American Journal of Medical Case Reports, 2017, Vol. 5, No. 4, 94-100
Available online at http://pubs.sciepub.com/ajmcr/5/4/5

DOI:10.12691/ajmcr-5-4-5

Complex Aortic Plaques; an Emerging Source of Life Threatening Cardioembolic Ischemic Infarction

Noman Ahmed Jang Khan*, Saad Ullah, Joshan Suri

Department of Internal Medicine, Temple University/Conemaugh Memorial Hospital, Johnstown, PA, USA
*Corresponding author: nkhan@conemaugh.org

Abstract Cardioembolic ischemic infarction, the most severe subtype of ischemic strokes account for approximately 15-30 % of all ischemic strokes. The most common associated disorders include atrial fibrillation, recent myocardial infarction, dilated cardiomyopathy, mechanical prosthetic valve, and mitral rheumatic stenosis. Complex calcified atheromatosis of the aorta, defined as calcified plaques measuring > 4mm in size, ulcerated or having a mobile component are potentially emerging sources of cardioembolic infarction. We present a case of severe cardioembolism with evidence of complicated calcified aortic plaques in an otherwise healthy78 year old female.

Keywords: *complex aortic plaques, embolic infarcts, ischemic infarction*

Cite This Article: Noman Ahmed Jang Khan, Saad Ullah, and Joshan Suri, "Complex Aortic Plaques; an Emerging Source of Life Threatening Cardioembolic Ischemic Infarction." *American Journal of Medical Case Reports*, vol. 5, no. 4 (2017): 94-100. doi: 10.12691/ajmcr-5-4-5.

1. Introduction

Complex aortic plaques defined as calcified plaques greater than 4 mm in thickness are considered an independent risk factor for life threatening embolic ischemic infarction. Several important risk factors of cardioembolic ischemic ischemic strokes have been discussed. No specific treatment has been identified so far with anti-platelets and anti-coagulants carrying very limited role.

2. Case History

A 78 year old Caucasian female with past medical history of hypertension was brought by the EMS to the emergency department with altered mental status and seizures. She was immediately intubated, put on mechanical ventilator and transferred to intensive care unit. She could not follow any verbal commands and was only able to withdraw all extremeties to pain stimuli with no apparent focal defecits. She had positive gag, cough and corneal reflexes and pupils were equally rective to light. No visible head trauma seen but CT head revealed a small 6mm acute subdural hematoma without any midline shift or herniation. X-ray trauma series and CT cervical spine was performed which did not show any fractures or dislocation of joints. EEG was significant for bifrontal and bitemporal periodic lateralized epileptiform discharges (PLEDs) and IV leviteracitam was initiated.Initial laboratory workup including prothrombin time/International normalized ratio (PT/INR) and activated partial thromboplastin time (APTT) were within normal limits. Cerebrospinal fluid cell count showed RBC of 232, WBC count of 4, glucose of 83 and protein of 99 and no growth on the cultures [Figure 8 and Figure 9]. MRI brain was then performed which revealed more than 20 small embolic strokes in the regions of basal ganglia, cerebral hemisphere and posterior pons (Figure 1, Figure 2, Figure 3). MRA of brain did not show any hemodynamically significant stenosis, aneurismal dilatation or dissection [Figure 5, Figure 6 and Figure 7]. Carotid Doppler showed mild stenosis of bilateral carotids in the range of 1 to 40 %. Trans esophageal echocardiogram was performed which showed ejection fraction of 60 % with normal chamber sizes including left atrium (2.7 cm). Pulmonary artery pressure was within normal limits. There was no evidence of patent foramen ovale or atrial septal defect on color flow Doppler and no evidence of inter atrial shunt was found on saline contrast injection. Multiple complex calcified plaques (measuring > 4 mm) were found in the ascending aorta and aortic arch. No evidence of any intra cardiac thrombus, vegetations or masses were appreciated (Figure 4). She did not have any previous cardiac history, hypercoagulable state, active malignancy or dysrhythmias including atrial fibrillation and remained in sinus rhythm throughout her hospital stay. The patient did not show any clinical and neurological improvement throughout the hospital stay and was transferred to palliative medicine care as per family decision

