Rapidly Progressive Dementia and Parkinsonism Secondary to Intracranial Dural Arteriovenous Fistula in a 60-year-old Male: A Case Report

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ABSTRACT

Simultaneous rapidly progressive dementia with parkinsonism is a rare initial presentation of dural arteriovenous fistula. Most cases of dural arteriovenous fistula present with either sudden-onset headache and cerebrovascular disease hemorrhage or other benign non-hemorrhagic symptoms. We present a unique case of a 60-year-old male who presented with progressive slowness of movement and forgetfulness. Physical examination was initially significant for bradykinetic movements, slowed gait, and reduced arm swing bilaterally, along with a MoCA score of 15/30, indicating moderate cognitive impairment. Cerebral catheter angiography revealed the presence of a complex tentorial-torcular-left transverse sinus-sigmoid sinus dural arteriovenous fistula. A month after the diagnosis, he eventually became stuporous and bedbound, with episodes of generalized tonic-clonic seizures, and was thus given Levetiracetam 500 mg twice daily. The patient underwent partial arterial feeder onyx embolization with a transverse approach. He was admitted to intensive care post-operatively, with gradual improvement of stupor and no seizure recurrence thereafter. Upon discharge, he was maintained on Memantine, slowly increased to 10 mg twice daily. Seven months after embolization, the patient ambulated without assistance. Although bradykinesia and dementia were still present, this represented a significant improvement from the condition pre-embolization. A high index of suspicion and awareness of this rare acquired cause of rapidly progressive dementia and parkinsonism is needed to avoid delays in the diagnosis and appropriate management.

Keywords: dural arteriovenous fistula, rapidly progressive dementia, Parkinsonism, DAVF-derived Parkinsonism, case report

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INTRODUCTION

Intracranial dural arteriovenous fistulae (DAVF) refer to pathologic anastomoses between meningeal arteries and dural venous sinuses or cortical veins. They have a diverse range of manifestations, with some being merely incidental findings on imaging, others presenting with only benign symptoms such as tinnitus and other ophthalmologic phenomena, and some with aggressive presentations such as intracerebral hemorrhage and effects of cortical venous hypertension.[1] Some DAVF may present as either rapidly progressive dementia in 12-22 % or Parkinsonism in 3-12% of the cases, but it rarely occurs simultaneously.[2,3] Herein, we present a case of dural arteriovenous fistula presenting with rapidly progressive dementia and Parkinsonism.[3]

CASE PRESENTATION

A 60-year-old Filipino male, hypertensive and diabetic, presented with slowness of movement and forgetfulness. He

was initially admitted to a local hospital due to gradually progressive gait difficulty and slowness of movement. The initial non-contrast cranial CT scan was unremarkable and was thus treated as a case of transient ischemic attack and prescribed Clopidogrel 75 mg, 1 tablet daily. However, his symptoms continued to progress and were now accompanied by cognitive decline a month later, prompting consultation at our institution. His systemic physical examination was unremarkable. He was bradyphrenic, needing prodding to respond and follow instructions. He scored 15/30 on the Montreal Cognitive Assessment (MoCA-English, version 7.1), indicative of a moderate cognitive impairment. He had masked facies, hypophonia, bradykinesia, micrographia, rigid extremities, and reduced bilateral arm swing on gait examination (Video 1; with written consent obtained). Family and psychosocial history were noncontributory regarding neurodegenerative disorders, including movement disorders, Parkinson's disease, and dementia.

A cranial magnetic resonance imaging with angiography and venography (MRI with MRA and MRV) showed flow signal findings of the straight and transverse sinus suspicious for a carotid-cavernous fistula, with prominent left cavernous sinus, bilateral superior ophthalmic veins, and both sphenoparietal sinuses (Figure 1a and 1b). Electromyography, nerve conduction studies, electrolytes, and thyroid panel were unremarkable. Other considerations of a left middle cerebral artery territory ischemic stroke, arteriovenous malformation, vascular dementia, and metabolic or Hashimoto encephalopathy were less likely at this time. Owing to MRI findings, Clopidogrel was discontinued, and cerebral catheter angiography was performed, which revealed a complex tentorial-torcular-left transverse sinus-sigmoid sinus dural arteriovenous fistula (Figure 1c). The patient was advised to undergo fistula embolization, but opted to delay due to financial constraints.

