JOURNAL OF CARDIOVASCULAR, NEUROVASCULAR & STROKE

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Volume 7 **Number 1** **March 2025**

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Case Report

A LARGE PARADIGM SHIFT LEADS TO A SMALL KEYHOLE PUNCTURE: A CASE STUDY OF THORACIC ENDOVASCULAR AORTIC REPAIR (TEVAR) FOR DISTAL AORTIC ARCH PSEUDOANEURYSM IN A PATIENT WITH SEVERAL SEVERE UNDERLYING MEDICAL CONDITIONS

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DOI: https://doi.org/10.32896/cvns.v7n1.1-10
Received: 07.07.2024
Revised: 03.03.2025
Accepted: 05.03.2024
Published: 31.03.2024

ABSTRACT

This case study highlights how far aortic surgical intervention has come along since the advent of open aortic repair in the 1950s to the monumental introduction of minimally invasive endovascular aortic repair in mid-1990s.

The following case depicts a successful management of distal aortic arch pseudo aneurysm with Thoracic Endovascular Aortic Repair (TEVAR) along with carotid-carotid bypass in an elderly gentleman with multiple severe underlying medical conditions - including left papillary renal cell carcinoma (RCC), toxic nodular goiter, and hypertension.

The subsequent presentation will highlight chronological management of this case across the timeline with the varying advancements of aortic repair from no intervention to open repair, and now with TEVAR.

The management of this case also highlights the importance of involving a multidisciplinary team approach to provide a holistic co-management for this patient.

Keywords: Distal aortic arch, pseudoaneurysm, aortic surgery, TEVAR, carotid-carotid bypass, open aortic repair, multidisciplinary approach, multiple co-morbidities

INTRODUCTION:

This case study presents the successful management of a distal aortic arch pseudoaneurysm with thoracic endovascular aortic repair (TEVAR) in a patient with other grave underlyings of left papillary renal cell carcinoma (RCC), toxic nodular goiter, and hypertension. Conventionally, a patient with pseudoaneurysm undergoes open repair. Open aortic repair carries with it a high risk of morbidity and mortality, which may include but is not limited to infection, stroke, massive bleeding, and death [1]. Because of this worrying number, an open aortic repair is risky, especially if the patient has other debilitating co-morbidities.

With the advent of TEVAR in the 90's, the data appears more and more hopeful for both surgeons and patients as the advancement of TEVAR makes the procedure easier and the prognosis more guarded [2]. In view of this patient's multiple severe co-morbidities, a cautious approach was considered and TEVAR was considered as a best choice for repair.

This case highlights the prompt recognition, careful management and successful resolution of the pseudoaneurysm while considering the patient's overall clinical profile. This was achieved through a multidisciplinary approach.

CASE REPORT:

This case looks at a 69-year-old gentleman, presented with symptoms of general lethargy and occasional dyspnea. His medical history was notable for left exophytic papillary RCC measuring 6x5x8cm, toxic nodular goiter with local mass effect to trachea, and hypertension. During the staging workup for RCC, a computed tomography (CT) aortogram (CTA) incidentally revealed a distal aortic arch outpouching with penetrating aortic ulcer (PAU) and no haematoma - measuring approximately 4.1x1.5cm, located 2.8cm distal to the left subclavian artery. Cardiomegaly and pulmonary arterial hypertension (PAH) were observed as secondary effects of the pseudoaneurysm (Figure 1 and 2). Considering the patient's multiple debilitating underlying medical conditions, careful consideration was given in the management approach. A multidisciplinary team (MDT) involving cardiovascular surgery, urology, and surgical endocrinology was assembled to optimize patient care. Given the perilous risks associated with open surgery, the decision was made to proceed with minimally invasive TEVAR first, and subsequently for surgical resection of the RCC. At the same time, the medical endocrine team was managing the toxic nodular goitre with Carbimazole first and subsequently planned for total thyroidectomy as there was mass effect affecting the trachea from the CT scan. From Cardiovascular surgical side, TEVAR and carotid-carotid bypass under general anesthesia to address the distal aortic arch pseudoaneurysm was decided after discussion by the surgical team and endovascular device team. TEVAR was still offered here instead of open surgery for several advantages, including minimally invasive access, reduced operative trauma, and shorter recovery time, which were all particularly beneficial considering the patient's underlying morbidities. In the same setting, a carotid-carotid artery bypass grafting was done to provide adequate circulation to the left carotid artery from the right carotid artery. TEVAR planning and assessment was done by the device team (Figure 3). During the surgery, the carotid-carotid bypass was carried out first with dacron graft tube. Subsequently, TEVAR was placed under imaging guidance. The landing zone was just distal to the inominate artery.

Following the TEVAR procedure, the patient was closely monitored in the cardiac intensive care unit (CICU) for one day to ensure postoperative stability (Figure 4 and 5). The patient exhibited a favorable recovery and was subsequently discharged well on the second day post TEVAR.

Following TEVAR, subsequent follow up in the outpatient clinic showed that the other relevant teams involved taking advantage of TEVAR by proceeding with their subsequent respective managements. The urology team proceeded with robotic left Nephrectomy 1 month after the TEVAR, and patient's toxic nodular goiter was well managed by the endocrinology team and planned for a total thyroidectomy down the road, following the successful resolution of the aortic pseudoaneurysm with TEVAR.

DISCUSSION:

This case highlights the successful application of TEVAR in a complex case of pseudoaneurysm. Distal aortic arch pseudoaneurysm, also known as false aneurysms, occurs when there is a breach in the arterial wall, resulting in the formation of a sac that is partially contained by surrounding tissues [3]. Aortic pseudoaneurysms can develop due to trauma, infection, or as a complication of previous surgical interventions. It is associated with significant morbidity and mortality up to 85% [4].

Thoracic endovascular aortic repair (TEVAR) is a minimally invasive surgical procedure for the management of an array of thoracic aortic pathologies such as aortic aneurysm, aortic dissection, penetrating ulcers, intramural haematoma and pseudoaneurysm. It involves the insertion of a stent graft through small incisions in the groin area, which is guided to the site of the pseudoaneurysm using fluoroscopy or computed tomography (CT) scan. The stent graft is expanded once in position, creating a new path for blood flow and effectively excluding the pseudoaneurysm from the circulation. The stent graft acts as a reinforcement for the weakened or damaged arterial wall, preventing further expansion and reducing the risk of rupture.

The utilization of TEVAR as a less invasive intervention allowed for the effective treatment of the distal aortic arch pseudoaneurysm. TEVAR also minimizes the morbid risks from 10-20% associated with open surgery, to 5-10% with TEVAR [5-9].

The application of TEVAR results in smaller incisions, reduced blood loss, and shorter recovery times [6]. It is generally associated with better patient outcomes. However, David et al indicated that although operative mortality was indeed halved with stent graft, late survival was similar for both cohorts (stent vs open surgery), with identical re-intervention rate and rate of spinal cord ischaemic complication, and a higher risk of stroke (6% in surgery vs 12% in stent) [9].

Hence, TEVAR may prove to be the favored choice of repair for thoracic aorta cases such as in this patient, but TEVAR still imposes certain risks and therefore more research needs to be done to explore the long-term effect of TEVAR.

A carotid-carotid bypass was done in this case to provide blood supply to the left carotid artery as the TEVAR was deployed just at the distal to the innominate artery. Although a conventional approach of carotid-carotid artery bypass and left carotid-left subclavian artery bypass was recommended by the endovascular device team, the surgeon deemed that the left subclavian artery system will be adequately perfused via the vertebral artery and hence did not proceed with the left carotid-left subclavian artery bypass in this case. Edward Y. Woo shared a similar view after a retrospective analysis was done in his center when he stated that "Zone 2 TEVAR with LSA coverage can be accomplished safely in both elective and emergency settings and with and without revascularization (with the exception of a patent LIMA-LAD bypass)" [8].

Lastly, it is also important to emphasize the marvel of multidisciplinary input in this case as each team was able to delineate their individual plan for the patient, and a collective decision was made for the best interest of this patient. All the while treating each debilitating diseases which the patient suffers, and at the same time addressing the treatment holistically treating the patient.

CONCLUSION:
In this case study, TEVAR proved to be a significant beneficial treatment modality for a distal aortic arch pseudoaneurysm in a patient with multiple severe underlying medical conditions. The minimally invasive nature of TEVAR, combined with careful multidisciplinary management, facilitated successful resolution of the pseudoaneurysm while considering the patient's complex medical profile. This case emphasizes the importance of tailoring treatment approaches to the individual patient and highlights the benefits of a collaborative and sequential management strategy.

DATA AVAILABILITY:
Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:
This work received no external funding.

