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Extracranial Thrombosed Vertebral Aneurysm Associated Neurofibromatosis Type1 Treated by Neuroendovascular Coil Embolization: A Case Report and Review of Literature

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ABSTRACT

A 46-year-old NF1 patient with sudden visual disturbance had a thrombotic vertebral artery aneurysm causing cerebral infarction. Endovascular internal trapping was performed successfully, with complete recovery and no neurological deficits. Although this is a rare case, it suggests that endovascular therapy could be successful for vascular lesions complicated by NF1.

1 | Introduction

Neurofibromatosis type 1 (NF1), also known as von Recklinghausen's disease, is an autosomal-dominant hereditary disease characterized by Café au lait spots, neurofibromas, axillary freckles, iris nodules, gliomas, and bone dysplasia [1]. Various osseous or central nervous system disorders associated with NF1 are well known, but arterial lesions, such as aneurysm, vascular stenosis, arteriovenous fistula, are much less well recognized [2, 3]. Orderich et al. reported 12 cases of aneurysms of the vertebral artery among 237 patients with vasculopathy in the scope of NF1, which makes it a rare lesion [2]. And the frequency of angiopathy is reportedly in 0.4%–6.4% of all NF1 cases [4–7]. It usually occurs in the aorta, renal artery, intracranial carotid artery, and vertebral artery, but extracranial vertebral artery lesions are rare [4–6]. Here, we report a patient with NF1 who developed a thrombotic extracranial vertebral fusiformic aneurysm which have caused embolic infarction due to the mechanism of artery to artery embolism.

2 | Case Presentation

A 46-year-old man presented to our department with a chief complaint of visual disturbance that had suddenly developed 2 weeks earlier in 2020. At the time of admission, his Glasgow Coma Scale (GCS) was E4V5M6. Neurological examination showed right upper quadrant homonymous anopsia.

Physiologically, the patient had multiple dermatofibromas, lentigines, and light brown spots on the back that were suggestive of neurofibromatosis. He had a familial medical history and was diagnosed with NF1 based on NF-1 clinical guideline. Routine blood examinations were normal. Chest X-ray echocardiography and electrocardiogram showed no obvious abnormal findings.

Diffusion-weighted image (DWI) of Magnetic resonance image (MRI, Achieva 3.0T TX Quasar, Philips) revealed multiple high-intensity areas at left posterior lobe, cerebellum, and left thalamus of the left posterior cerebral artery (PCA) and the right superior cerebellar artery (SCA) territory (Figure 1A,B). Magnetic

