

Renal and Intracranial Vasculopathy in an Adolescent with Neurofibromatosis Type 1: A Case Report

Emily Patron Chi^{*1,2}, Veronica Leon Burgos²

¹Faculty of Medicine. Autonomous University of Yucatan, Mérida, Yucatán, México.

²Department of Pediatrics, Yucatán Health Services, “Dr. Agustín O’Horán” General Hospital, Mérida, Yucatán, Mexico.

ABSTRACT

BACKGROUND: Neurofibromatosis type 1 (NF1) is an autosomal dominant multisystem disorder characterized by variable clinical expression. Although cutaneous manifestations are the most recognizable features, NF1 may be associated with clinically significant vascular abnormalities involving multiple arterial territories. We report an adolescent with NF1 and concomitant renal and intracranial vasculopathy.

CASE PRESENTATION: A 15-year-old girl with no relevant family history presented with a 4-day history of severe frontal headache, vomiting, generalized weakness, and left eyelid ptosis. Physical examination revealed multiple café-au-lait macules and axillary and inguinal freckling, fulfilling the revised 2021 diagnostic criteria for NF1. During hospitalization, persistent hypertension was documented, with blood pressure values up to 180/90 mmHg. Complete blood count, liver function tests, serum electrolytes, and renal function remained within age- and sex-appropriate reference ranges. Renal angiography demonstrated approximately 80% proximal stenosis of the left renal artery followed by aneurysmal dilatation measuring 4 × 8 mm. The patient subsequently developed acute neurologic deterioration with intraventricular hemorrhage and ventriculomegaly. Cerebral angiography revealed a 13 × 15 mm saccular aneurysm of the left posterior communicating artery associated with complete ipsilateral third cranial nerve palsy. Coil embolization of the intracranial aneurysm and balloon angioplasty of the left renal artery were successfully performed, resulting in complete angiographic occlusion of the aneurysm and improved blood pressure control, although antihypertensive therapy remained necessary.

CONCLUSION: This case highlights the potential for NF1-associated vasculopathy to affect multiple arterial territories simultaneously. Difficult-to-control hypertension and new focal neurologic findings in patients with NF1 should prompt targeted vascular evaluation. Early recognition and multidisciplinary management are essential to identify potentially severe renal and intracranial vascular complications.

KEYWORDS: Neurofibromatosis type 1; NF1; vasculopathy; renovascular hypertension; renal artery aneurysm; intracranial aneurysm; posterior communicating artery; third cranial nerve palsy.

ARTICLE DETAILS

Published On:
28 September 2026

Available on:
<https://ijmscrs.com>

I. INTRODUCTION

Neurofibromatosis type 1 (NF1) is an autosomal dominant genetic disorder characterized by nearly complete penetrance and variable clinical expressivity. It is caused by heterozygous pathogenic variants in the NF1 gene, located on chromosome 17q11.2, which encodes the protein

neurofibromin. Approximately 50% of cases result from de novo pathogenic variants, whereas the remaining cases are inherited from an affected parent. NF1 has an estimated incidence of approximately 1 in 3,000 individuals and represents the most common form of neurofibromatosis. [1,2]

Table I. Comparison of the NIH Diagnostic Clinical Criteria for NF1 from 1988 with the Newly Revised NF1 Diagnostic Criteria According to Legius 2021)

Revised diagnostic criteria for NF1 according to Legius et al. (2021)	NF1 diagnostic criteria according to the National Institutes of Health Consensus Development Conference (1988)
A- The diagnostic criteria for NF1 are met in an individual who does not have a parent diagnosed with NF1 and if two or more of the following features are present	An individual meets the diagnostic criteria for NF1 if two or more of the following features are present:
• ≥ 6 café-au-lait macules >5 mm in greatest diameter in prepubertal individuals and >15 mm in post pubertal individuals • Freckling in the axillary or inguinal region • ≥ 2 neurofibromas of any type or one plexiform neurofibroma • Optic pathway glioma • ≥ 2 Lisch nodules identified by slit-lamp examination or ≥ 2 choroidal anomalies defined as bright patchy nodules imaged by optical coherence tomography (OCT)/ near-infrared reflectance (NIR) imaging • A distinctive osseous lesion such as sphenoid wing dysplasia anterolateral bowing of the tibia or pseudoarthrosis of a long bone • A heterozygous pathogenic NF1 gene variant with a variant allele frequency of 50% in apparently normal tissue such as white blood cells	• ≥ 6 café-au-lait macules >5 mm in greatest diameter in prepubertal individuals and >15 mm in post pubertal individuals • Axillary or inguinal freckles (Crowe's sign) • ≥ 2 neurofibromas of any type or one or more plexiform neurofibroma • Optic glioma • ≥ 2 Lisch nodules • A distinctive osseous lesion such as sphenoid wing dysplasia or thinning of the cortex of the long bones (with or without pseudoarthrosis)
B- A child of a parent who meets the diagnostic criteria specified in A meets the diagnosis of NF1 if one or more of the criteria in A are present.	A first degree relative NF1 (parent, sibling or offspring) diagnosed with NF1 by the above criteria