CONFLICT OF INTEREST:
The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

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Conrad, Thomas K. Chung, Richard P. Cambria. Stent-graft versus open-surgical repair of the thoracic aorta: Mid-term results. Journal of Vascular Surgery, Volume 44, Issue 6. 2006

FIGURE LEGENDS:

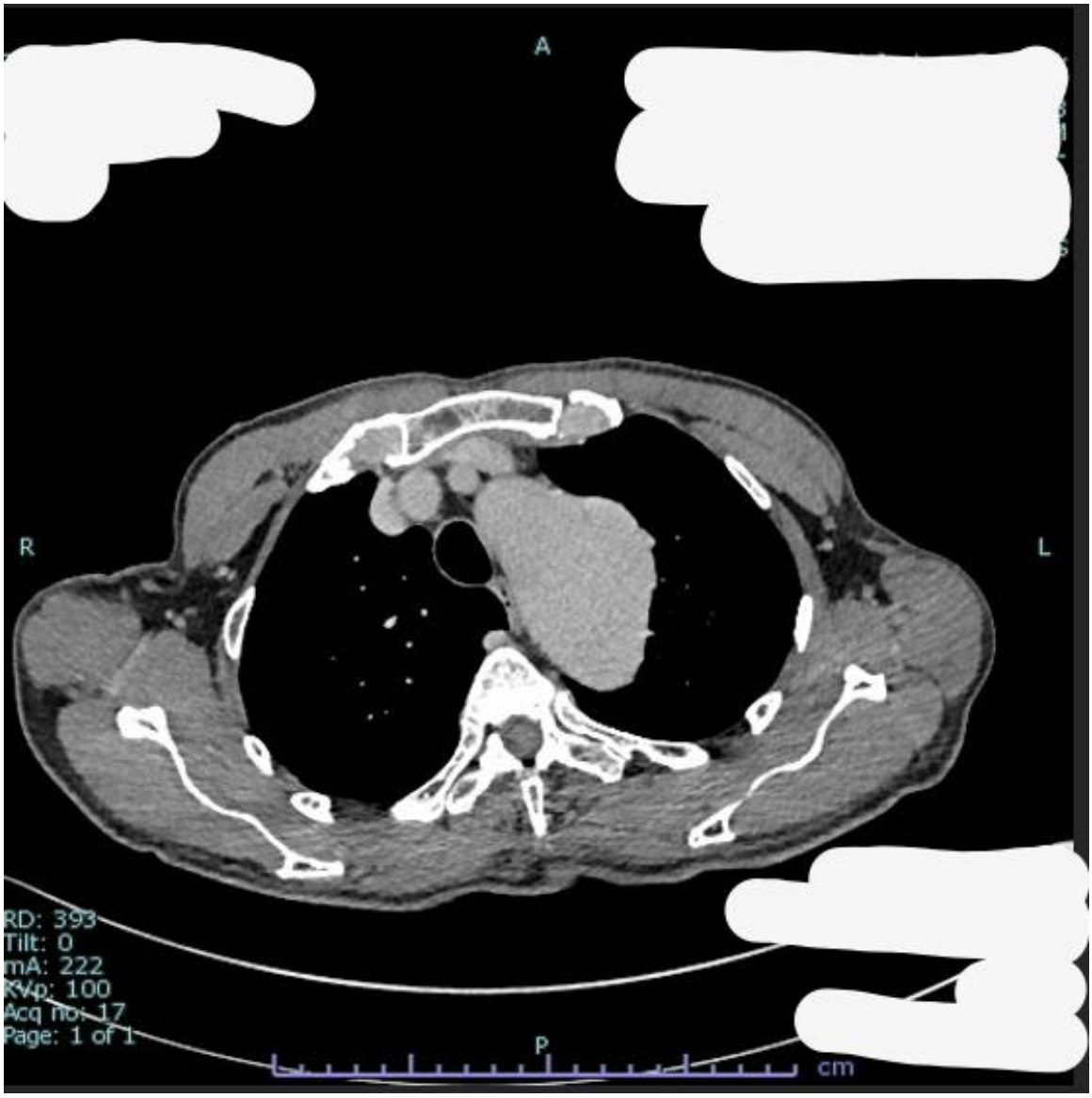

Figure 1: Axial view of CTA showing the pseudoaneurysm in the distal aortic arch.

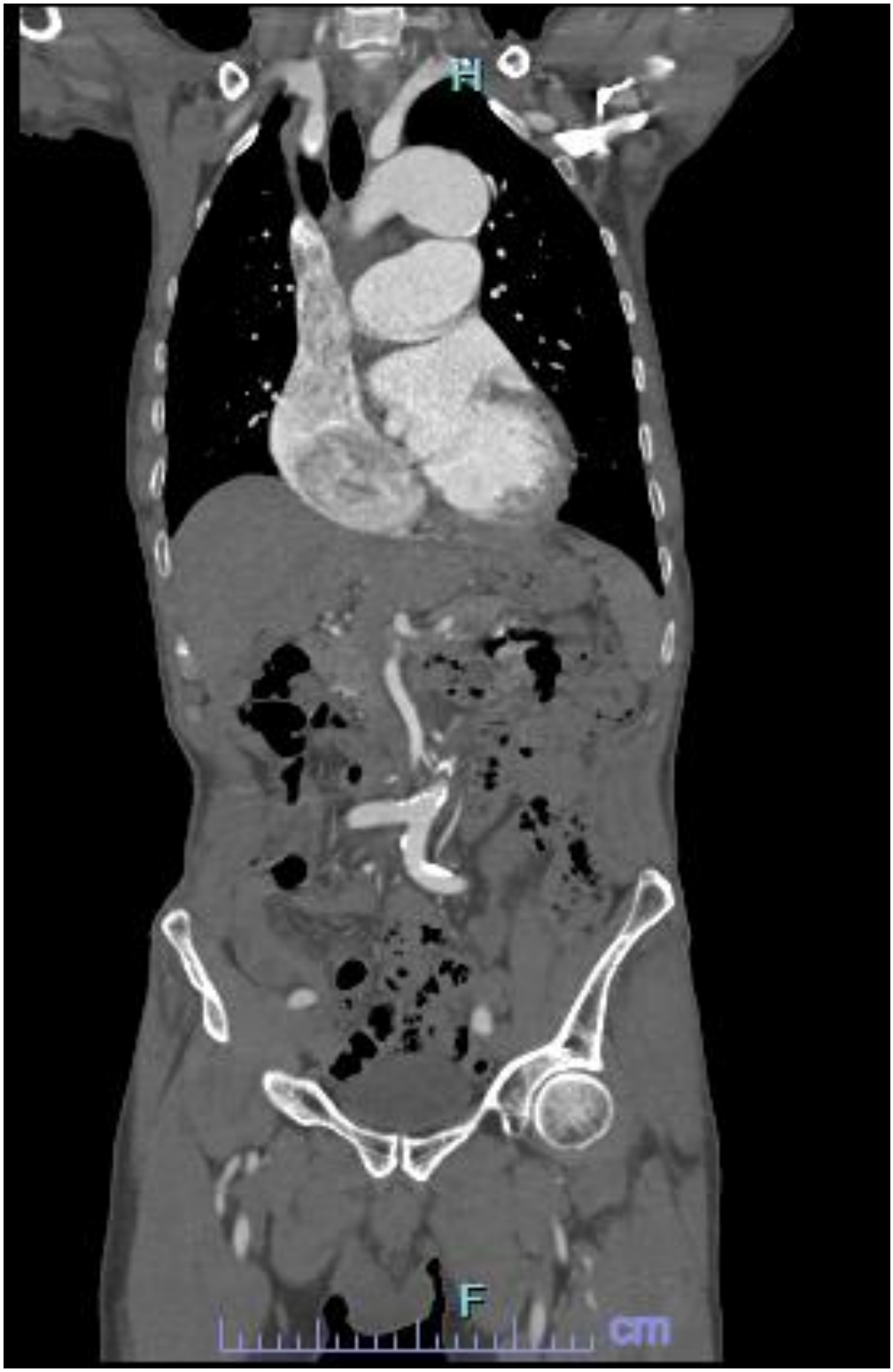

Figure 2: Coronal view of the CTA showing the pseudoaneurysm outpouching of distal aortic arch.

