with infrequent movement of the patient might have had a role in the development of the acute stroke [13]. In the second case, our 85-year patient was brought to the emergency room suffering from locked in syndrome. Locked-in syndrome is a distinctly rare condition characterized by damage in the brainstem, hence patients present with: quadriplegia, bulbar palsy as in anarthria and dysphagia and difficulties in breathing, yet patients are usually awake. Main causes include haemorrhagic and ischemic strokes [14]. Locked-in syndrome is usually associated with mid-basilar occlusion, and infrequently with top of the basilar artery infarction which may cause locked-in syndrome due to infarction of the bilateral cerebral peduncles [15, 16]. Hypertension was present in almost 40% of the locked in syndrome cases in a study done by Patterson and Brabois (1986) [17]. Rarely, patients can respond fully to thrombolytic therapy, but unfortunately this wasn't in our case [18]. Yet we were able to manage the patient with i.v antibiotics renal dose, dual antiplatelet therapy, enoxaparin 4,000 i.u O.D, blood transfusion and frequent session of haemodialysis in hope to stabilize the patient, as this syndrome has a very poor prognosis. The patient remained static which unfortunately is not a favourable outcome. Urgent therapy is necessary for a better prognosis, and has been related to good prognostic factors such as recovered consciousness, following commands and purposeful movement of limbs after therapy, which are associated with a favourable outcome in the long term [15].

Declarations

Both Written and verbal consents for publication were obtained from patients. The Authors declare no conflict of interest