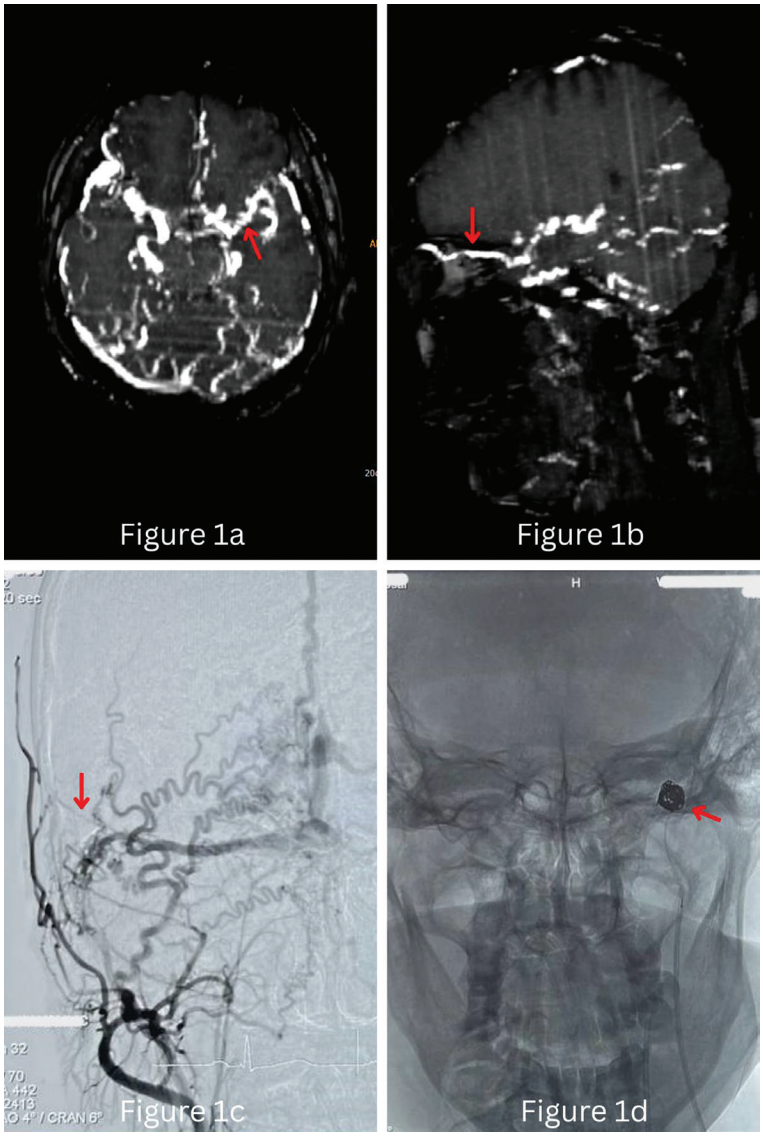

Figure 1. **(A, B)** Cranial MRI suspicious for carotid-cavernous fistula, with prominent bilateral sphenoparietal sinuses and superior ophthalmic veins *(red arrow)* seen on MRV. **(C)** Cerebral catheter angiography showing the presence of a complex tentorial-torcular-left transverse sinus-sigmoid sinus dural arteriovenous fistula *(red arrow)*. **(D)** The cerebral angiography image shows the insertion of a coil *(red arrow)* during partial arterial feeder onyx embolization with a transverse approach.

A month later, the patient's condition severely worsened, now becoming stuporous and bedbound, with episodes of generalized tonic-clonic seizures, likely secondary to venous congestion and edema. He was admitted and started on Levetiracetam 500 mg twice daily, then underwent partial arterial feeder onyx embolization via a transverse approach (Figure 1d) instead, as total embolization would acutely worsen venous congestion and edema.