Source: Adapted from Legius et al. [5] and Kehrer-Sawatzki and Cooper [2]

NF1 is a multisystemic disorder with a phenotype characterized by substantial clinical variability and age-dependent manifestations. The most common features include café-au-lait macules and axillary or inguinal freckling, followed by cutaneous or subcutaneous neurofibromas. Plexiform neurofibromas, optic pathway gliomas, Lisch nodules, osseous abnormalities, and neurological and neurodevelopmental manifestations may also occur. In addition, patients with NF1 have an increased risk of developing certain neoplasms, particularly malignant peripheral nerve sheath tumors and gliomas, among others. [1,2]

In the Mexican population, NF1 is one of the most frequently identified neurocutaneous syndromes. In a Mexican series of patients with neurocutaneous syndromes, NF1 accounted for 62% of cases, followed by tuberous sclerosis complex at 18%, highlighting the importance of recognizing its clinical manifestations in pediatric practice. [3]

Although cutaneous manifestations are the most recognizable features of the disease, NF1 may be associated with potentially severe vascular complications. NF1-associated vasculopathy can affect vessels of different

calibers and manifest as arterial stenosis, aneurysms, vascular ectasia, aortic disease, or cerebrovascular disease. Hypertension is a relatively common complication and, particularly in children and adolescents, may be associated with renal artery stenosis or middle aortic syndrome. Abnormalities of the cerebral arteries, including stenosis, ectasia, and intracranial aneurysms, have also been described. [1,4]

The diagnosis of NF1 is primarily clinical. The criteria established by the National Institutes of Health (NIH) in 1988 were revised in 2021 through an international consensus (Table I), incorporating new ophthalmologic findings and the identification of pathogenic variants in the NF1 gene. In an individual without a parent diagnosed with NF1, the diagnosis can be established when two or more criteria are present, including six or more café-au-lait macules measuring more than 5 mm in diameter before puberty or more than 15 mm after puberty; axillary or inguinal freckling; two or more neurofibromas or one plexiform neurofibroma; an optic pathway glioma; two or more Lisch nodules or characteristic choroidal abnormalities; a distinctive osseous lesion; or a heterozygous pathogenic variant in NF1. Therefore, molecular testing is not essential for establishing the

Renal and Intracranial Vasculopathy in an Adolescent with Neurofibromatosis Type 1: A Case Report

diagnosis in patients who fulfill sufficient clinical criteria. [5]

The presence of hypertension in a patient with NF1 should prompt targeted evaluation to exclude renovascular disease, particularly renal artery stenosis or middle aortic syndrome. Similarly, the development of atypical headache, seizures, or focal neurological deficits warrants targeted neurological evaluation and neuroimaging, as cerebrovascular complications may result in significant morbidity. [4,6]

We present the case of an adolescent with a clinical diagnosis of NF1 who developed multisystemic arterial vasculopathy, manifested by stenosis and aneurysmal dilatation of the left renal artery associated with renovascular hypertension, as well as an intracranial aneurysm of the left posterior communicating artery accompanied by ipsilateral third cranial nerve palsy and intraventricular hemorrhage. Both vascular lesions were treated with endovascular intervention.

II. CASE REPORT

admitted to the Pediatric Emergency Department with a 4-day history of severe frontal headache, rated 10/10 on the visual analog scale, associated with four episodes of vomiting. She subsequently developed generalized muscle weakness, gait disturbance, and left eyelid ptosis.