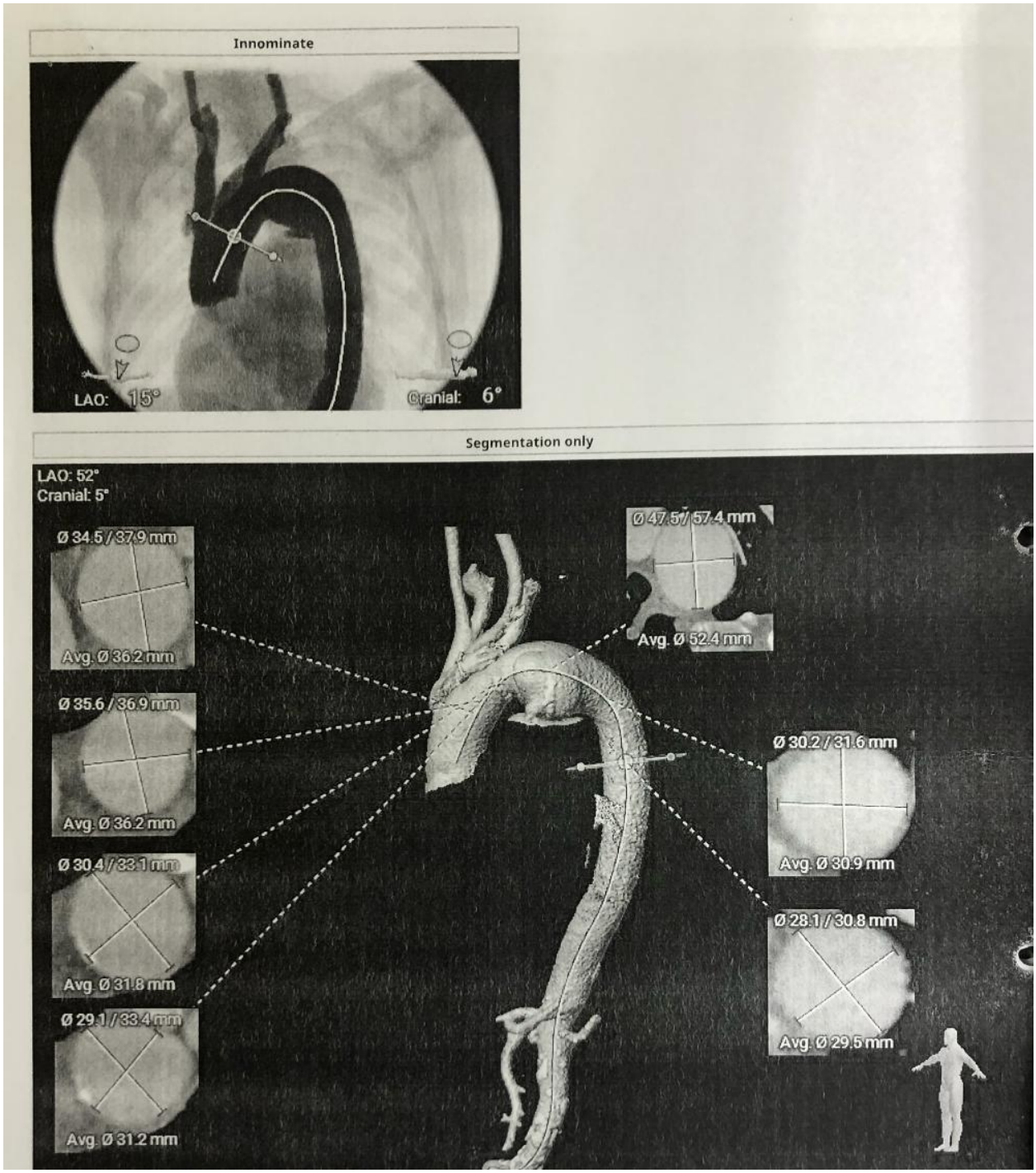

Figure 3: TEVAR Planning.

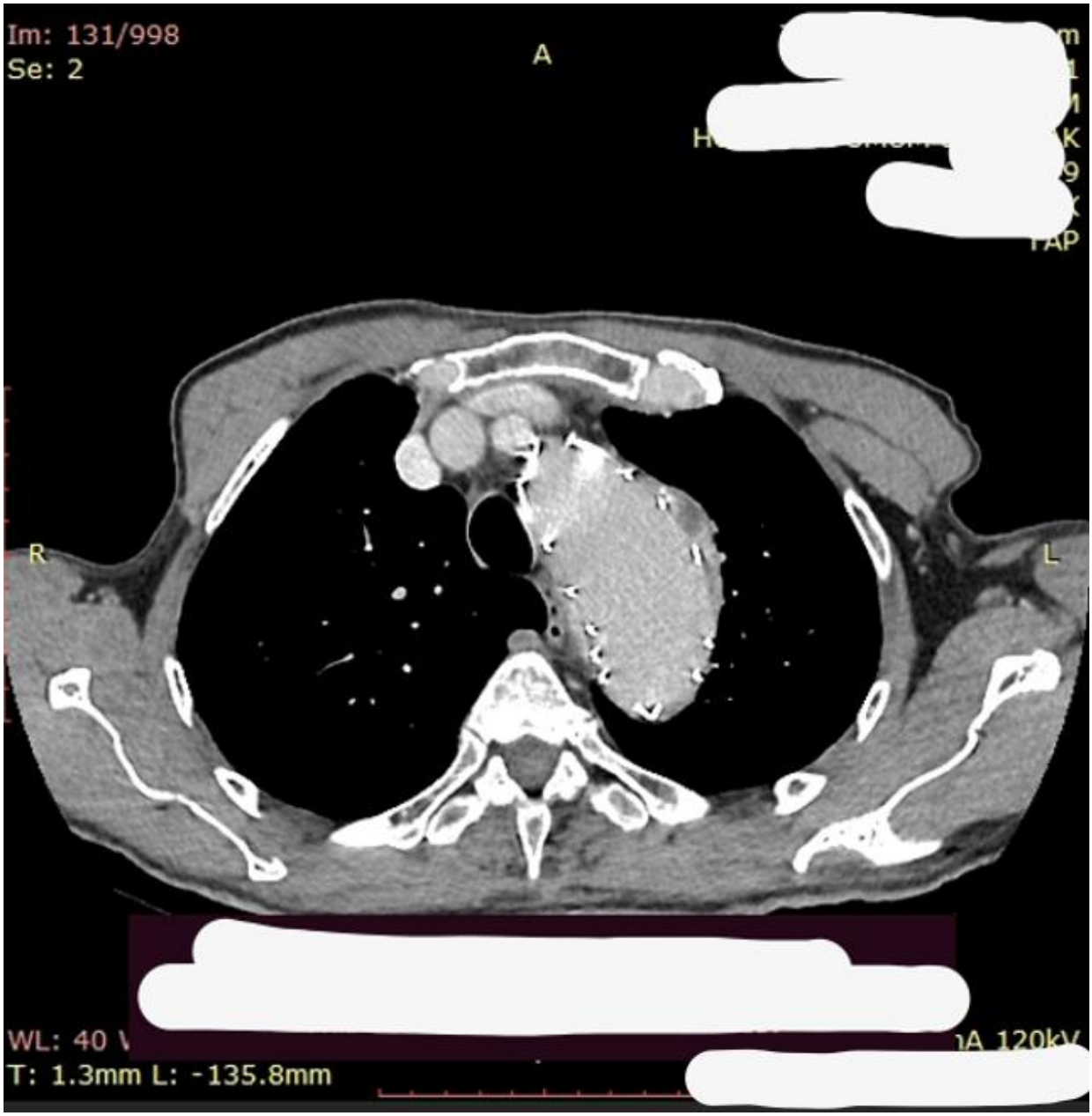

Figure 4: Axial view of CTA post TEVAR.