Post-embolectomy, the patient was admitted to the intensive care unit, with gradual improvement of his consciousness and resolution of his seizures, and was eventually discharged. He then underwent continuous intensive physical therapy. During discharge, he was maintained on Levetiracetam 500 mg twice daily, and Memantine was slowly increased to 10 mg twice daily. Adherence to the intervention was confirmed by completion of the planned endovascular embolization as documented in the procedural report. Tolerability was assessed through intra-procedural monitoring, post-procedural neurologic assessment, and surveillance for complications. On follow-up seven months after embolization, a repeat MoCA was done, and he scored 12/30 (Figure 2). There was still bradykinesia, but he could

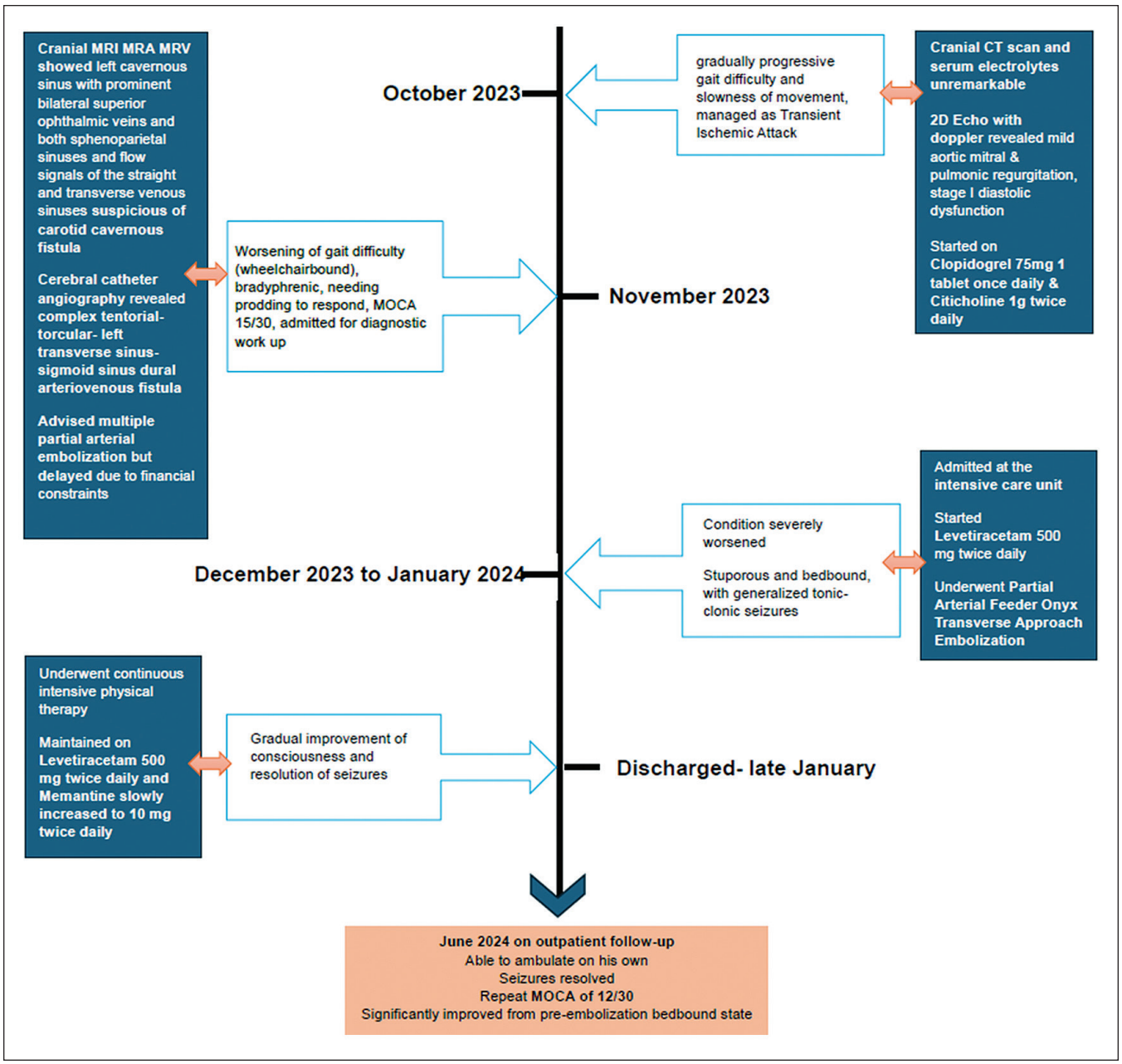

Figure 2. ***Timeline of Disease Progression.*** A graphical figure depicting the onset of symptoms (gait difficulty and slowness of movements), development of seizures with decline in sensorium, primary diagnostic workup, and therapeutic intervention.