Upon admission, her blood pressure was 155/80 mmHg, elevated for age. She was afebrile and oriented but had difficulty walking. Physical examination revealed multiple café-au-lait macules, six of which were clearly demarcated, located on the left forearm, abdomen, chest, left iliac crest, right thigh, and anterior aspect of the right leg, measuring approximately 3 cm, 3 cm, 3.5 × 1 cm, 6 × 4 cm, 3 × 2 cm, and 12 × 6 cm, respectively. Axillary and inguinal freckling was also observed. (Figure 1)

Neurological examination revealed anisocoria, with a normally reactive right pupil and a dilated left pupil measuring approximately 4 mm, with absent pupillary light response. Left eyelid ptosis was observed, with inability to spontaneously open the eye. Right extraocular movements were preserved. These findings were subsequently



Figure 1. Cutaneous manifestations of neurofibromatosis type 1. (A, B) Café-au-lait macules involving the upper limb. (C) Axillary freckling.

A previously healthy 15-year-old female patient, with no relevant family history related to the current condition, was

confirmed by ophthalmological evaluation as complete left third cranial nerve palsy.

Renal and Intracranial Vasculopathy in an Adolescent with Neurofibromatosis Type 1: A Case Report

During the initial evaluation, a cranial computed tomography (CT) scan was performed, with no evidence of acute structural lesions. In the context of multiple café-au-lait macules and axillary and inguinal freckling, a clinical diagnosis of neurofibromatosis type 1 (NF1) was established according to the international diagnostic criteria revised in 2021. The patient fulfilled two diagnostic criteria: six or more café-au-lait macules measuring more than 15 mm after puberty and axillary or inguinal freckling.

During hospitalization, the patient developed persistently elevated and difficult-to-control blood pressure, with measurements reaching up to 180/90 mmHg, requiring antihypertensive treatment. She initially received telmisartan/hydrochlorothiazide, followed by serial adjustments to her antihypertensive regimen. Throughout hospitalization, complete blood count, liver function tests, serum electrolytes, and renal function parameters remained

within age- and sex-appropriate reference ranges, with no relevant biochemical abnormalities. Initial serum creatinine was 0.3 mg/dL.

Subsequently, she experienced an episode of sudden neurological deterioration characterized by severe headache followed by transient decreased level of consciousness and an inadequate response to stimuli. During the event, capillary blood glucose was 157 mg/dL and blood pressure was 151/85 mmHg. Following the episode, she experienced a postictal recovery period of approximately 1 minute, with subsequent recovery of consciousness. Given the clinical suspicion of a seizure, levetiracetam was initiated.

Cranial CT performed during this episode revealed intraventricular hemorrhage, radiologically reported as modified Fisher grade IV. Follow-up CT demonstrated a decrease in the hemorrhage and cerebral edema, associated with ventricular dilatation consistent with