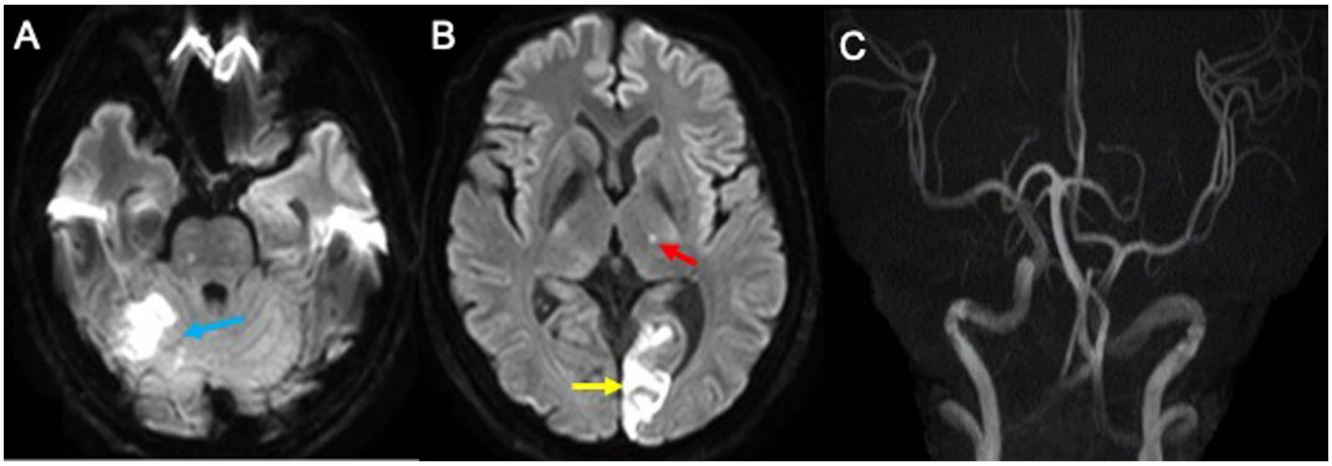

FIGURE 1 | (A, B) MRI diffusion weighted images showed multiple high-intensity areas at left posterior lobe(yellow arrow), right cerebellum(blue arrow) and left thalamus(red arrow) of the left posterior cerebral artery (PCA) and the right superior cerebellar artery (SCA) territory (C) Magnetic resonance angiography (MRA) showing no significant stenosis of the intracranial main artery.

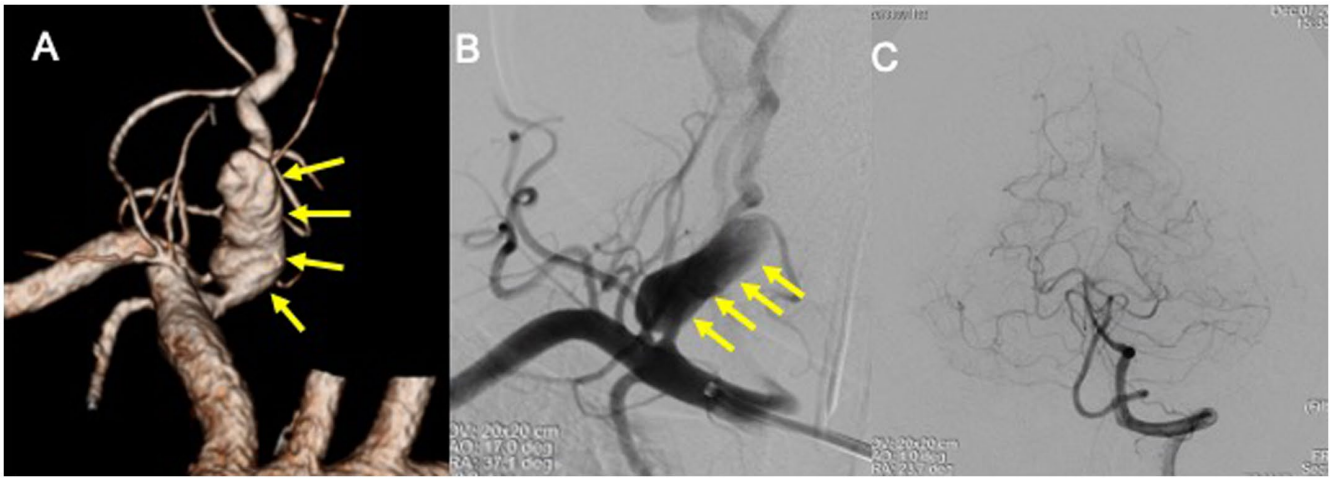

FIGURE 2 | (A) Three-dimensional computed tomography angiography showed a 40 mm diameter fusiformic aneurysm from the proximal right vertebral artery(yellow arrows). (B) Antero-posterior view on right vertebral artery angiography showed a gourd-shaped aneurysm at theV1 segment of vertebral artery (yellow arrows). (C) Antero-posterior view on left vertebral artery angiography showing elongation of intracranial vertebral and basilar artery.