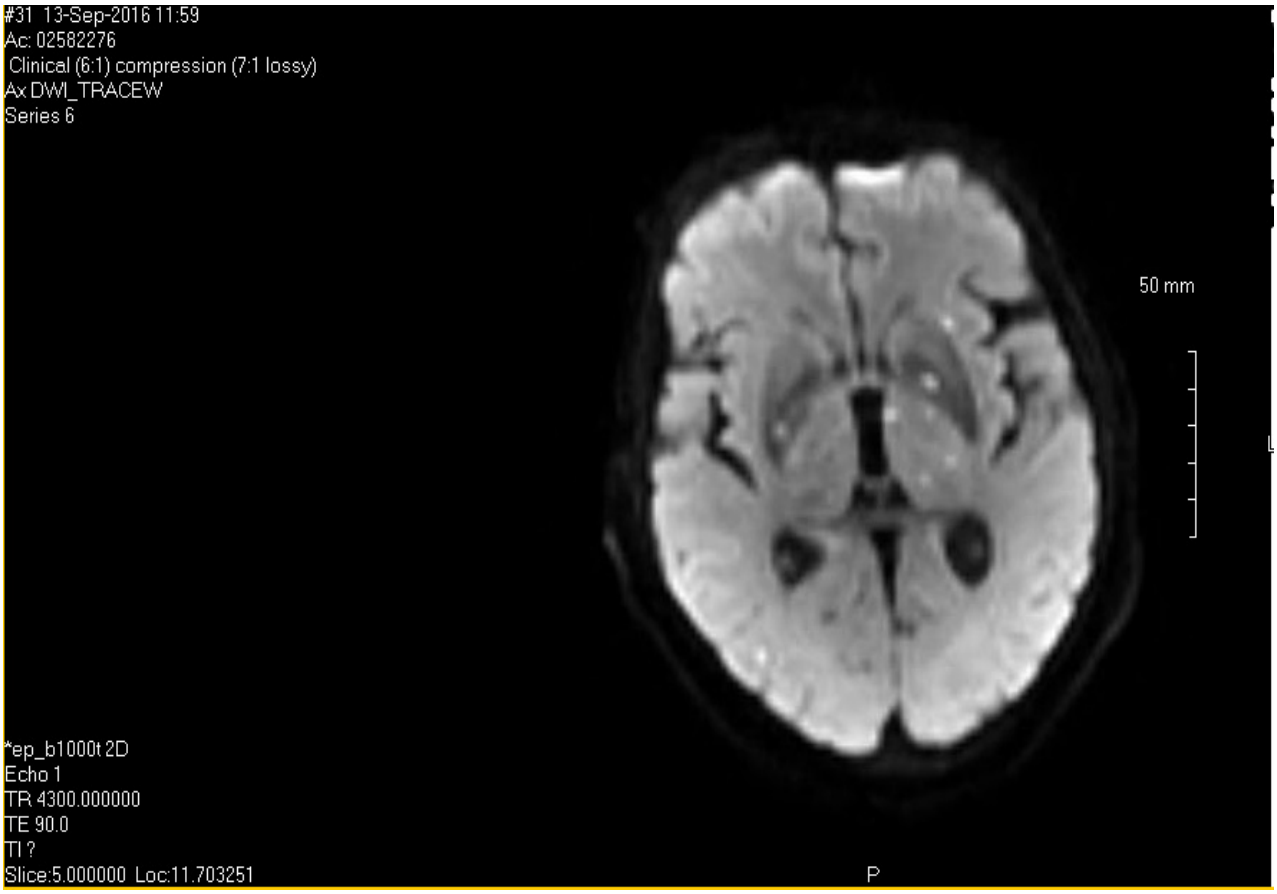

Figure 1. Axial view of MRI brain showing multiple small embolic infarcts (DWI)