Table 1. Reported Cases with Corresponding Fistula Location, Clinical Manifestations, Treatment, and Outcomes

References	Number of cases	Location of fistula	Clinical manifestation	Treatment (including medications tried, surgical interventions)	Outcomes
Xie et al., 2023 [7]	2	Sagittal sinus and bilateral transverse sinus	Parkinsonism Dementia	Levodopa + Benserazide (Madopar), Refused endovascular treatment	Worsened
		Straight Sinus fistula combined with intracranial venous thrombosis	Parkinsonism Dementia	Anti-viral treatment	Expired
Kawasaki et al., 2022 [8]	1	Right transverse sigmoid sinus and Right sigmoid sinus	Parkinsonism	Levodopa 400 mg/day, Intravascular embolization	Improved
Prosperini et al., 2022 [9]	1	Left transverse sinus	Rapidly progressive parkinsonism Dementia	Levodopa, Transarterial embolization	Improved
Tominaga et al., 2022 [3]	1	Left sigmoid sinus	Parkinsonism	Levodopa, Transarterial embolization	Improved
Velz et al., 2020 [10]	1	Straight Sinus	Parkinsonism Dementia	Suboccipital mini-craniotomy with microsurgical disconnection of fistula, followed by Levodopa Carbidopa and Ropinirole post-surgery	Improved
Chang et al., 2019 [11]	1	Left Transverse sinus	Parkinsonism	Combined surgery, puncture of the left transverse sinus directly through a burrhole in the left occipital bone, and embolization	Improved
Wiblin et al., 2019 [12]	1	Straight sinus	Parkinsonism	Levodopa 125mg three times per day, two sessions of embolization with liquid embolic agent (PHIL 25%)	Improved
Enofe et al., 2017 [13]	1	Right transverse sinus	Dementia	Carbidopa- Levodopa, Transarterial endovascular embolization	Improved
Gopinath et al., 2017 [14]	1	Torcular and the Superior sagittal sinus with residual recurrent Torcular dAVF after 6 months	Parkinsonism Dementia	Endovascular Squid embolization with repeat embolization followed by Levodopa + Carbidopa	Improved
Lai et al., 2017 [15]	2	Superior sagittal sinus Both transverse sinuses Torcula Right sigmoid sinus	Parkinsonism Dementia	Levodopa 400mg/day, Transvenous Onyx and Coil Embolization	Improved
		Right transverse-sigmoid sinus	Parkinsonism Dementia	Transvenous coil embolization of the fistula with complete obliteration	Improved
Pu et al., 2017 [16]	1	Straight sinus	Parkinsonism	Endovascular embolization through the left occipital artery	Improved
Ma et al., 2015 [5]	1	Superior sagittal sinus	Parkinsonism Dementia	Transarterial embolization	Improved
Luo et al., 2014 [17]	2	Right transverse sigmoid sinus	Parkinsonism Dementia	Levodopa + Benserazide (Madopar) 187.5 mg every 6 hours Refused endovascular embolization	Lost to follow up
		Left transverse sigmoid sinus	Parkinsonism Dementia	Levodopa + Benserazide (Madopar)	Died
Kural et al., 2011[18]	2	Torcula	Parkinsonism Dementia	Low Molecular Weight Heparin Embolization	Minimal improvement
		Superior sagittal sinus	Parkinsonism Dementia	Unsuccessful transvenous and transarterial embolization, Levodopa	Deteriorated

dAVF – dural arteriovenous fistula

now ambulate on his own, seemingly significantly improved compared to his condition pre-embolization (Video 2).

However, eight months post-embolization, the patient was readmitted due to recurrent difficulty in ambulation and was no longer able to walk. A cranial CT scan showed high-attenuating material likely related to the previous embolization procedure along the right middle meningeal artery, left ascending pharyngeal artery, and left jugular foramen, but no acute intracranial hemorrhage was observed. Electrolytes, thyroid tests, and bleeding parameters remained within normal range at this time. Repeat cerebral catheter angiography showed progression of the bilateral transverse sinus-torcular dural arteriovenous fistula, multiple onyx coils on the bilateral external carotid artery feeders, and a coil mass at the left transverse sinus. Therapeutic options were discussed, but the family chose to be discharged due to financial constraints. Regrettably, a formal patient's perspective was not obtained due to loss to follow-up.