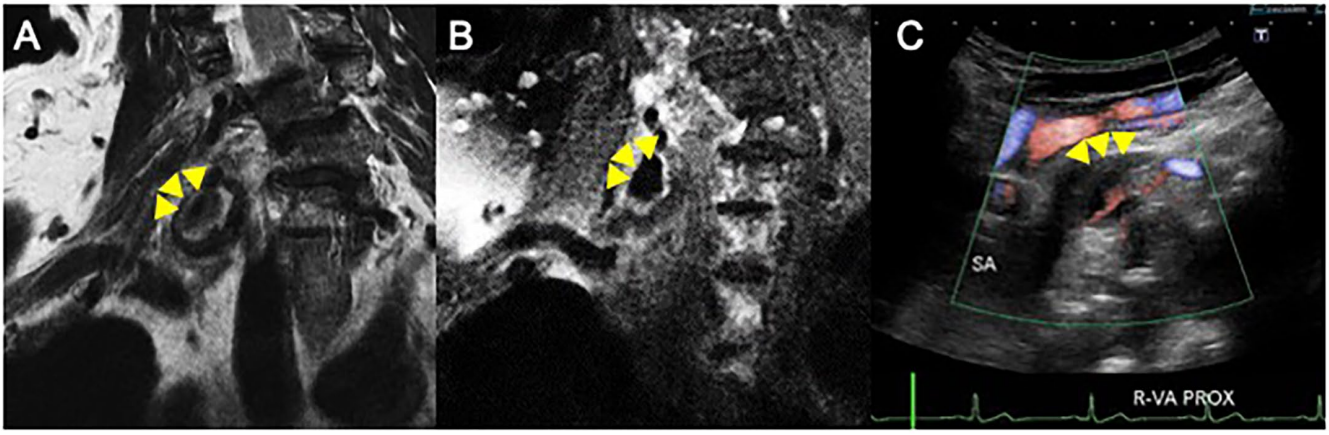

FIGURE 3 | MRI T1 (A) and T2-weighted coronal images (B) showed a low intensity into the aneurysm suggested thrombotic change. (C) Ultrasound sonography also showed thrombotic change as low echo signal at the ventral side of aneurysm (yellow arrow heads).

resonance angiography (MRA) showed no significant severe stenosis or occlusion of the intracranial arteries (Figure 1C). Three-dimensional computed tomography angiography (3D-CTA, Aquilion PRIME, Canon Medical Systems Corporation) and digital subtraction angiography (DSA) demonstrated right subclavian artery fusiformic aneurysm with thrombosis, which diameter was 40 mm involving the right extracranial vertebral artery (VA) at V1 segment (Figure 2A,B). The right intracranial VA was elongated, but there was no severe stenosis or occlusion. There was no difference in the development of both vertebral arteries (Figure 2C). MRI T1- and T2-weighted image showed low intensity area within the aneurysm (Figure 3A,B). And the ultrasonography also showed a low-echo signal on the ventral side of the aneurysm (Figure 3C), which was thought to be a thrombus. Based on above findings, we diagnosed artery to artery embolism induced by right thrombosed giant fusiformic extracranial VA aneurysm associated with neurofibromatosis.

2.1 | Treatment and Follow-Up

To prevent recurrence of embolization and rupture of the aneurysm, medical treatment with antithrombotic therapy (Clopidogrel 75 mg, use for a week) was initially selected because of thrombotic aneurysm and fragility of the arterial wall of NF1. However, rupture of an aneurysm was fatal, and the decision was made to use endovascular therapy as a more aggressive treatment. We planned the internal trapping of the aneurysm by neuroendovascular treatment. We performed internal trapping of the aneurysm to prevent recurrence and rupture of the aneurysm. Under general anesthesia, a 7Fr guiding catheter (Fubuki, Asahi, Japan) was placed at the origin of the right vertebral artery through the right femoral artery, and then double microcatheters (Excelsior SL-10, straight and 45° pre-shaped [Boston Scientific, Natick, Massachusetts, USA]) were placed at distal of aneurysm. Then the coil packing was applied from this region to occlude the distal side of the right vertebral artery (Figure 4A). The aneurysm was almost completely occluded after insertion of all stocked 39 detachable coils. After the procedure, no aneurysm was visualized by postoperative right subclavian artery angiography (Figure 4B), and left subclavian artery angiography showed the right distal VA was filled retrogradely (Figure 4C). Although visual field defect remained, there was no new neurological deficit. After embolization, he had returned to work and was functioning at his premorbid capacity. There have been no apparent aneurysmal recurrences at 4 years of follow-up.