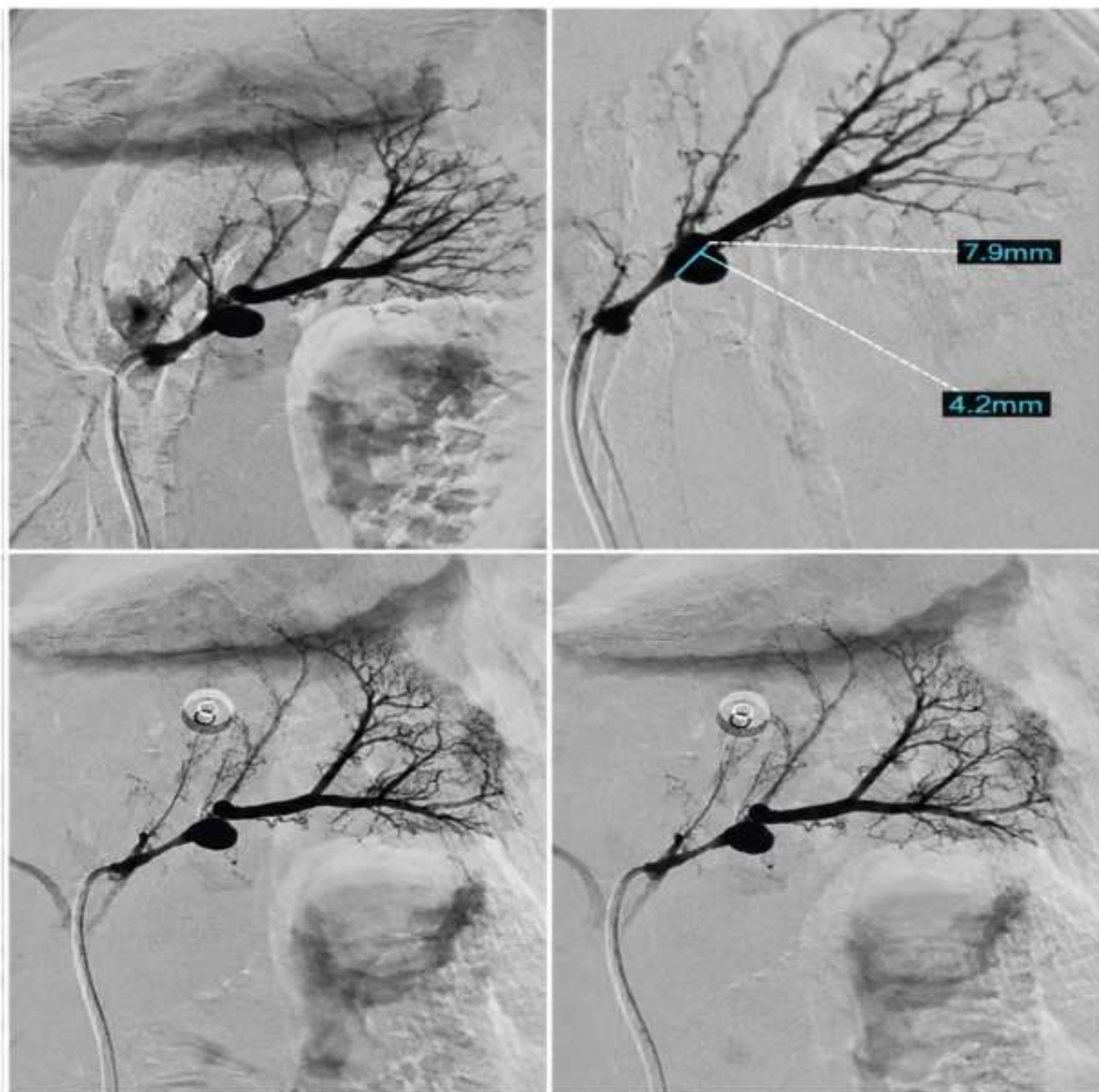


Figure 2. Renal angiography demonstrating aneurysmal dilatation of the left renal artery.

ventriculomegaly/hydrocephalus. Urgent neurosurgical

Renal and Intracranial Vasculopathy in an Adolescent with Neurofibromatosis Type 1: A Case Report

evaluation was obtained, and conservative management was recommended.

Because of persistent, difficult-to-control hypertension and the suspicion of a renovascular etiology, renal ultrasonography was performed and showed no evident structural abnormalities. Subsequently, abdominal CT identified a finding suggestive of a left renal artery aneurysm. Subsequent renal angiography demonstrated proximal stenosis of the left renal artery, with approximately 80% luminal narrowing, followed by aneurysmal dilatation measuring approximately 4×8 mm. In this context, the findings were considered consistent with NF1-associated renovascular disease related to the patient's hypertension. (Figure 2)

As part of the neurological evaluation, brain magnetic resonance imaging (MRI) demonstrated ventriculomegaly, with an Evans index of 3.1, as well as periventricular white matter hyperintensity, predominantly involving the frontal horns, suggestive of transependymal edema. Cerebral vascular imaging identified a lesion consistent with an intracranial aneurysm involving the left carotid circulation.

On September 29, 2025, cerebral panangiography was performed, identifying a saccular aneurysmal dilatation measuring approximately 13×15 mm, with a 3.2-mm neck, directed posteroinferiorly relative to the parent vessel, corresponding to a left posterior communicating artery aneurysm arising from the left internal carotid artery. Endovascular treatment was performed by coil embolization, achieving complete angiographic occlusion of the aneurysm without immediate complications.

During the same period, left renal artery angioplasty was performed using a 3.5×20 mm noncompliant balloon, inflated to 20 atm, achieving adequate restoration of renal artery patency. The procedural report did not document stent placement.

Following endovascular treatment, improvement in blood pressure control was observed, although antihypertensive therapy remained necessary. After the neuroendovascular procedure, she received a tirofiban infusion, followed by transition to antiplatelet therapy with aspirin and clopidogrel. Clopidogrel was administered for only 24 hours. At discharge, aspirin was prescribed for 1 month, along with prazosin for blood pressure control. Levetiracetam was continued.