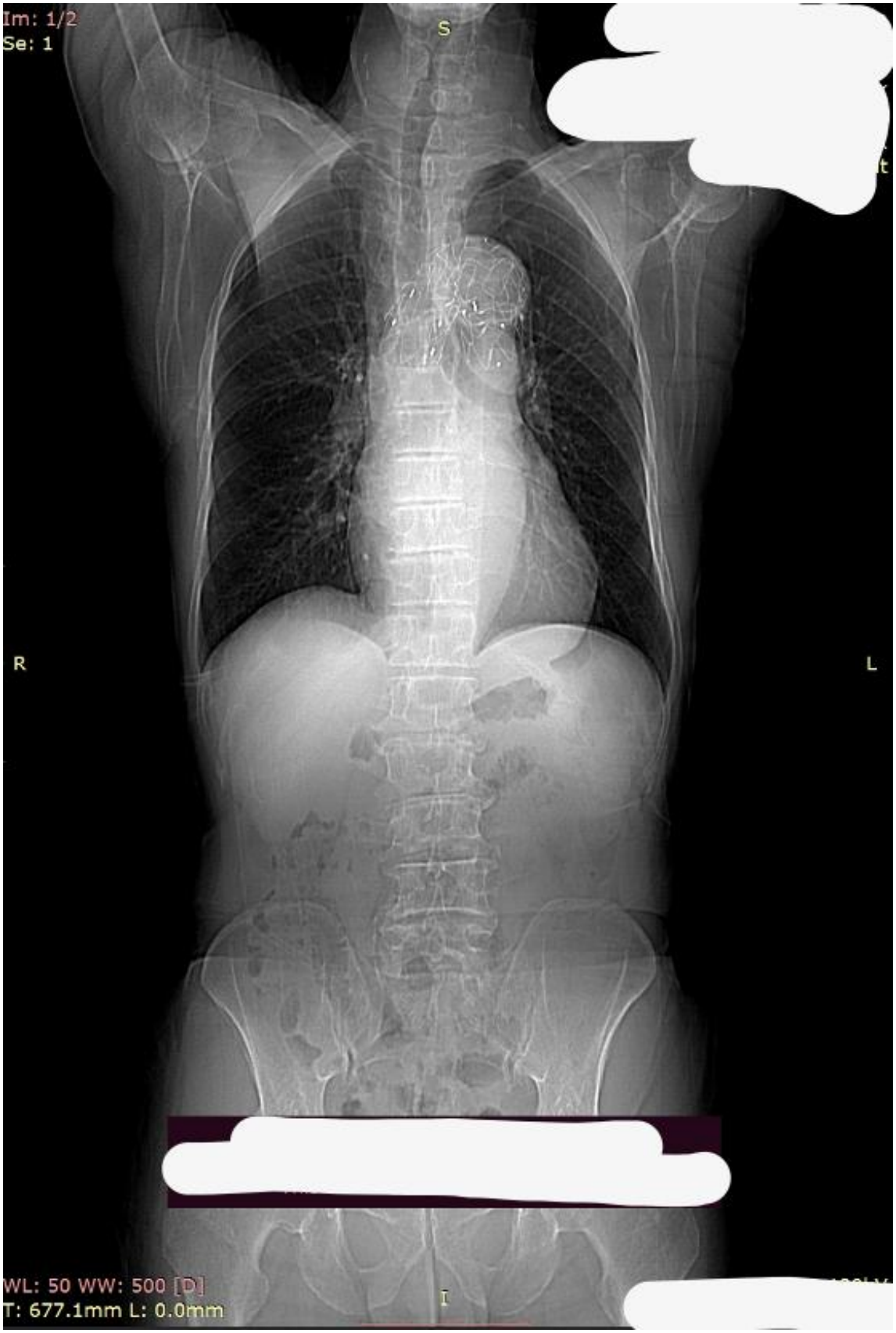

Figure 1 : Coronal view of CTA post TEVAR.

CLINICAL AND RADIOLOGICAL APPROACH TO THE DIAGNOSIS OF BRANCH ATHEROMATOUS DISEASE: CASE REPORT AND REVIEW OF LITERATURE

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DOI: https://doi.org/10.32896/cvns.v7n1.11-18
Received: 10.12.2024
Revised: 26.03.2025
Accepted: 28.03.2024
Published: 31.03.2024

ABSTRACT

A form of ischemic stroke known as branch atheromatous disease (BAD) is brought on by plaque accumulation that obstructs or stenoses the entrance of penetrating branches. Despite its therapeutic importance, this concept is still poorly understood and underutilized in clinical practice and research. BAD is strongly linked to an increased risk of disability and early neurological degeneration (END). Based on clinical status, risk factors, laboratory results, and radiographic aspects of infarct and blood vessel morphologies, BAD has several characteristics. Determining treatment options and each patient's prognosis depends on early BAD identification.

Keywords: Branch atheromatous disease, stroke, imaging

INTRODUCTION:

A subtype of ischemic stroke known as branch atheromatous disease (BAD) is caused by proximal atherosclerosis of the arteries, which extends to the origin of perforating branches leading to occlusion [1]. Louis Caplan developed the idea of BAD in 1989 based on autopsy results described as an obstruction at the start of a deep penetrating artery in the brain related to a junctional plaque or micro atheroma and caused a small infarct in the internal capsule or pons [2]. Although it is considered a mild deep brain infarct, this condition can result from either atheromatous occlusion, as seen in Branch Arterial Disease, or lipohyalinotic degenerative changes, which are often associated with a true lacunar infarct [3]. Because the prognosis and treatment approaches for these two vascular disorders differ, it is essential to distinguish between them [4].

Ten to fifteen percent of cases of acute ischemic stroke (AIS) are BAD, a prevalent subtype of AIS [5]. BAD-induced stroke has been strongly associated with worse outcomes than lacunar strokes, including high rates of early neurological deterioration (END), recurrent transient ischemic events, and disability. Within 48 to 72 hours of the stroke's commencement, END happens in 17% to 75% of BAD-related stroke [1,6]. Based on their neurological symptoms or signs and the results of their admission magnetic resonance imaging (MRI), patients with BAD are frequently misdiagnosed as having lacunar infarcts brought on by lipohyalinotic degeneration. This leads to a miscalculation of the estimated prognosis and a delay in therapeutic therapy [7].

BAD is mainly determined by indirect imaging findings, such as particular morphological traits of the ischemic lesion thought to be produced by it because conventional imaging techniques cannot show tiny vessel alterations [2,8]. Infarctions in the subcortical regions in which blood perfusion dependent on deep and small perforating arteries were commonly referred to as BAD-related infarctions [2]. The lenticulostriate artery (LSA), anterior choroidal artery, thalamoperforating artery, paramedian pontine artery (PPA), and Heubner's artery are among the arteries that are classified as perforating arteries. The characteristics of ischemic lesions are used to indirectly study the involvement of LSA and PPA [6]. Recent studies have shown that high-resolution magnetic resonance imaging (HRMRI) with T2-weighted turbo spin echo (T2 TSE) sequence with black blood technique and 3D mapping can be used to study intracranial artery vessel morphology, including atherosclerotic plaque, irregular wall thickening, arterial remodelling, and focal geometrical features of the artery. These features are all expected to be crucial vascular risk factors in the development of atherosclerosis [4,9].

CASE REPORT:

A 58-year-old man with hypertension, diabetes, and dyslipidemia suddenly became weak on the left side of his body; however, he was still able to ambulate. The symptoms worsen in the evening, accompanied by dysarthria when the patient cannot ambulate. When he was brought to our hospital, a neurological test showed that he had left hemiparesis in his arm, leg, face, and dysarthria with GCS E4V5M6, blood pressure 142/78 mmHg, NIHSS score 8 – which increased to 10 after 3 hours of admission, and high C-Reactive Protein (CRP) level (164 mg/dl). The patient also routinely consumed direct oral anticoagulants (DOAC) because there was a history of deep vein thrombosis. MRI and MR angiography (MRA) revealed acute right basal ganglia, corona radiata infarction, and right terminal middle cerebral artery (MCA) segment M1 stenosis (Figure 1). Digital subtraction angiography (DSA) showed mild stenosis at the right MCA (Figure 2). Based on focal geometric characteristics of the middle cerebral artery, our patient is categorized as a straight type

of M1 on both sides (Figure 3). Despite starting a statin and antiplatelet medication, his left hemiparesis worsened, and the next day, he went flaccid. The patient was then discharged and continued with rehabilitation therapy.

DISCUSSION:

Branch atheromatous disease (BAD), which causes occlusion or stenosis at the penetrating branch's entrance due to localized atherosclerotic plaque-based thrombus, contributes 10.3%–10.8% of all cerebral infarctions [9]. BAD is distinguished from the other small and big arterial disorders by a single subcortical infarction greater than a lacunar stroke in the regions of deep perforators without accompanying severe arterial stenosis [5,10]. BAD-related stroke is categorized as an embolic stroke of unknown source (ESUS) under the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification, which is commonly used to categorize cerebral infarction [10]. Since different vascular pathologies require distinct treatments, it should be possible to distinguish between lacunar infarct induced by lipohyalinotic degenerative changes (LD), and the primary differential diagnosis of BAD-related stroke [3]. Some epidemiologic studies indicate that BAD is more prevalent in male patients with age ≤ 60 years old who reside in Eastern Asia and Eastern Europe as opposed to Western nations. Patients with BAD are more likely than those with LD to have hypertension and hyperlipidemia [5,11].

Based on clinical symptoms, BAD is inferred when there is the progressive and gradual development of the ischemia, based on the signs and symptoms, suggesting intrinsic “thrombotic” disease rather than occult embolism [7]. Despite being understudied and lacking a recommended treatment, BAD-related stroke is linked to significant rates of early neurological deterioration (END) and disability. There are several criteria for END. First, the interval of symptoms is approximately seven days after the onset. The neurologic impairments worsen after the initial evaluation, which includes an increase of at least four points in the NIHSS score or a rise of at least one point in the NIHSS motor score for ischemic patients, or the attacks continue to occur at least three times after hospitalization or progress to persistence status for patients with internal capsule warning syndrome or pontine warning syndrome. Elevated inflammatory markers and CRP were also predictive of poor outcomes of the stroke [6,10].