DISCUSSION

Parkinsonism may be seen in 3-12% of cases of DAVF, with most of these patients being male and of Asian ethnicity. Rigidity is noted in most cases of DAVF-derived Parkinsonism, while tremors are seen in half of the affected patients. Our case further highlights the need for MRA and MRV instead of a routine MRI, especially in patients presenting with rapidly progressive Parkinsonism and dementia.[3]

The pathogenesis of DAVF-derived Parkinsonism is poorly understood.[2] The proposed mechanisms include impaired venous outflow leading to basal ganglia dysfunction or partial arterial steal phenomena causing basal ganglia ischemia.[4,5]

Moreover, rapidly progressive dementia may be present in 12-22% of cases of DAVF, and similar to DAVF-derived Parkinsonism, its pathogenesis is also not well known. Proposed mechanisms for dementia include cortical dysfunction secondary to disturbance of CSF resorption from medullary venous congestion and thalamic dysfunction arising from profound venous system hypertension causing bilateral basal ganglia dysfunction, thalamic dysfunction, and posterior fossa stasis.[2]

The natural history of low-grade dural AV fistula without cerebrovascular disease was usually benign, with an up-conversion rate of 0.8%, which may progress to cerebrovascular disease over time due to progressive stenosis, venous outlet thrombosis, increased arterial flow, and recruitment of the extension of the fistulous connection.[6] In our case, based on its characteristic cerebral angiographic findings, the classification according to Zipfel was 3S, Borden-Shucart Type III, and Cognard Type IV, with an intracerebral hemorrhage risk of 7.4-7.6%, 3.8% death risk, and treatment should be early to prevent hemorrhage or non-hemorrhagic neurologic deficits.[1]

In a case series of patients with DAVF-derived rapidly progressive dementia and Parkinsonism, endovascular therapy (13.7%) and surgery (13.7%) were performed, with outcomes mainly improved (56%) and stable (14.7%), but with some cases of worsening (1.9%) and death (2.9%).[2] There are also published cases treated with trans-arterial embolization, levodopa, among others (Table 1).

In patients treated with levodopa, the medication was either discontinued post-embolization due to resolution of the Parkinsonism or discontinued due to lack of benefit.[6,9] Our patient underwent trans-arterial endovascular embolization, which was the preferred treatment for high-grade dural AVF with cortical venous drainage. It took almost three months for the patient to stand and walk again, possibly due to the progression of venous congestion, which takes time to resolve.[6]

Recurrence after initial treatment could occur in complex dural arteriovenous fistulas (dAVFs) with multiple arterial feeders or high-flow characteristics, where even small residual shunts can lead to sustained venous hypertension and persistent neurological symptoms.[15] Although long-term neurologic outcome was not available, and causality cannot be fully established, this patient's follow-up angiography revealed progression of the fistula, illustrating the evolving nature of dAVFs, where recanalization or changes in drainage patterns can occur if the initial embolization is incomplete.[14] The return of gait impairment and cognitive slowing showed the link between hemodynamic changes and symptoms of venous hypertensive encephalopathy, as even slight increases in venous pressure can cause rapid cognitive decline and motor dysfunction.[2] While these symptoms may resemble neurodegenerative disorders, they can be reversible if cortical venous reflux is eliminated, highlighting the need for careful follow-up and complete fistula closure to prevent ongoing neurological injury.[1]

CONCLUSION

Uncommon, acquired causes should be considered in patients with rapidly progressive dementia and Parkinsonism. Herein, we present a case of dural arteriovenous fistula as the cause of the patient's cognitive decline and Parkinsonism. Diagnosis of this condition can be made through neuroimaging, particularly cerebral angiography.

Although this condition may be reversible, this case emphasizes the importance of complete embolization of the fistula and represents a minority of cases with progressive symptoms despite treatment. Recurrent neurologic symptoms after treatment should prompt clinicians to consider the possibility of residual or progressive shunting, emphasizing the need for vigilant follow-up and reintervention. A high index of suspicion and awareness of this unusual presentation are needed to avoid delays, missed diagnoses, and underdiagnosis, and to ensure appropriate management.

Ethical Considerations

Informed consent was obtained separately for the videotapes taken as part of the routine clinical evaluation, and the study has received institutional review board approval. The patient and his family understood that the result could be used for publication, but all personal identification would remain confidential. They have read, signed, and possessed a copy of their Informed Consent Form (ICF). The authors confirmed that they have read the Journal's position on ethical publication and affirmed that this work was consistent with those guidelines.

Data Availability Statement

The video files supporting the findings of this study are available from the corresponding author upon reasonable request.

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Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

All authors declared no conflicts of interest.

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