Ophthalmological evaluation confirmed complete left third cranial nerve palsy, with persistent ipsilateral ptosis and anisocoria. Cardiac evaluation by echocardiography demonstrated mild aortic regurgitation and left ventricular hypertrophy with an eccentric pattern, with preserved biventricular systolic and diastolic function and normal pulmonary artery pressure, without evidence of cardiovascular aneurysms. Electrocardiography showed no abnormalities.

At discharge, the patient was hemodynamically stable, with a blood pressure of 100/70 mmHg, without further episodes

suggestive of seizures or evidence of acute neurological deterioration. Left eyelid ptosis and anisocoria persisted, with partial improvement in ptosis compared with the initial presentation.

Multidisciplinary outpatient follow-up was arranged with Neurology, Neurosurgery, Nephrology, Cardiology, Genetics, Rehabilitation, and Pediatrics, in addition to follow-up cerebral angiography and electroencephalography.

III. DISCUSSION

Neurofibromatosis type 1 (NF1) is a multisystemic disorder in which vascular involvement represents an uncommon but potentially serious complication. NF1-associated vasculopathy may involve small-, medium-, and large-caliber arteries and can manifest as stenosis, occlusion, aneurysms, ectasia, dissection, moyamoya disease, and other vascular abnormalities. The overall frequency of vasculopathy reported in different series ranges from 0.4% to 6.4%. These figures should be interpreted with caution, as they are derived from heterogeneous cohorts, and the absence of systematic vascular imaging may result in underdiagnosis. A review published in 2026 specifically highlighted the heterogeneity of the available evidence and the lack of standardized protocols for the diagnosis and surveillance of neurovascular complications in NF1. [4,7]

The present case is notable for the coexistence of clinically significant renal and intracranial vascular involvement in an adolescent with NF1. The patient presented with difficult-to-control hypertension, and approximately 80% proximal stenosis of the left renal artery was identified, followed by aneurysmal dilatation, in addition to an intracranial aneurysm of the left posterior communicating artery. The presence of vascular lesions in different arterial territories supports a multisystemic manifestation of NF1-associated vasculopathy, although it does not necessarily imply that all lesions share an identical histopathological mechanism. [7,13]

Hypertension is one of the most relevant cardiovascular complications of NF1. In children and adolescents, the identification of hypertension should prompt a targeted evaluation for secondary causes, particularly renal artery stenosis and abdominal aortic disease. In our patient, persistently elevated blood pressure readings, reaching up to 180/90 mmHg, with inadequate control despite pharmacological treatment, prompted investigation for a renovascular etiology. Renal angiography demonstrated significant proximal stenosis of the left renal artery and aneurysmal dilatation distal to the lesion, providing an anatomical substrate for the diagnosis of renovascular hypertension. [4,6]

Renovascular disease associated with NF1 is well described in the pediatric population. A case involving a 17-year-old adolescent with NF1 and severe hypertension demonstrated approximately 70%–80% stenosis of the left renal artery,

Renal and Intracranial Vasculopathy in an Adolescent with Neurofibromatosis Type 1: A Case Report

with post-stenotic dilatation and a small pseudoaneurysm; the lesion subsequently progressed and required angioplasty with stent placement, resulting in sustained blood pressure control. Similarly, a retrospective series of pediatric renal angioplasty included seven patients with NF1 and showed that five of the seven experienced resolution or improvement of hypertension following the intervention, although the clinical benefit was lower than the overall technical success rate. These findings support the use of revascularization in selected patients with symptomatic renovascular disease, while also demonstrating that clinical outcomes do not always depend exclusively on angiographic correction of the stenosis. [8,9]

Persistent hypertension after technically successful renal angioplasty has been specifically described in NF1. Ueda et al. reported an adolescent with right renal artery stenosis who showed normalization of renin activity after angioplasty but persistent hypertension for more than six months. The authors suggested that arterial stiffness associated with NF1 vasculopathy might contribute to persistent hypertension. Our patient exhibited a similar course: blood pressure control improved following renal angioplasty, but prazosin was still required at discharge. This finding should be interpreted as being consistent with the known clinical course of NF1-associated renovascular disease rather than as evidence of procedural failure. [10]