The vascular territory and/or shape of the acute ischemic lesion are used to radiologically diagnose BAD because the branching arteries may not be readily visible by conventional imaging techniques [2]. LSA and PPA are the most frequently affected of the previously mentioned perforating arteries. The lenticulostriate arteries supply the lateral part of the globus pallidus and the head of the caudate nucleus, the anterior limb of the internal capsule, putamen, and the anterior part of the periventricular corona radiata [3]. In the LSA area, BAD is diagnosed using the radiological criteria listed below: a) A "comma-like" infarct lesion and has a "fan-shaped" extension that from bottom to top on the coronal slice with size more than 10 mm in diameter on axial slice, visible on 3 or more axial slices on diffusion-weighted imaging (DWI) of the LSA area; b) MRA or computed tomography angiography (CTA) or digital subtraction angiography (DSA) demonstrate that the parent artery of the diseased vessel (corresponding middle cerebral artery) does not have > 50% stenosis [9,10]. Additional radiological features can aid in diagnosing Branch Atheromatous Disease (BAD). Notably, leukoaraiosis and microbleeds, which are indicators of small vessel disease, are less frequently observed in patients with BAD [8]. BAD can develop from conditions such as arterial tortuosity, which can develop due to hereditary conditions, advanced age, and hypertension. It can also cause haemodynamic alterations and initiate the

formation of atherosclerosis in specific vessel [11]. It is anticipated that focal geometric features will also be a significant vascular risk factor in the onset of atherosclerosis. Because the shear stress is weak on the inside of the curve, plaque is likely to form at blood vessel curvatures.
The M1 segment morphology can be classified as either straight or curved based on pictures from three-dimensional time-of-flight magnetic resonance angiography (MRA) in coronal projection. From the anterior cerebral artery and M1 bifurcation site, a line was drawn to the M2 bifurcation point. The curve was categorized as upward type M1 if its vertex was above this line and downward type if it was below. If it had no vertices, it was classified as straight type. According to a study by Nagasawa, 2023, since the lenticulostriate artery typically branches out from the superior side of the MCA M1 segment, plaque is likely to form at the entry of the penetrating branch, making BAD more likely to occur in patients with downward type M1. Additionally, straight type M1 is more likely to be the site of plaque rather than upward type M1 [9].

CONCLUSION:

Diagnosing and differentiating BAD with LD is essential to determine the patient's initial management. Since BAD is caused by arteriosclerosis-induced non-cardiogenic cerebral infarction, antiplatelet medicine is the primary treatment for this condition. Antithrombotic medication should be given if BAD is identified at admission to avoid END [10]. Classifying BAD-related stroke as a distinct stroke subtype and investigating whether these patients will experience different outcomes or treatment responses in comparison to other traditional stroke subtypes are suggested for further research. This underutilized idea can further be strengthened by developing and validating the diagnostic criteria for BAD using advanced imaging modality.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:

This work received no external funding.

CONFLICT OF INTEREST:

The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

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Yamada T, Liu YY, Hart RG, NAVIGATE ESUS Investigators. Branch atheromatous disease diagnosed as embolic stroke of undetermined source: a sub-analysis of NAVIGATE ESUS. International Journal of Stroke. 2019 Dec;14(9):915-22.

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FIGURE LEGENDS:

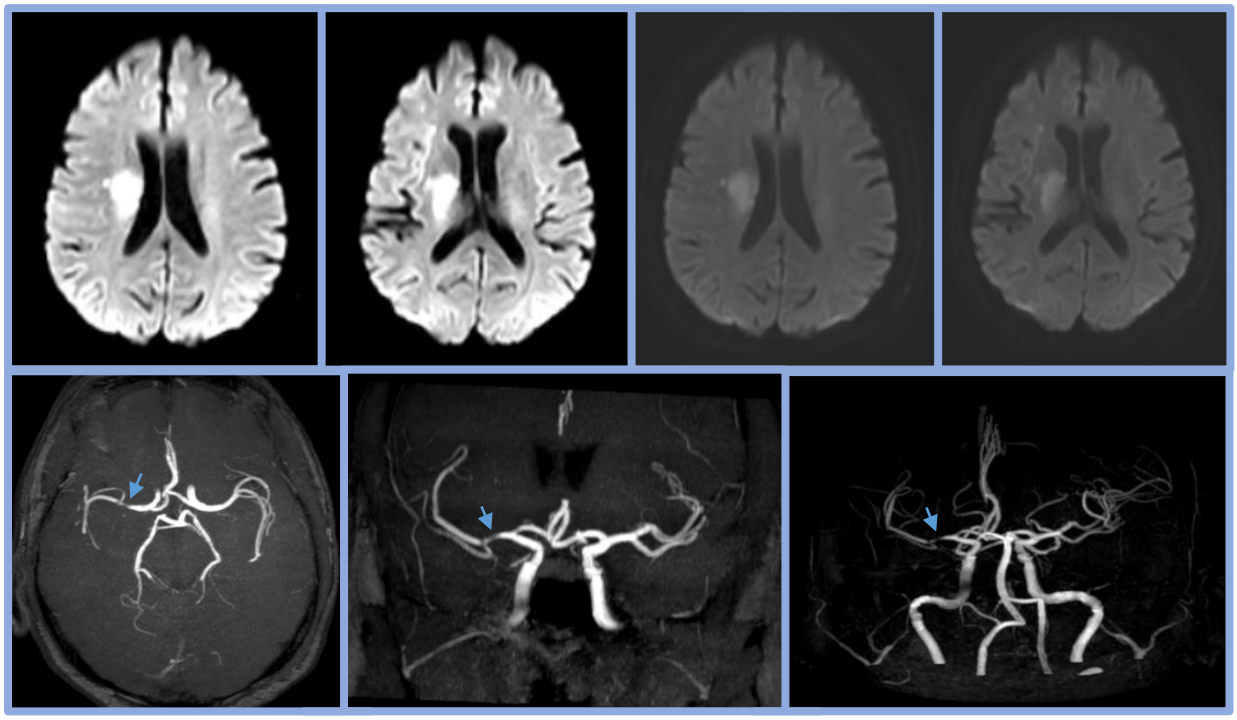

Figure 1: MRI and MR Angiography showed acute right corona radiata and basal ganglia infarction, and right terminal middle cerebral artery (MCA) segment M1 stenosis.

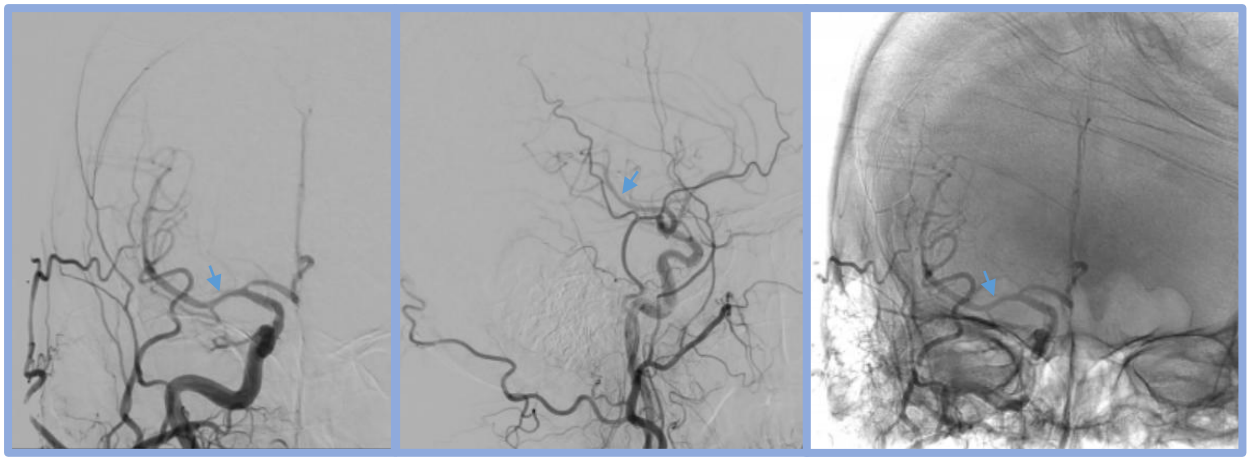

Figure 2: Digital subtraction angiography (DSA) showed mild stenosis at right MCA but was less than 50%.