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Figures

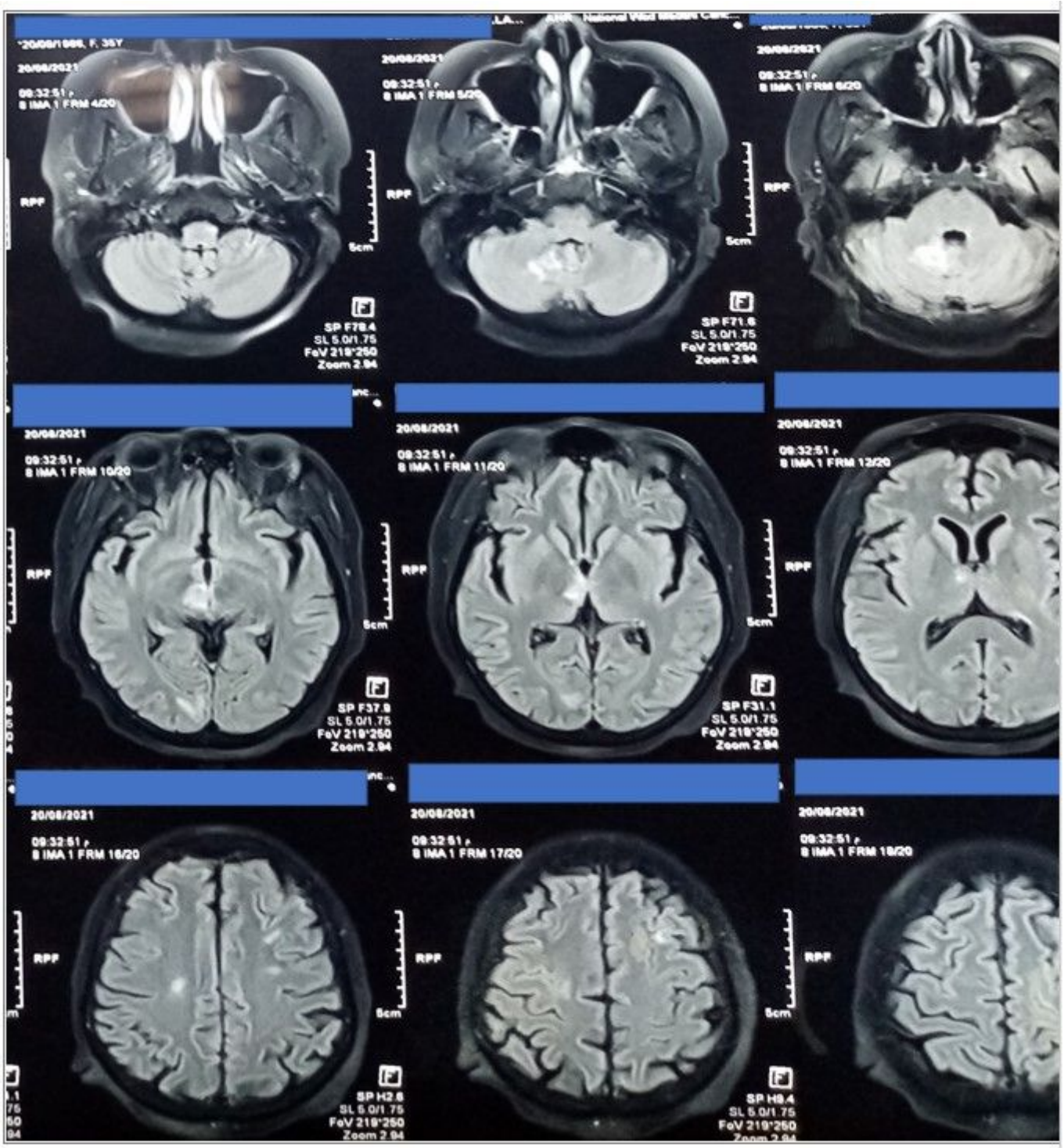

Figure 1

Case (1):-

Picture (1) Brain MRI shows Top of Basilar Artery Infarction

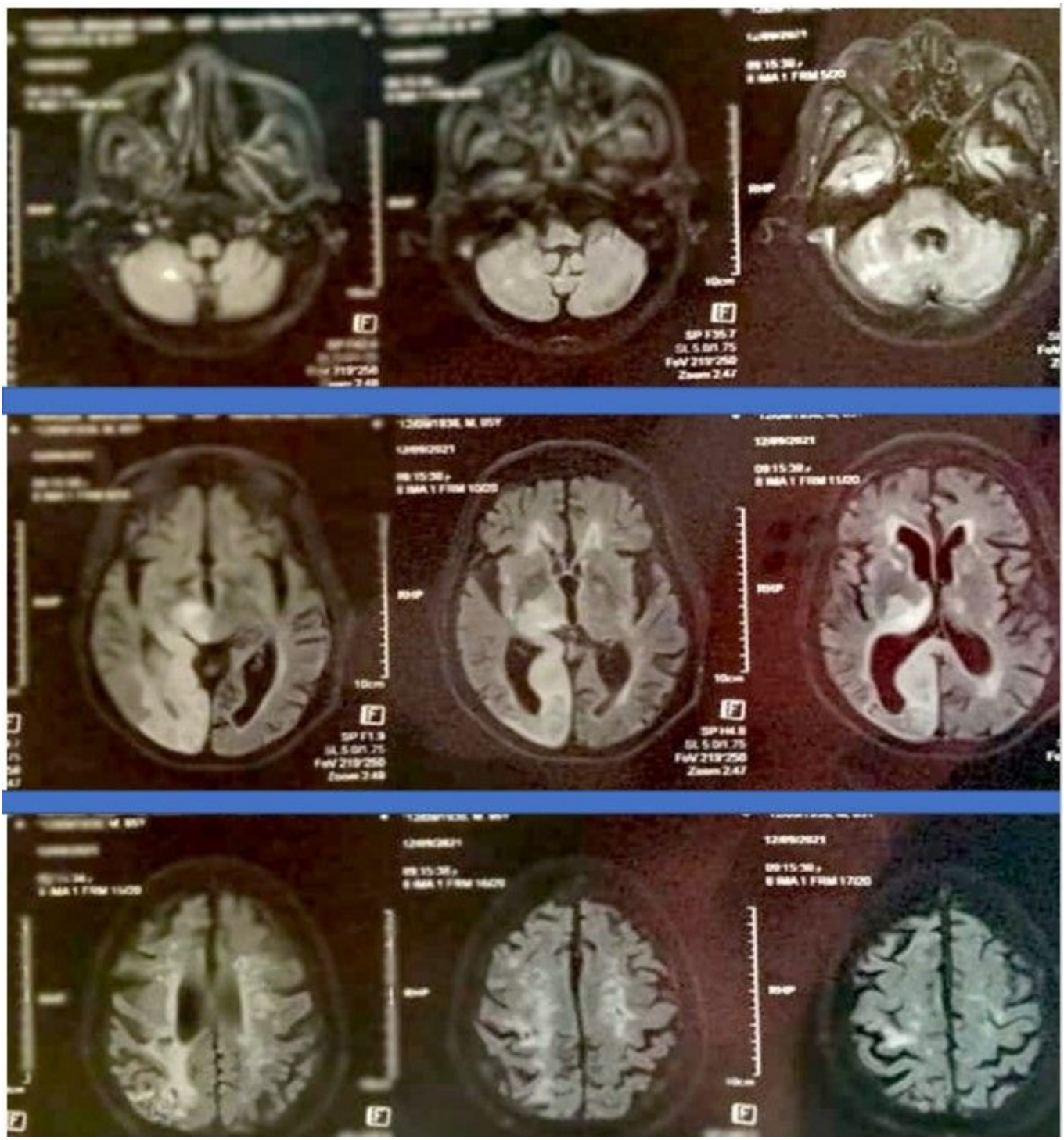