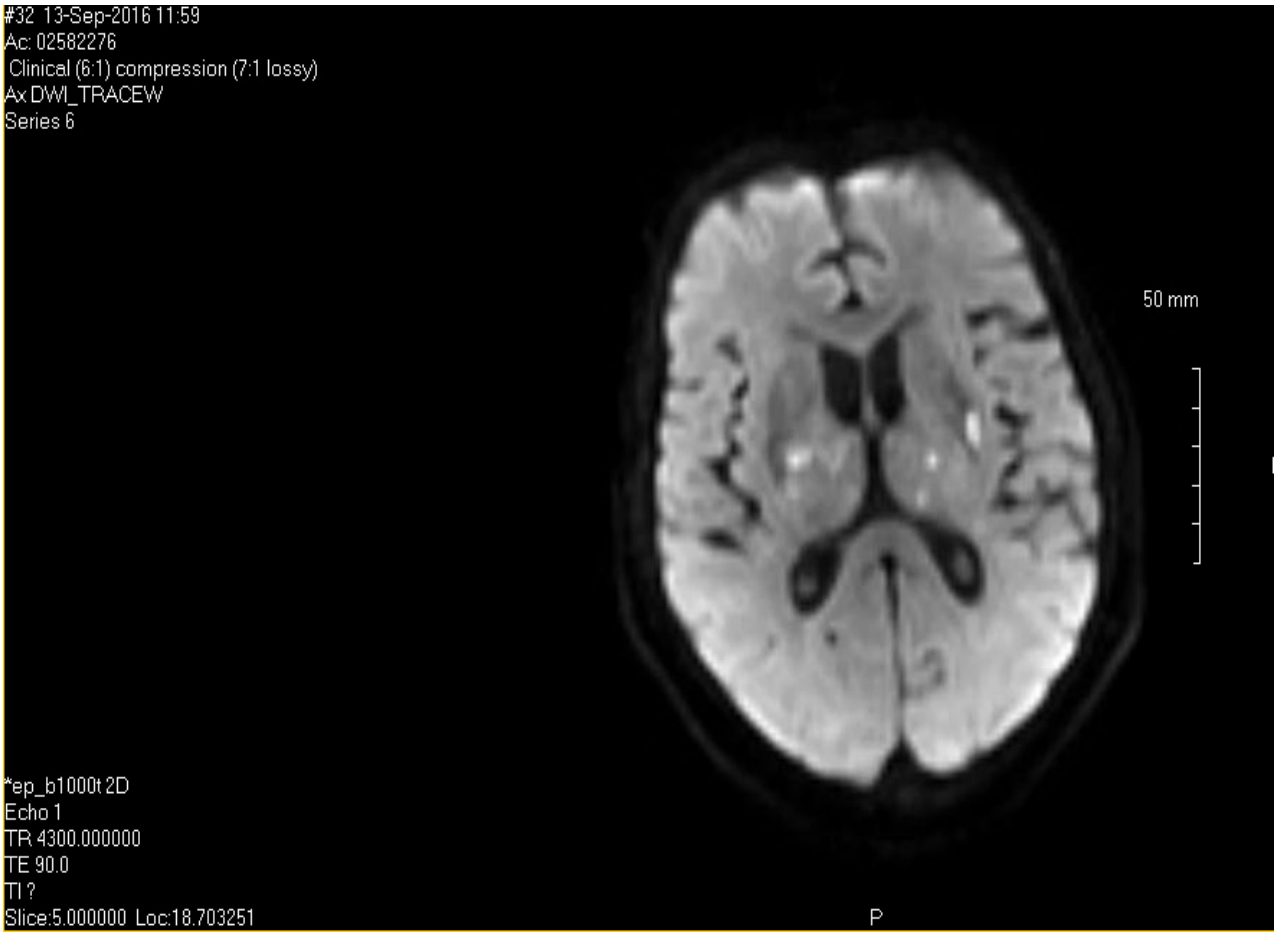

Figure 2. Axial view of MRI brain showing multiple small embolic infarcts (DWI)