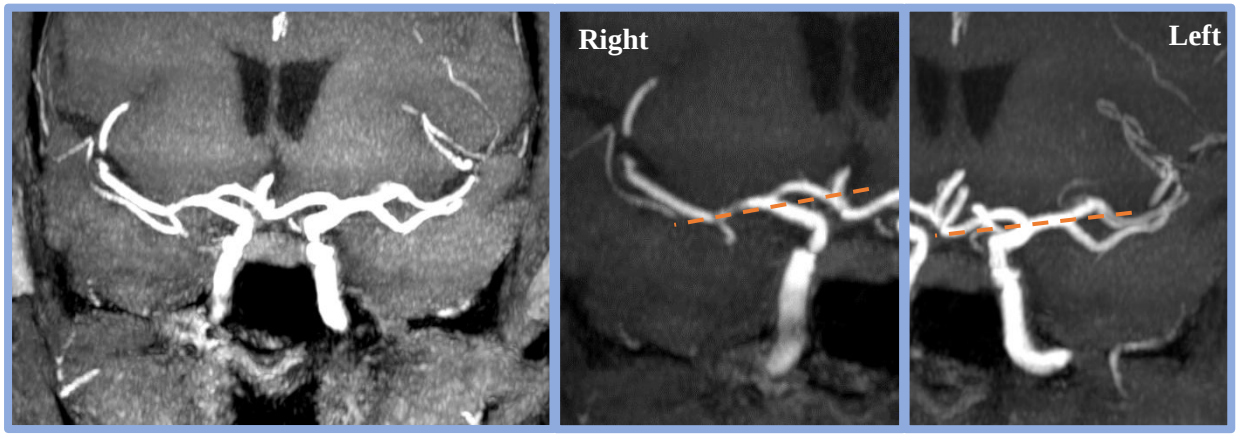

Figure 3: M1 segment shape classification on the coronal maximum intensity projection images of three-dimensional time-of-flight magnetic resonance angiography. A line was drawn from the M1 origin to the M2 bifurcation, and the curves' vertices were evaluated based on the position of the line. Our patient shows the straight type of both MCA (M1 segment).

CASE REPORT: SUCCESSFUL STAGED EMBOLIZATION OF A DIRECT CAROTID-CAVERNOUS FISTULA USING COMBINED COIL AND LIQUID EMBOLIC TECHNIQUE

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DOI: https://doi.org/10.32896/cvns.v7n1.19-27
Received: 09.03.2024
Revised: 28.03.2025
Accepted: 29.03.2024
Published: 31.03.2024

ABSTRACT

Carotid-cavernous fistulae (CCFs) are a common complication after head trauma, accounting for 75% of all cases. They typically present with proptosis, ocular bruit, and chemosis, known as the classical triad. However, other symptoms may occur depending on the involvement of vascular and neural structures in the cavernous sinus. Digital Subtraction Angiography (DSA) is the gold standard for diagnosing CCFs. Traditionally, treatment involved open surgical procedures such as carotid ligation, trapping, and cavernous sinus exploration. Other options include conservative management and radiosurgery. In recent years, endovascular treatment (EVT) has become the preferred approach. Both transarterial and transvenous embolization using various agents aim to completely close the fistula while preserving carotid artery flow.
This report presents the successful management of a right direct CCF in a 47-year-old male following a motor vehicle accident (MVA). A staged embolization approach, combining transvenous and transarterial techniques, effectively reduced the residual fistula without immediate complications. This case highlights the complexities of post-traumatic CCF treatment and the importance of tailored endovascular strategies.

Keywords: Neurointerventional surgery, carotid-cavernous fistula, digital subtraction angiography, endovascular procedure, therapeutic embolization

INTRODUCTION:

Carotid-cavernous fistulae (CCFs) are abnormal arteriovenous connections between the carotid arterial system and the cavernous sinus, often resulting from head trauma. These fistulas can cause increased venous pressure, leading to significant ophthalmological and neurological symptoms such as proptosis, chemosis, elevated intraocular pressure, vision impairment, and cranial nerve dysfunction. Symptoms vary depending on the severity of the fistula and its impact on vascular and neural structures [1-6]. CCFs are classified based on the Barrow classification. Type A (Direct CCF) is a direct connection between the internal carotid artery (ICA) and the cavernous sinus, often caused by trauma. Types B-D (Indirect CCFs) involve branches of the internal or external carotid artery and are usually spontaneous or due to underlying vascular abnormalities [7-9]. Diagnostic approach involving the noninvasive imaging methods, such as CT, MRI, and CT/MR angiography, can help identify cavernous sinus enlargement, superior ophthalmic vein dilation, and extraocular muscle swelling, which are suggestive of CCF. However, digital subtraction angiography (DSA) remains the gold standard for definitive diagnosis and treatment planning [5]. Management strategies involving the endovascular treatment (EVT) has become the first-line approach due to its minimally invasive nature and high success rates. The goal is to completely occlude the fistula while preserving carotid artery function. Mild or low-risk CCFs may resolve spontaneously.

Urgent intervention is needed for cases with pseudoaneurysms, large venous varices, cortical venous drainage, or venous thrombosis, which increase the risk of hemorrhage or neurological deterioration. Two primary endovascular techniques are commonly used. Transarterial embolization is preferred for direct, high-flow CCFs, often using detachable balloons or other embolic agents. However, complications such as balloon rupture, premature deflation, or detachment may occur. Transvenous embolization is used for indirect fistulas or cases where transarterial access is challenging. Studies report a 70–90% success rate with this approach [1].

This case report presents the successful management of a post-traumatic direct CCF in a 47-year-old male following a motor vehicle accident. A staged embolization approach combining transvenous and transarterial techniques was performed, leading to significant reduction of the fistula without immediate complications. This case highlights the complexities of CCF treatment and emphasizes the importance of tailored endovascular strategies based on individual patient anatomy and fistula characteristics.

CASE REPORT:

A 47-year-old male was involved in a motor vehicle accident (MVA) in September 2024. 4 days later, he developed vision loss in his right eye, proptosis, conjunctival chemosis, elevated intraocular pressure, absence of eye movement, and pulsatile tinnitus on the right side. Initial imaging suggested a right carotid-cavernous fistula with acute intraparenchymal hemorrhage in the right cerebellum (Figure 1). He also sustained fractures of the right sphenoid and petrous part of the right temporal bone. One week after his MVA, an initial coil embolization procedure was performed using both transvenous and transarterial access. Seven fibered coils were deployed into the venous sac of the cavernous sinus, resulting in partial occlusion of the CCF. An attempt to use a detachable silicone balloon was unsuccessful due to the small orifice of the fistula. A post-procedure angiogram showed only minimal reduction in CCF flow (Figure 2). After more than six hours, the procedure was stopped, and a staged embolization approach was planned. Staged embolization involves performing embolization in multiple sessions rather than all at once. This approach helps maintain normal blood circulation, allows the body to gradually adapt, and minimizes

complications. However, there was no significant improvement, as the patient continued to experience symptoms.

A second embolization was performed 2 weeks after MVA, employing a more aggressive and multifaceted approach. One fibered coil was deployed into the right cavernous sinus, followed by the injection of 1 ml of Precipitating Hydrophobic Injectable Liquid (PHIL) 30%. An Eclipse 2L balloon catheter was inflated at the fistula point of the right ICA to prevent reflux into the parent artery. This procedure was repeated several times until satisfactory occlusion was achieved. Post-procedure angiogram showed significant reduction of the CCF flow, with only approximately 20% residual flow remaining. After five months, MRA showed complete resolution of the CCF. Vertigo, right ear ringing, right eye bulging has already resolved and right eyelid drooping improved. Patient ambulating well without assistance and able to drive again.

DISCUSSION:

This case exemplifies the complexities involved in managing direct post-traumatic CCFs and highlights several key points in their treatment. Traumatic CCFs constitute the majority of direct CCFs, accounting for approximately 87.24% of cases [4]. The patient's presentation with classic signs of orbital venous congestion is typical for high-flow direct CCFs. Endovascular embolization has become the gold standard in CCF management. The combination of transvenous and transarterial techniques, as employed in this case, offers enhanced control over embolic materials and reduces procedural risks.

The second procedure demonstrates the effectiveness of a multi-modal approach in challenging cases. The use of PHIL 30% as a liquid embolic agent, in conjunction with fibered coils and balloon-assisted techniques, provided precise control and minimized the risk of embolic migration [7]. PHIL 30% has higher viscosity enable more control and less risk of penetration beyond the venous sac of the fistulae. This approach aligns with current trends in neurointerventional practice, where combinations of embolic materials are used to achieve optimal results. The staged embolization effectively decreased fistula flow while preserving adjacent vascular structures, demonstrating the value of adaptable treatment strategies. In cases with small fistula orifices and complex venous drainage patterns, achieving complete occlusion in a single session may not always be feasible.