The presence of aneurysmal dilatation associated with renal artery stenosis represents an additional finding of interest. Renal artery aneurysms are uncommon in NF1, and the available evidence is based primarily on isolated case reports. In a nine-year-old pediatric patient with NF1, multiple aneurysms involving branches of the renal artery were identified; one of these was treated with coil embolization. The authors emphasized that identification of a vascular abnormality in one vascular territory should prompt consideration of vascular involvement in other territories. Another report published in 2023 described a fusiform aneurysm of the left renal artery in a 30-year-old woman with NF1 and hypertension, which was treated with placement of a covered stent, followed by improved blood pressure control. These cases demonstrate that endovascular strategies may be useful in selected patients, although treatment selection depends on the anatomy, location, and extent of the lesion. [11,12]

Cerebrovascular disease represents another important component of NF1-associated vasculopathy. In a national cohort of 2,068 adults with NF1, 1,234 had undergone brain imaging, and 43 (3.5%) had a vascular abnormality. Most patients with vascular abnormalities were symptomatic. Among the 10 patients with aneurysms, the most common location was the internal carotid artery, followed by other vascular territories, including the posterior communicating artery. Of particular relevance to our case, one patient presented with painful third cranial nerve palsy associated with an aneurysm of the posterior communicating artery.

The anatomical and clinical similarity to our patient is notable. [13]

In our patient, the aneurysm was located in the left posterior communicating artery, arising from the left internal carotid artery, with approximate dimensions of 13 × 15 mm and a 3.2-mm neck. The lesion was associated with left eyelid ptosis, mydriasis, and absence of the pupillary light reflex; these findings were subsequently confirmed as complete left third cranial nerve palsy. This anatomical-clinical correlation is consistent with the close relationship between the posterior communicating artery and the oculomotor nerve. Expansion of an aneurysm at this location may cause direct compression of the third cranial nerve, with pupillary involvement when its superficial parasympathetic fibers are affected. In this context, complete third cranial nerve palsy with a dilated pupil constitutes a warning sign warranting urgent neurovascular evaluation.

Recent literature also indicates that NF1 vasculopathy may be multifocal and dynamic. In the cohort reported by Sheerin et al., eight patients presented with more than one type of vascular abnormality; one had stenosis, ectasia, and an aneurysm, in addition to a history of renovascular disease. [13]

Likewise, patients with NF1 and multiple intracranial aneurysms or the development of new vascular lesions during follow-up have been described. These data do not establish a specific pattern of association between renal and intracranial aneurysms; however, they support consideration of NF1 as a potentially multisystemic vascular disorder. [7,13]

Regarding the diagnostic approach, this case highlights the importance of regular blood pressure measurement in patients with NF1 and of investigating secondary causes when hypertension is identified, particularly in children and adolescents. The recommendations of the American College of Medical Genetics and Genomics consider renovascular disease an important cause of hypertension in young patients with NF1. In contrast, the utility of systematic neurovascular screening in asymptomatic patients remains uncertain. The 2026 review indicates that no uniform protocols currently exist for screening for aneurysms and other cerebrovascular abnormalities. Therefore, our case should not be interpreted as evidence supporting universal cerebral angiography in all patients with NF1; rather, it emphasizes the need for targeted neurovascular evaluation in the presence of severe or atypical headache, seizures, altered mental status, focal neurological deficits, or cranial nerve palsy. [6,7]

The intraventricular hemorrhage observed during the clinical course represents another relevant finding but should be interpreted with caution. In our patient, computed tomography was reported as modified Fisher grade IV intraventricular hemorrhage, and subsequent imaging documented ventriculomegaly consistent with hydrocephalus. Although an intracranial aneurysm was subsequently identified, the available information does not

allow us to establish that the aneurysm was the direct source of the hemorrhage. Therefore, it is more appropriate to describe the coexistence of intraventricular hemorrhage and intracranial aneurysm without attributing causality. This distinction is particularly important because the Fisher grading system was developed to stratify subarachnoid hemorrhage and, by itself, cannot establish an aneurysmal origin for an intraventricular hemorrhage.