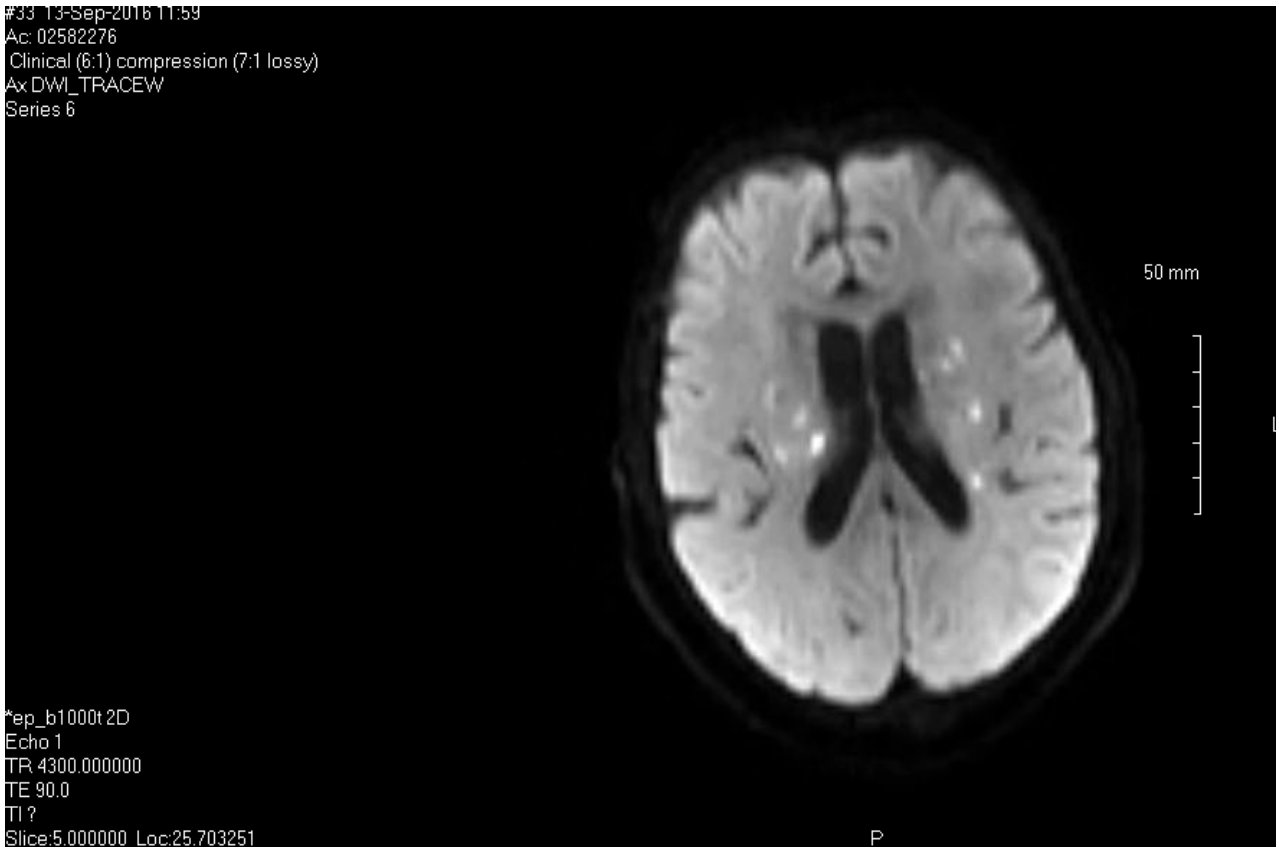

Figure 3. Axial view of MRI brain showing multiple small embolic infarcts (DWI)

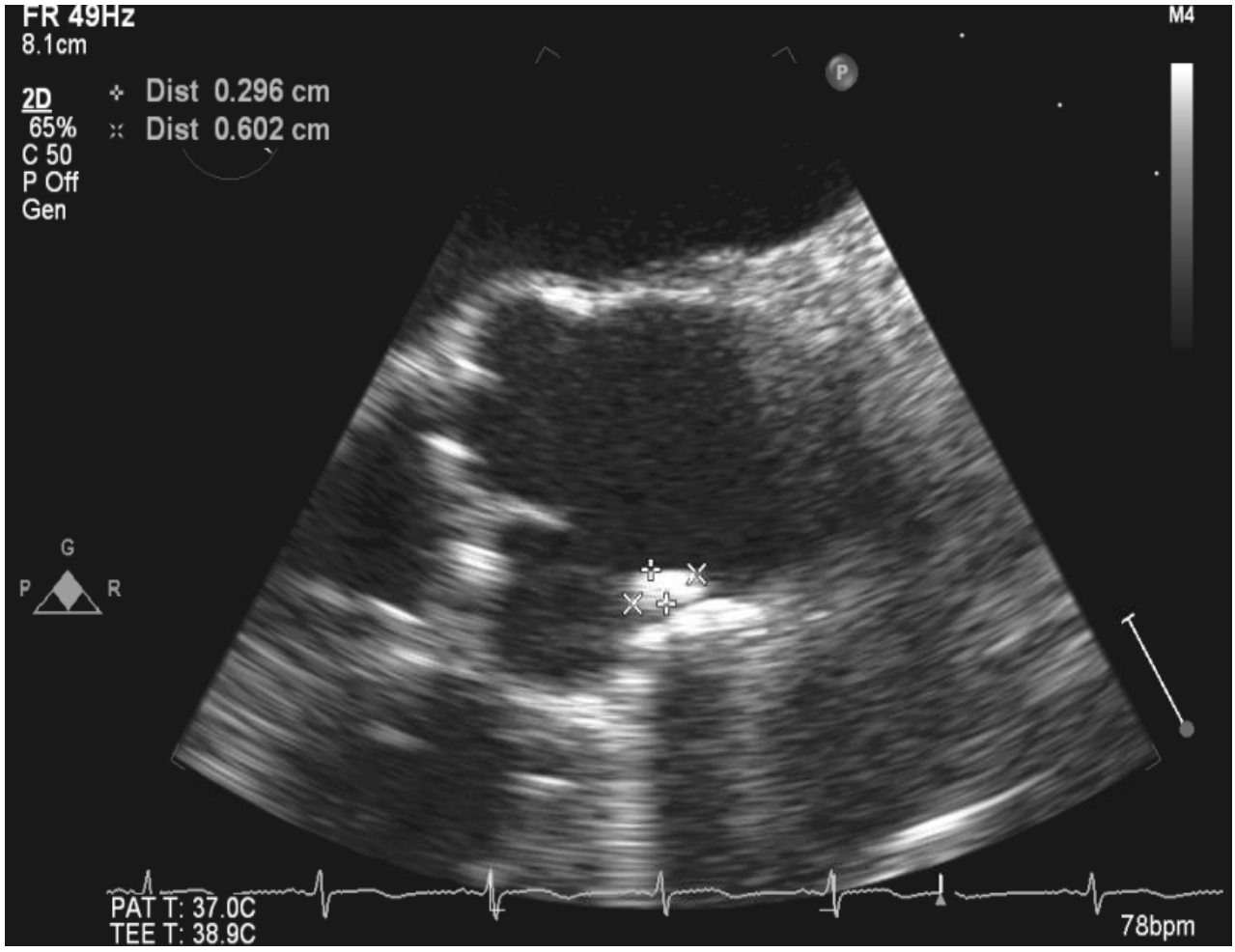

Figure 4. Trans esophageal echocardiogram showing complex aortic plaques measuring 6.02 mm.