The incorporation of PHIL, a relatively new liquid embolic agent, in the second procedure is noteworthy. PHIL offers several advantages, including reduced artifacts on follow-up imaging and a cohesive nature that minimizes the risk of distal embolization. Its use in this case, combined with balloon protection, exemplifies the ongoing evolution of embolic techniques in neurointerventional procedures. PHIL's unique properties make it particularly suitable for CCF embolization. Its non-adhesive nature allows for more controlled delivery, while its precipitating mechanism provides rapid and stable occlusion. The tantalum-based radiopaque component offers excellent visibility during injection, enabling precise placement and reducing the risk of non-target embolization.

While multiple embolization procedures can be physically and emotionally challenging, patient accept them as necessary for better long-term results. Clear communication with the medical team, psychological support, and reassurance about the safety and benefits of staged embolization can help ease their concerns.

While staged embolization improves safety and treatment success, it increases hospitalization duration and financial strain. Patients may need financial planning, insurance support, and social assistance programs to help manage costs.

CONCLUSION:

This case report illustrates the successful management of a complex, post-traumatic direct CCF through a staged endovascular approach. The combination of transvenous and transarterial techniques, along with the use of both traditional (coils) and novel (PHIL) embolic agents, proved effective in significantly reducing fistula flow. This case underscores the importance of individualized treatment planning in CCF management. It highlights the value of a flexible approach, where initial incomplete results can be addressed through subsequent, more aggressive interventions. The use of cutting-edge embolic agents like PHIL, when combined with established techniques, offers new possibilities in tackling challenging neurovascular lesions. Future follow-up will be crucial to assess long-term outcomes and the potential need for additional treatment. This case contributes to the growing body of evidence supporting multi-modal, staged approaches in the management of complex CCFs, and underscores the ongoing evolution of neurointerventional techniques in addressing these challenging vascular anomalies.

DATA AVAILABILITY:

Further information regarding the data used for this work can be obtained from the corresponding author upon reasonable request.

FUNDING:

This work received no external funding.

CONFLICT OF INTEREST:

The authors have no conflicts of interest to declare and is in agreement with the contents of the manuscript.

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FIGURE LEGENDS:

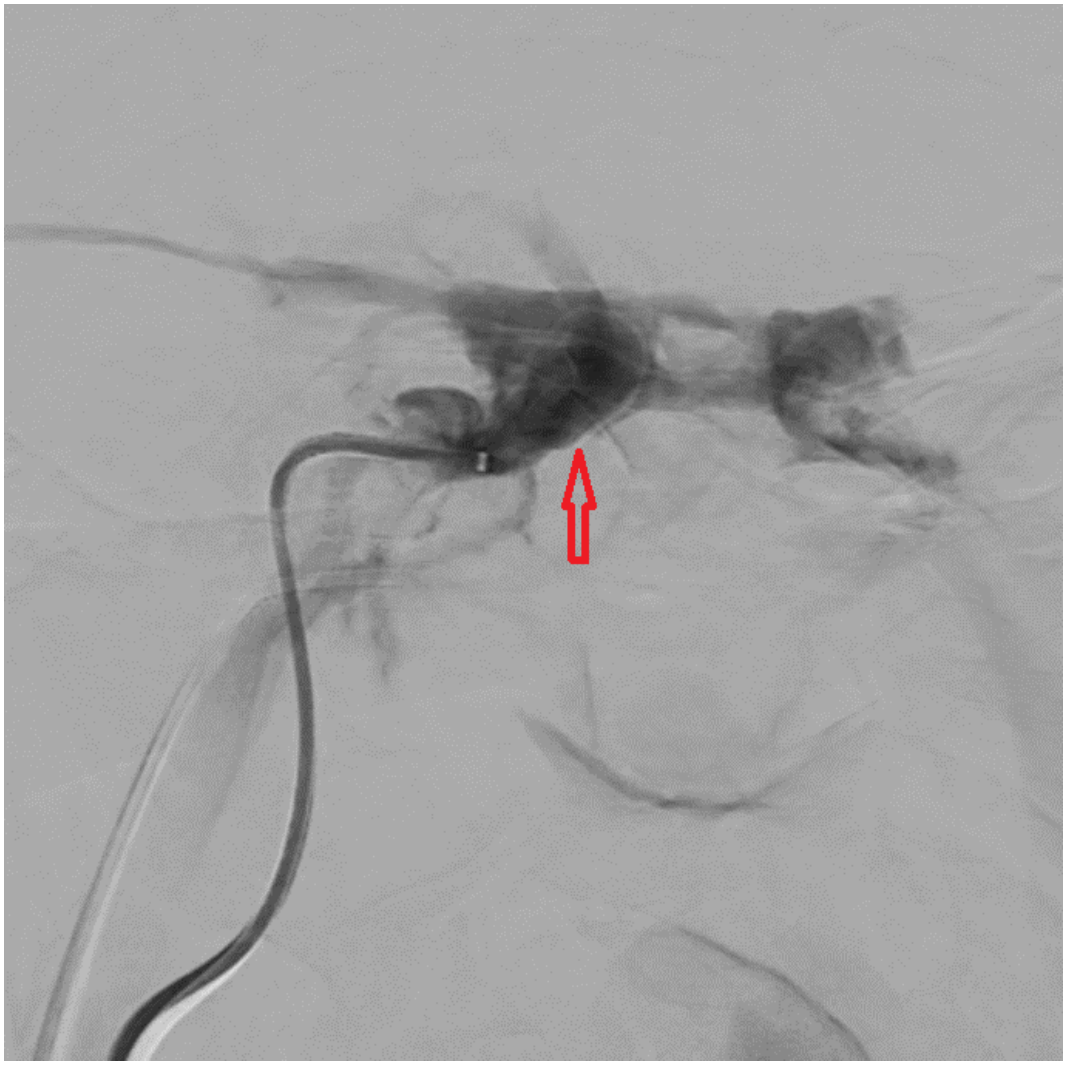

Figure 1: Cerebral angiogram pre procedural showed a right direct CCF (arrow).

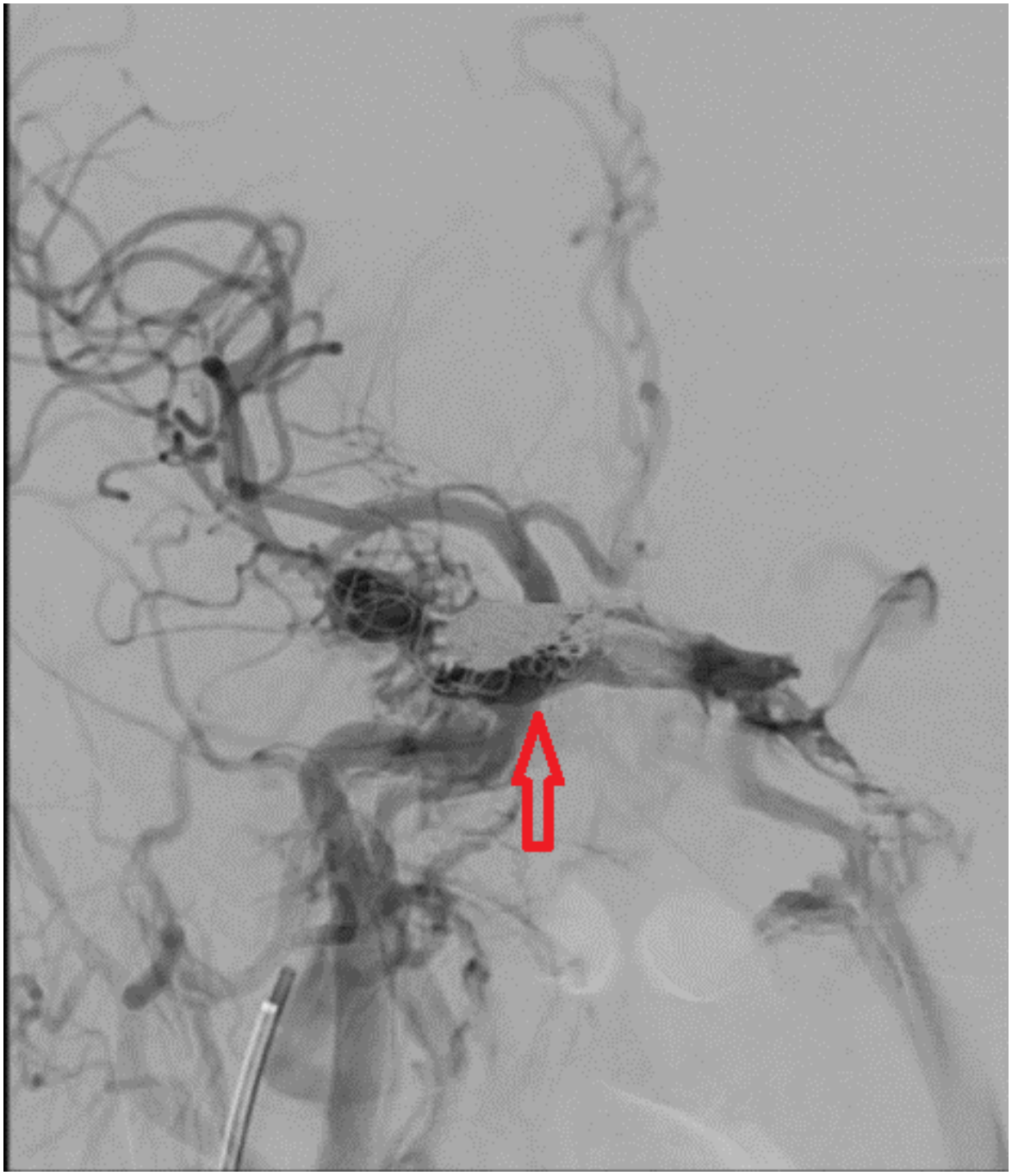

Figure 2: Partial occlusion of the CCF after 1st embolization procedure with seven fibered coils (arrow) successfully deployed into the venous sac of the cavernous sinus.