3 | Discussion

Spontaneous extracranial VA aneurysm (EVA) is very rare and usually associated with hereditary connective tissue disorders, including Ehrler-Danlos syndrome, Marfan disease, and neurofibromatosis [8]. To the best of our knowledge, a review of the literature revealed 26 cases of EVA associated with NF1 (Table 1) [2, 3, 7, 9–32]. Clinical presentations might vary and 53% (14/26) of these cases were found after rupture. Size ranged from 15 to 90 mm, and thrombotic aneurysms were found in four of the six presented findings. Aneurysms can be manifested by radiculopathy and neck pain, and unruptured cases can also be symptomatic. Regarding the site of occurrence, the incidence rate in the V1 segment is relatively high, and poor outcome cases ware included.

In our review, there were no reports occurring the cerebral embolism from EVA, so our case is the first reported. The unique feature of our case is that thrombosed aneurysm was the cause of the cerebral embolism. In our patient, medical treatment with antithrombotic therapy (Clopidogrel 75 mg) was initially selected because of thrombotic aneurysm and fragility of the arterial wall of NF1. But we thought that the aggressive treatment should have been performed at the appropriate time, because poor outcome cases have been reported that 21% (3/14) of ruptured EVA associated with NF-1 died of hemothorax and Uranishi et al. reported a case in which an intracranial VA thrombotic aneurysm caused a hemorrhagic infarction in the PICA territory and died by tonsilar herniation [33].

Treatment strategies for EVA described in the literature surgery and endovascular treatment. When discussing treatment strategies, we

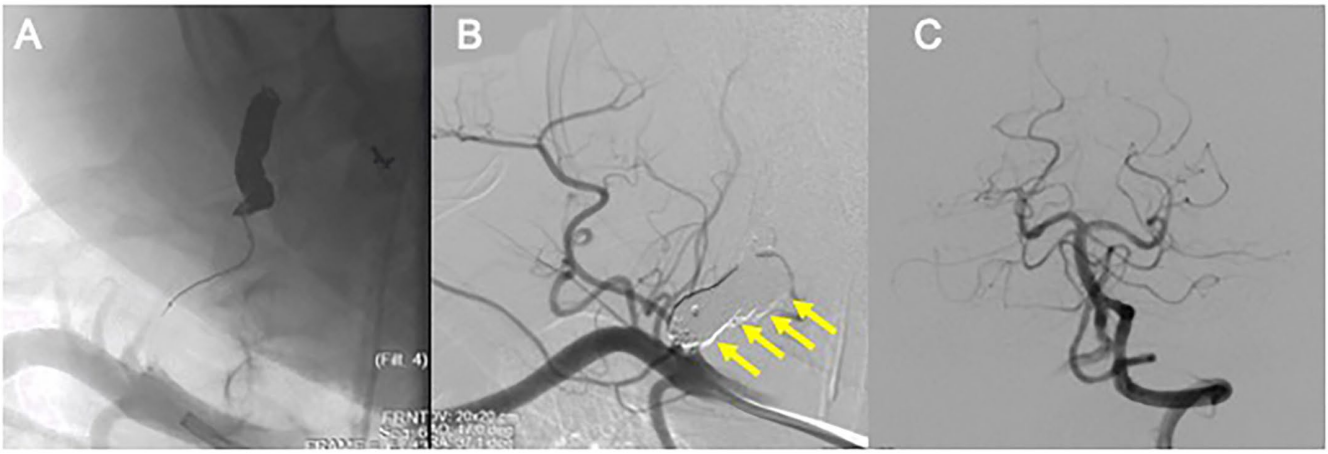

FIGURE 4 | (A) Intraoperative findings: Antero-posterior view on right vertebral angiography: Coiling was filled from the right vertebral artery distal to the aneurysm. (B) Anteroposterior view on postoperative right subclavian angiography confirming complete obliteration of the aneurysm and occlude right vertebral artery (yellow arrows). (C) Anteroposterior view on postoperative left vertebral angiography showed retrograde flow of the right distal vertebral artery.