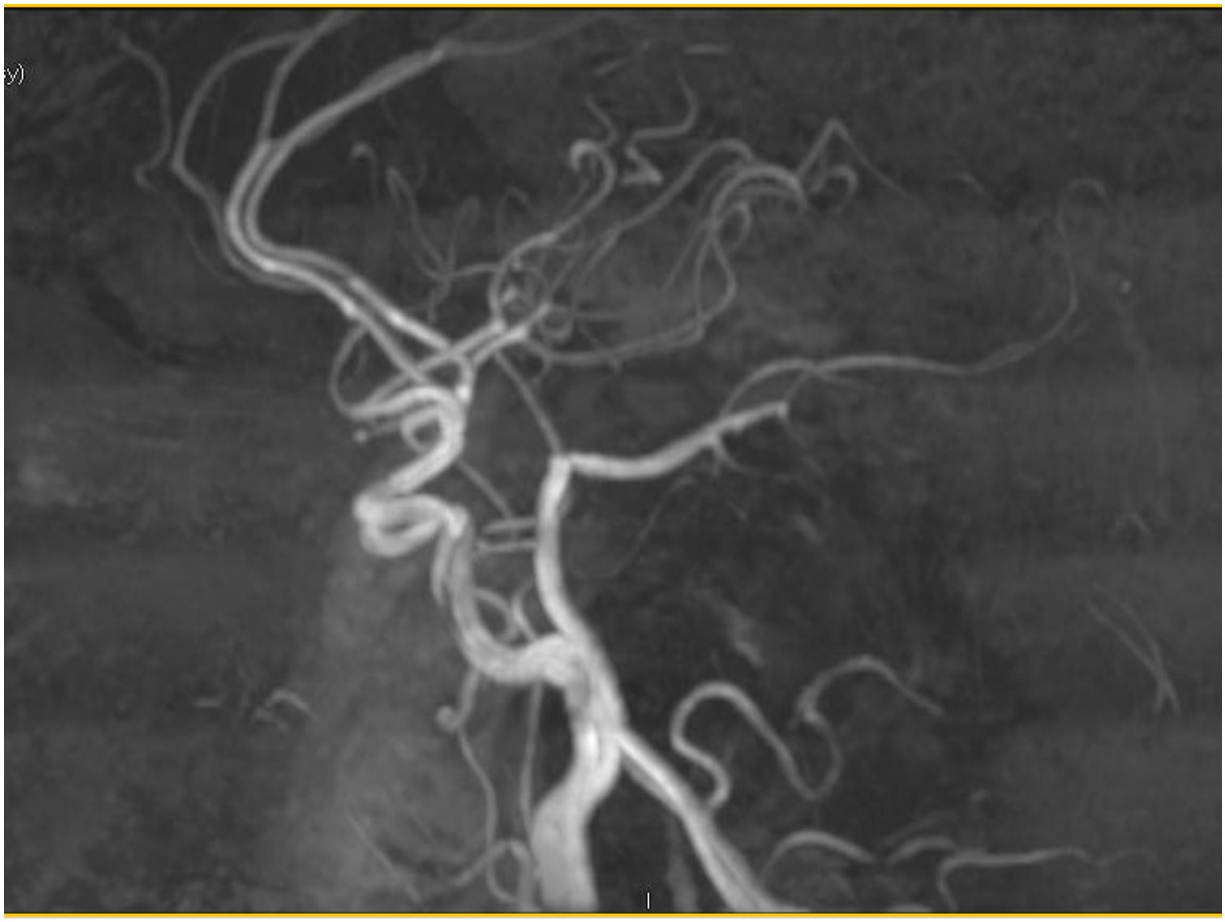

Figure 5. MRA brain, sagital view

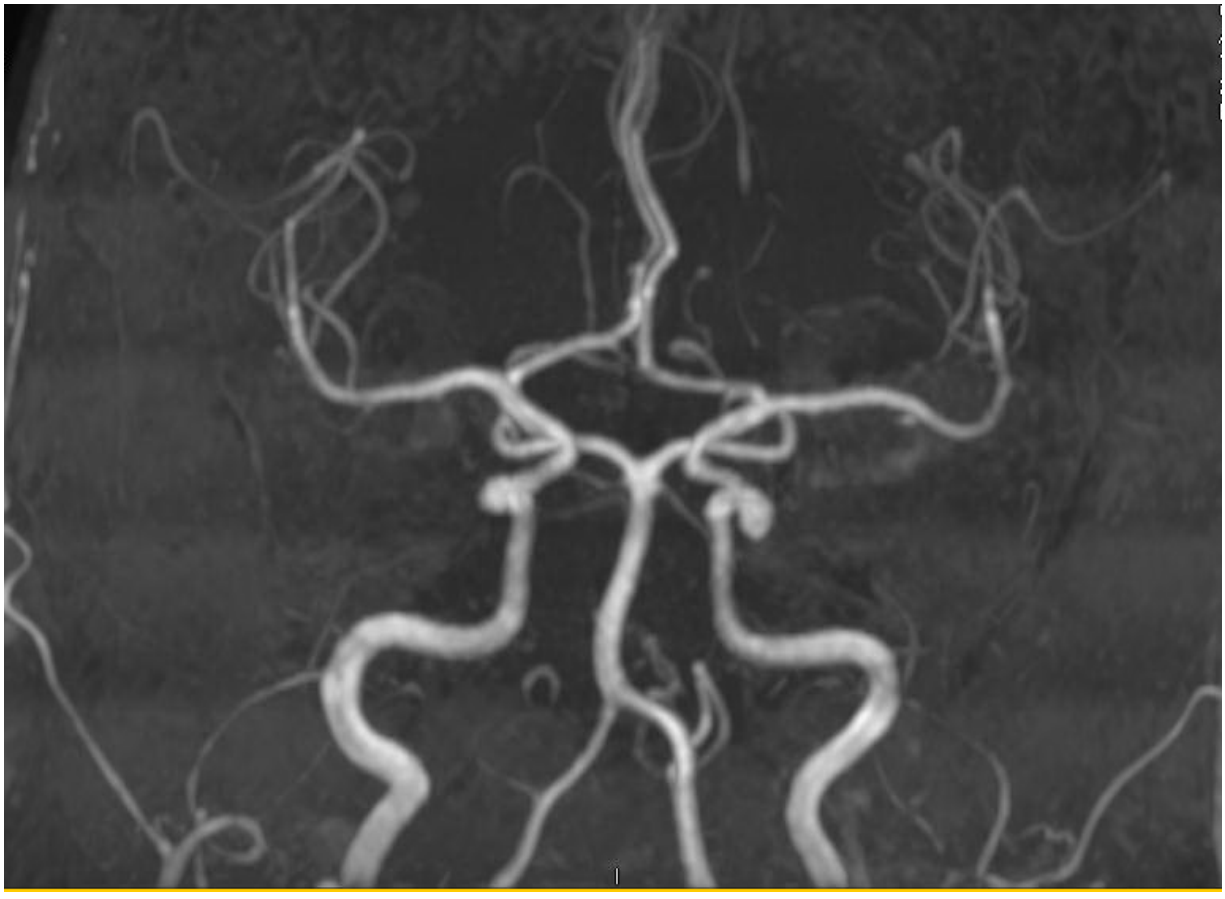

Figure 6. MRA brain, coronal view

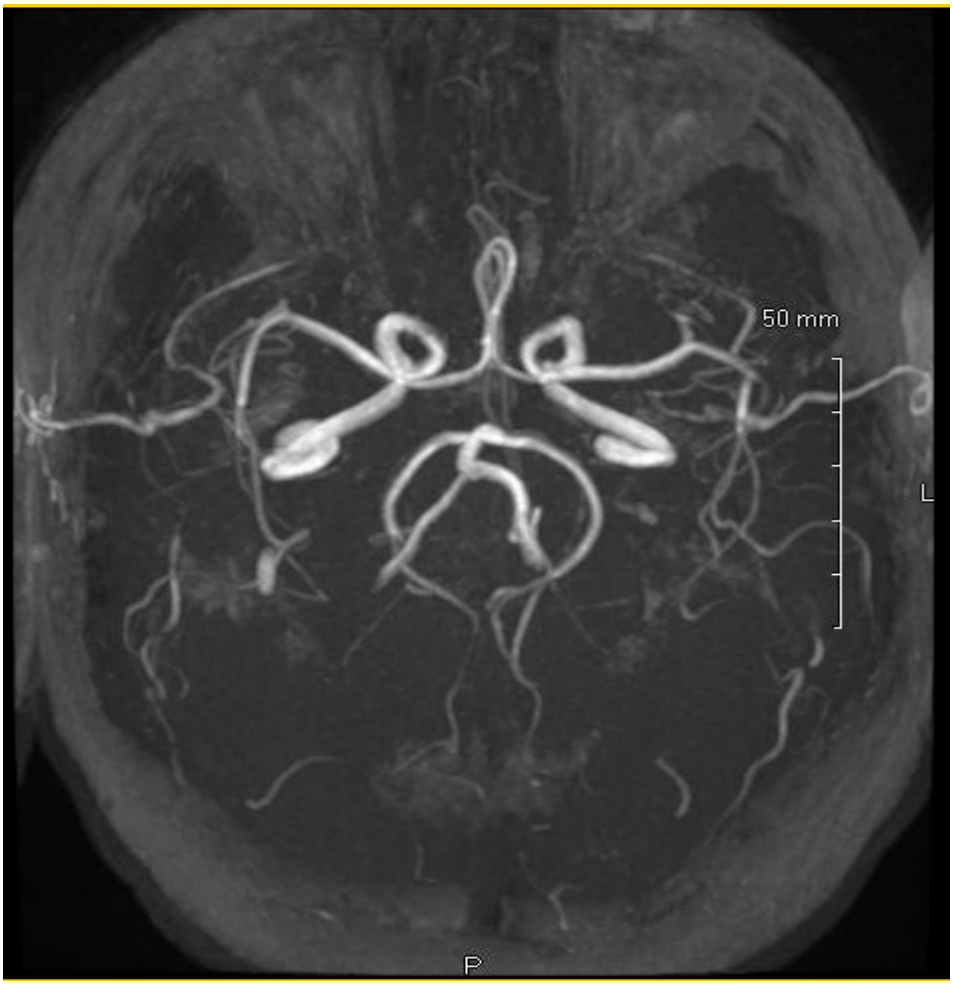

Figure 7. MRA brain, transverse view

Appearance, Fluid Comments: TUBE ONE		CLEAR COLORLESS
RBC, Fluid Comments: TUBE ONE	(0) /CUMM	232 (H)
WBC, Fluid Comments: TUBE ONE	(0-5) /CUMM	4
Neutrophil Count, Fluid Comments: TUBE ONE	(0-7) %	23 (H)
Lymphs Comments: TUBE ONE	(28-96) %	58
Other Cells, Fluid Comments: 19% MONOCYTES TUBE ONE	%	19
Glucose, CSF	(40-70) MG/DL	83 (H)
Protein, CSF	(15-45) mg/dl	99 (H)

Figure 8. CSF fluid studies

Specimen	CEREBROSPINAL FLUID (CSF)
Additional Information:	NONE NOTED
Gram Stain Result	WHITE BLOOD CELLS
Comments: NO BACTERIA SEEN ON SMEAR.	
Result	NO GROWTH AFTER 3 DAYS
REPORT STATUS	FINAL 09/20/2016

Figure 9. CSF fluid studies

3. Discussion

Stroke is among the leading causes of morbidity and mortality worldwide. Approximaltely 85 % of strokes are ischemic in nature. Cardioembolic ischemic strokes, the most severe subtype of ischemic infarction accounts for 15 to 30 % of all acute multiple infarcts involving the anterior circulation [1]. Bogousslavsky et al [2] identified several important