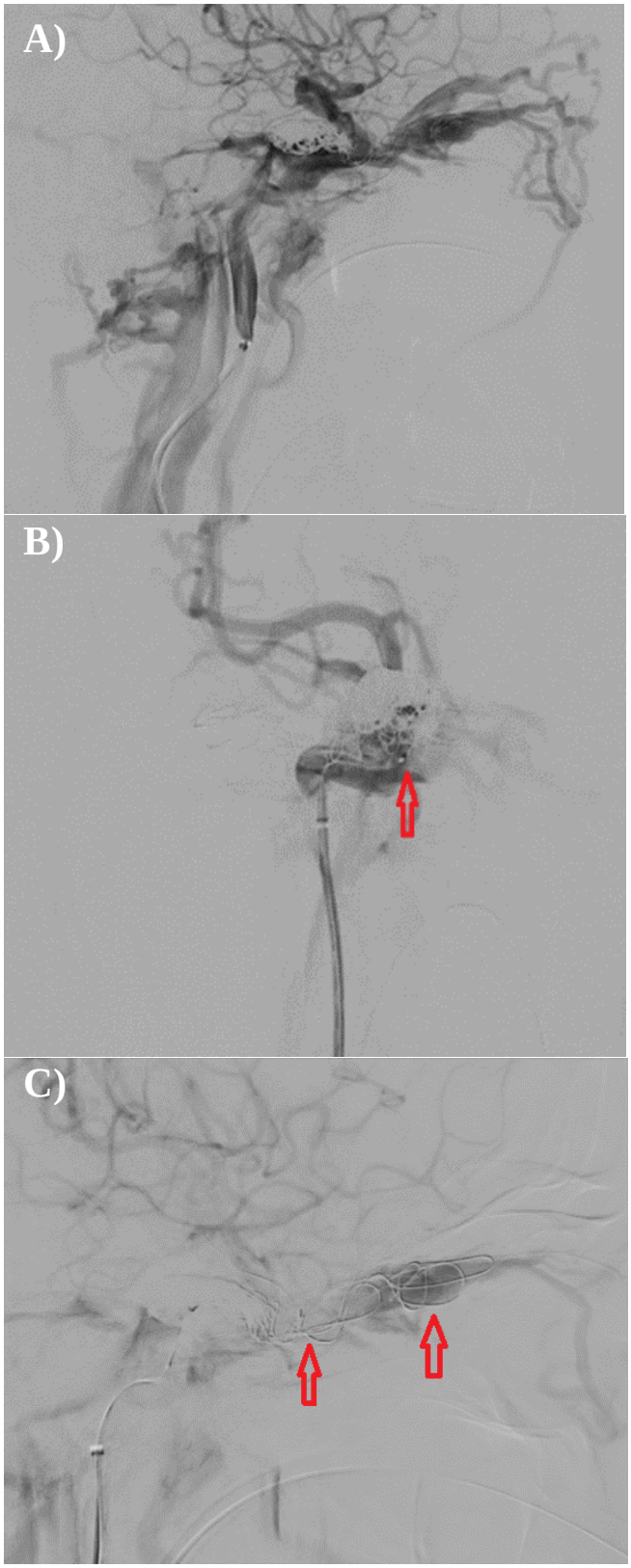

Figure 3. Cerebral angiogram, right ICA. A: lateral projection pre. B: AP projection post. C: Lateral projection post. Second embolization successfully deployed one fibered coil (arrow) followed by the injection of 1 ml of Precipitating Hydrophobic Injectable Liquid (PHIL) 30%. Cerebral angiogram post procedure showed significant reduction of the CCF flow.

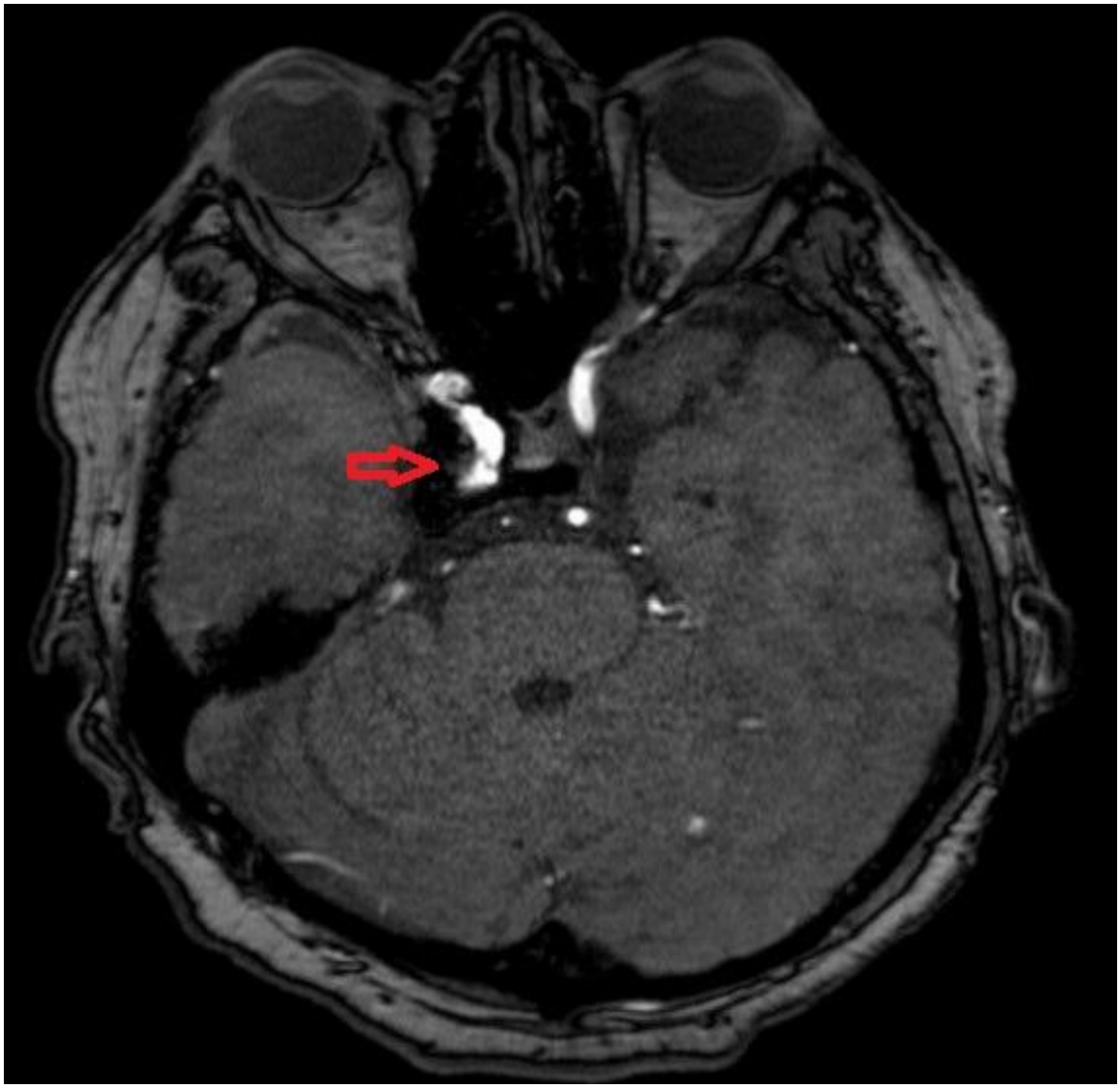

Figure 4: MRA 5 month later showed complete resolution of right CCF (arrow) with patient symptoms have improved.