Figure 2

Case (2):-

Picture (2)Brain MRI and MRA shows Top of Basilar Artery Infarction

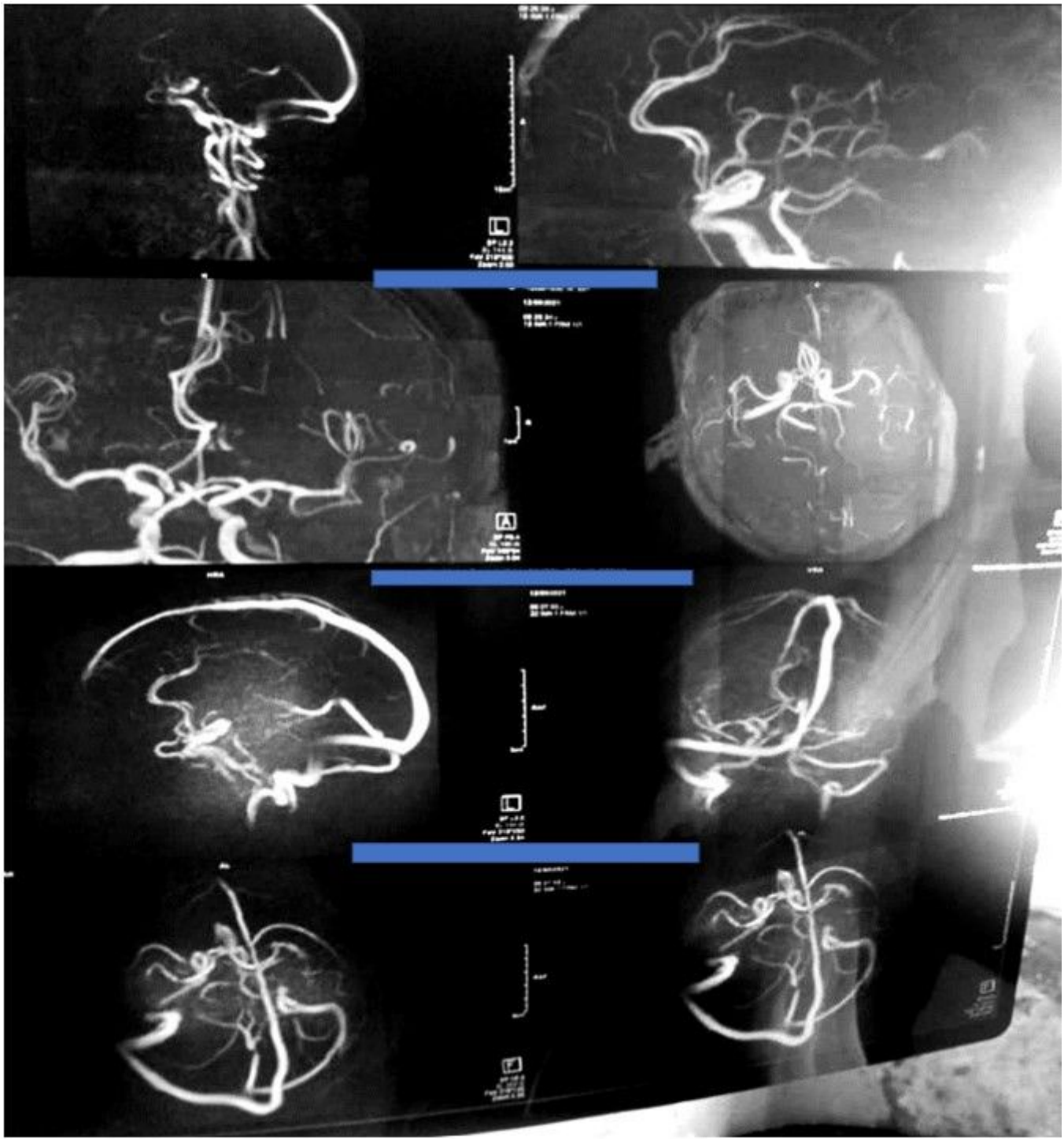

Figure 3

Case (2):-

Picture (3)Brain MRI and MRA shows Top of Basilar Artery Infarction