cardiovascular risk factors including hypertension (50%), cigarette smokering (38%) hypercholesterolemia (23%); diabetes (23%), chronic atrial fibrillation (25%), left ventricular akinesia (8%), septal hypokinesia (5%), patent foramen ovale (5%), previous transient ischemic attacks in the anterior circulation (30%). Interestingly bilateral infarcts as in our patient are mostly related to cardioembolism. Complex calcified aortic plaques are defined as plaques > 4 mm in size, ulcerated or with a mobile component. These complex plaques of the ascending aorta and arch are potentially emerging source of cardioembolic ischemic infarction. All the above mentioned risk factors were ruled out in our patient leaving behind her complex aortic plaques the most probable reason for her embolic infarcts

The prevalence of aortic atheromas in general population was assessed in different studies. As per Stroke Prevention Assessment of Risk in the Community (SPARC) study [3], the prevelance of aortic atheromas was 51.3 % with 7.3 % of all the plaques were characterized as complex in nature. The association of aortic plaques with embolic strokes has been studied extensively too. Amarenco et al. [4] reviewed 500 autopsies and found ulcerated plaques in 26% of patients who died from stroke but in only 5% of patients who died from some other neurological diseases ($P < 0.001$). Moreover such plaques were seen in 61% patients who died from cerebral infarction of unknown etiology as compared to only 22% patients with a known cause of ischemic stroke ($P <0.001$).

Not all aortic plaques are notorious for causing embolism. Certain factors including plaque thickness, morphology, ulceration and mobility increases the risk of embolism. The renowned French Study of Aortic Plaque in Stroke (FSAPS) Group [5], followed 331 consecutive stroke patients aged ≥ 60 years for 2 to 4 years. At follow-up, the incidence of stroke in aortic plaques > 4 mm thickness was found to be 11.9 per 100 patient-years as compared with 3.5 per 100 patient-years in patients with plaques between 1 and 3.9 mm, and 2.8 per 100 patient-years in those with plaques < 1 mm ($P <0.001$). In another study Ferrari et al. [6] found that patients with plaques 1–3.9 mm thick had an incidence of stroke, peripheral embolism, or death of 8.8% compared with 24.0% in patients with plaques ≥ 4 mm thick and 39.2% in those with mobile components irrespective of plaque thickness ($P = 0.007$).