TABLE 1 | Summary of extracranial vertebral aneurysms associated NF1.

Extracranial vertebral aneurysm associated NF1											
No	**Author**	**Age, Sex**	**Side**	**Location**	**Symptoms**	**Ruptured**	**Size (mm)**	**Thrombosed**	**Treatment**	**Detail**	**Outcome**
1	Schubiger and Yasrgil [9], 1978	50/m	L	V2	Radiculopathy	−	ND	ND	DS	Trapping	NA
2	Pentecost et al. [10], 1981	1/f	L	V1	Limited neck movement	−	ND	ND	DS	Observation (intraoperative bleeding of other aneurysm)	SD
3	Detwiler et al. [11], 1987	52/f	L	V3	Neck pain	−	70×90	+	IVR	Proximal occlusion (balloon)	GR
4	Negoro et al. [12], 1990	47/f	L	V2	Neck pain	+	ND	−	IVR	Proximal occlusion (balloon)	GR
5	Muhonen et al. [13], 1991	52/f	L	V3	Neck pain, radiculopathy	−	ND	ND	IVR	Proximal occlusion (balloon)	GR
6	Schievink and Piepgras [14], 1991	43/f	L	V1	Incidental	−	ND	ND	Observe	Observation	GR
7	Ohkata et al. [15], 1994	48/f	L	V1	Radiculopathy	−	ND	ND	DS	Trapping	GR
8	Horsley et al. [16], 1997	56/f	L	V2	Neck pain, parestesia	+	ND	ND	DS	Internal trapping (coil)	GR
9	Hoffmann et al. [17], 1998	59/m	R	V1	Dyspnoea, tachycardia	−	45×50	−	Observe	Observation	GR
10	Ushikoshi et al. [3], 1999	40/f	L	C3	Occipotalgia	+	ND	ND	IVR	Proximal occlusion (balloon)	GR
11	Miyazaki et al. [7], 2004	52/f	L	C2	Hypotension, altered consciousness, radiculopathy	+	ND	−	Both	Proximal occlusion (balloon)	Death
12	Arai et al. [18], 2007	38/m	L	V1	Chest pain	+	20×20	−	Observe	Observation	Death
13	Hieda et al. [19], 2007	36/f	L	V1	Back pain	+	ND	−	IVR	Internal trapping (coil, NBCA)	Death
14	Oderich et al. [20], 2007	43/f	L	Multiple	Brachial plexopathy	+	ND	ND	DS	Trapping + bypass	GR

(Continues)

TABLE 1 | (Continued)

Extracranial vertebral aneurysm associated NF1											
No	**Author**	**Age, Sex**	**Side**	**Location**	**Symptoms**	**Ruptured**	**Size (mm)**	**Thrombosed**	**Treatment**	**Detail**	**Outcome**
15	Hiramatsu et al. [21], 2007	67/m	L	V1	Dizziness	−	ND	−	IVR	Internal trapping (coil)	GR
16	Pereira et al. [22], 2007	14/f	R	V2	Radiculopathy	−	ND	−	IVR	Internal trapping (balloon)	GR
17	Peyre et al. [23], 2007	18/f	R	V2	Radiculopathy	−	ND	−	IVR	Coil	GR
18	Horie et al. [24], 2008	30/f	R	V1	Radiculopathy	−	ND	+	IVR	Internal trapping (coil, balloon)	GR
19	Higa et al. [25], 2010	60/f	L	C2	Respiratory failure	+	ND	ND	IVR	Proximal occlusion (coil)	SD
20	Morvan et al. [26], 2011	36/f	L	V2	Headache, vomiting	+	13×17	−	IVR	Stent-assisted coil	NA
21	Hiramatsu et al. [27], 2012	31/f	R	V1	Neck pain, radiculopathy	+	ND	−	IVR	Internal trapping (coil)	GR
22	Gouaillier-Vulcain et al. [28], 2014	32/m	L	V1	Neck pain, radiculopathy	−	39	−	Both	Stent (SA) + trapping	GR
23	Uneda et al. [29], 2016	35/f	R	V2	Radiculopathy	+	15	−	IVR	Internal trapping (coil)	GR
24	Lin et al. [30], 2017	18/f	L	V1	Neck swelling	+	ND	−	IVR	Distal trapping	GR
25	Abdulrazeq et al. [31], 2019	30/f	R	V1	Hypotension	+	ND	−	IVR	Internal trapping (coil)	GR
26	Han [32], 2019	36/m	L	V2	Chest discomfort	+	40	−	IVR	Internal trapping (coil)	MD
	Present case, 2020	46/m	R	V1	Infarction	−	30	+	IVR	Internal trapping (coil)	GR

Abbreviations: DS, direct surgery; GR, good recovery; IVR, interventional radiology; MD, moderate disability; ND, not described; SD, severe disability; VA, vertebral artery.

need to consider the pathological changes in the arterial wall of NF1. The pathological changes in EVA of NF1 described in the literature was a fragility of arterial wall due to following factors: (1) Proliferation of neurofibromatosis in the arterial wall, (2) dysplasia of mesoderm components, (3) proliferation of spindle-shaped cells in the endometrium, (4) mechanical effects of scoliosis on vessels [33–35]. Miyazaki et al. seemed that the fragility of arterial vessels gradually increases, and eventually the blood vessels are destroyed, resulting in the formation of aneurysms [7]. Negoro et al. hypothesized that direct surgery for NF1 associated aneurysm is fragile because of its fragility of the arterial wall, which could make it difficult to control intraoperative bleeding [12]. Based on these considerations, we chose endovascular treatment instead of direct surgical treatment in this case.

In our review, proximal occlusion was performed using a detachable balloon in the first three cases, 73% (19/26) cases were performed endovascular treatment, and internal coil trapping mainly was performed in the other cases. We considered using a stent to ensure blood flow in the right vertebral artery, but this strategy was impossible for the following reasons: there were no compatible stents, no proximal landing zone, many blood clots, and abnormal vessel walls. Stents were thought to cause further damage to the endometrium due to fragility of arterial vessels [22].

In our case, we performed internal trapping with neuroendovascular treatment of the aneurysm including its distal normal vertebral artery in order to preserve complication such as intraoperative embolism. It seemed that internal trapping was the best treatment strategy. There was concern that this procedure to occlude the right vertebral artery would increase the blood flow load to the left vertebral artery and cause aneurysms and other vascular injuries. Nothing has been reported about the occurrence of contralateral vascular lesions after unilateral VA occlusion. We should continue to follow up on images in the future.

4 | Conclusions

We experienced a rare case of cerebral infarction because of thrombosed extracranial vertebral artery aneurysm associated with NF1. And it was successfully treated with endovascular coil embolization. Although the vascular vulnerability of NF1 is a concern, endovascular therapy might be one treatment option for NF1 vascular lesions.

Author Contributions

Kenshi Sano: conceptualization, writing – original draft. **Atsushi Kuge:** conceptualization, writing – review and editing. **Rei Kondo:** methodology, resources, supervision. **Tetsu Yamaki:** methodology, resources. **Kazuki Nakamura:** data curation, methodology. **Shinjiro Saito:** supervision. **Yukihiko Sonoda:** conceptualization, supervision.

Ethics Statement

Patient confidentiality and informed consent were strictly adhered to throughout the documentation and publication of this medical case report, ensuring respect for the patient's autonomy and privacy rights.

Consent

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The authors have nothing to report.

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