Magnetic resonance imaging (MRI) and Computed Tomography (CT) scanning have have been implicated in the diagnosis of aortic plaques and their characteristics but cost effectiveness and limited access to CT and MRI makes trans esophageal echocardiogram (TEE) the gold standard modality of choice in the diagnosis of atherosclerotic plaques of the aortic arch as well as in the visualization of their morphological characteristics [7,8]. Trans thoracic echocardiogram (TTE) in some cases can be a useful initial screening imaging modality. In our patient TEE showed complex aortic plaques in the ascending aorta and aortic arch [Figure 4] however TTE done initially was unremarkable. Diffusion weighted (DW) magnetic resonance imaging (MRI) have better capability to detect early ischemic brain events as compared to the conventional T1-WI and T2-WI and are also able to differentiate between new and old infarcts [9].

Appropriate treatment is uncertain. Smoking cessation, treatment of hypercholesterolemia with lipid lowering drugs and blood pressure control play important role in the prevention of such lesions. Studies have shown role of anticoagulation in lowering the risk of recurrent srokes in patients with plaques with mobile component [10]. Antiplatelet therapy with aspirin has shown some risk reduction of vascular events from 22 % to 18 % [11] with some studies showing small additional benefit of dual antiplatelet therapy with aspirin and clopidogrel [12]. In some patients with recurrent cardioembolic ischemic strokes and pedunculated or mobile plaques in the aortic arch, surgical removal [13] and thrombolysis could become a potential treatment option.

4. Conclusion

In the light of above studies, the potential increase in the risk of ischemic stroke in patients with plaques ≥ 4 mm in thickness in the ascending aorta and aortic arch emphasize that such lesions should always be considered as an independant risk factor for ischemic stroke, particularly in the cases where no other likely or possible cause could be identified. Treatment is mainly conservative. Antiplatelets and anticoagulation have shown some benefit in select group of patients in non randomized studies however prospective randomized studies should be done in order to identify the efficacy of different treatment options.

Conflicts of Interests

There are no conflicts of interest for this article.

References

[1] Arboix A, Alioc J. Cardioembolic stroke: clinical features, specific cardiac disorders and prognosis. Current cardiology reviews. 2010 Aug 1; 6(3): 150-61.

[2] Bogousslavsky J, Bernasconi A, Kumral E. Acute multiple infarction involving the anterior circulation. Archives of neurology. 1996 Jan 1; 53(1): 50-7.

[3] Agmon Y, Khandheria BK, Meissner I, et al. Relation of coronary artery disease and cerebrovascular disease with atherosclerosis of the thoracic aorta in the general population. Am J Cardiol. 2002; 89: 262-7.

[4] Amarenco P, Duyckaerts C, Tzourio C, Henin D, Bousser MG, Hauw JJ. The prevalence of ulcerated plaques in the aortic arch in patients with stroke. N Engl J Med. 1992; 326: 21-5.

[5] The French Study of Aortic Plaques in Stroke Group. Atherosclerotic disease of the aortic arch as a risk factor for recurrent ischemic stroke. N Engl J Med. 1996; 334: 1216-21.

[6] Ferrari E, Vidal R, Chevallier T, Baudouy M. Atherosclerosis of the thoracic aorta and aortic debris as a marker of poor prognosis: benefit of oral anticoagulants. J Am Coll Cardiol. 1999; 33: 1317-22

[7] Katsanos AH, Patsouras D, Tsivgoulis G, Kosmidou M, Katsanos KH, Kyritsis AP, Giannopoulos S. The value of transesophageal echocardiography in the investigation and management of cryptogenic cerebral ischemia: a single-center experience. Neurological Sciences. 2016 Apr 1; 37(4): 629-32.

[8] Zaidat OO, Suarez JI, Hedrick D, et al. Reproducibility of transesophageal echocardiography in evaluating aortic atheroma in stroke patients. Echocardiography. 2005; 22: 326-30.

[9] Altieri M, Metz RJ, Müller C, Maeder P, Meuli R, Bogousslavsky J. Multiple brain infarcts: clinical and neuroimaging patterns using diffusion-weighted magnetic resonance. European neurology. 1999 Aug 26; 42(2): 76-82.

[10] Tenenbaum A, Fisman EZ, Schneiderman J, et al. Disrupted mobile aortic plaques are a major risk factor for systemic embolism in the elderly. Cardiology. 1998; 89: 246-51.

[11] Antiplatelet trialists' collaboration. Collaborative overview of randomized trials of antiplatelet therapy. Prevention of death, myocardial infarction, and stroke by prolonged antiplatelet therapy in various categories of patients. BMJ. 1994; 308: 81-106.

[12] Bhatt DL, Fox KAA, Hacke W, et al. Clopidogrel and aspirin versus aspirin alone for the prevention of atherothrombotic events. N Engl J Med. 2006; 354: 1706-17.

[13] Hausmann D, Gulba D, Bargheer K, Niedermeyer J, Comess KA, Daniel WG. Successful thrombolysis of an aortic-arch thrombus in a patient after mesenteric embolism. N Engl J Med 1992; 327: 500-501.