Endovascular treatment was used in our patient to address both vascular territories. Balloon angioplasty of the renal artery achieved adequate restoration of patency of the left renal artery and was accompanied by improved blood pressure control, although antihypertensive therapy remained necessary. Coil embolization of the posterior communicating artery aneurysm resulted in complete angiographic occlusion and was accompanied by partial neurological improvement, particularly in ptosis. Published experience in patients with NF1 demonstrates that endovascular interventions can be effective but also that there are risks of recurrence, dissection, pseudoaneurysm formation, and the need for additional procedures. A pediatric case published in 2025 described complications following renal angioplasty, including dissection, pseudoaneurysm formation, and persistent stenosis, whereas another pediatric case published in 2025 ultimately required renal autotransplantation after angioplasty failed. These reports reinforce the need for prolonged vascular follow-up after intervention. [14,15]

A pediatric case published in 2026 is particularly illustrative of NF1-associated renovascular disease. A three-year-old girl presented with hypertension secondary to significant right renal artery stenosis and was treated with percutaneous transluminal angioplasty. Subsequent Doppler ultrasonography demonstrated a transient vascular fistula that resolved spontaneously. Although the patient's age and vascular anatomy differed from those of our patient, this case demonstrates that NF1-associated renovascular disease may present early in life and that multimodal evaluation may be essential for characterizing both the primary lesion and post-procedural complications. [16]

We consider that the primary relevance of this case lies not solely in the presence of an intracranial aneurysm in a patient with NF1, but in the coexistence of clinically significant renal and intracranial vascular disease in an adolescent. The patient presented with renovascular hypertension associated with stenosis and aneurysmal dilatation of the left renal artery and, simultaneously, a left posterior communicating artery aneurysm associated with ipsilateral third cranial nerve palsy. The endovascular resolution of both lesions also provides an example of a multidisciplinary approach to a multisystemic vascular manifestation of NF1.

IV. CONCLUSIONS

This case illustrates a severe and multisystemic presentation of NF1-associated vasculopathy. The presence of difficult-to-control hypertension in a pediatric or adolescent patient with NF1 should prompt evaluation for renovascular disease, while the development of focal or warning neurological symptoms should lead to timely neurovascular assessment. The coexistence of arterial lesions in the renal and intracranial vascular territories highlights the potentially multisystemic nature of this vasculopathy and underscores the need for long-term multidisciplinary and vascular follow-up.

ACKNOWLEDGMENTS

We thank the patient and her family for their willingness to participate and for granting permission for the publication of this case. We also extend our gratitude to Hospital General Agustín O'Horán and to all healthcare professionals involved in her care.

CONFLICT OF INTEREST

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

ETHICS STATEMENT

Written informed consent was obtained from the patient's legal representative for the publication of clinical data and images. In addition, informed assent was obtained from the patient in accordance with institutional requirements. Written informed consent was obtained for the publication of any potentially identifiable images or data included in this article.

FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

REFERENCES

- I. Peduto C, Zanobio M, Nigro V. Neurofibromatosis type 1: pediatric aspects and review of genotype–phenotype correlations. *Cancers (Basel)*. 2023;15(4):1217. <https://doi.org/10.3390/cancers15041217>
- II. Kehrer-Sawatzki H, Cooper DN. Challenges in the diagnosis of neurofibromatosis type 1 (NF1) in young children facilitated by means of revised diagnostic criteria including genetic testing for pathogenic NF1 gene variants. *Hum Genet*. 2022;141(2):177-191. <https://doi.org/10.1007/s00439-021-02410-z>
- III. Hernández-Castañeda Y, Juárez-Melchor D, Vera-Loaiza A, Pérez-Arzola AA, Crisanto-López IE, Rodríguez-Hurtado PO. Caracterización clínica de pacientes con síndrome neurocutáneo. *Rev Med*

Renal and Intracranial Vasculopathy in an Adolescent with Neurofibromatosis Type 1: A Case Report

- Inst Mex Seguro Soc.*2025;63(2):e6419.
<https://doi.org/10.5281/zenodo.14616948>
- IV. Friedman JM. Neurofibromatosis 1. 1998 Oct 2 [updated 2025 Apr 3]. In: Adam MP, Bick S, Mirzaa GM, Pagon RA, Wallace SE, Amemiya A, editors. GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993-2026. <https://www.ncbi.nlm.nih.gov/books/NBK1109/>
 - V. Legius E, Messiaen L, Wolkenstein P, Pancza P, Avery RA, Berman Y, et al. Revised diagnostic criteria for neurofibromatosis type 1 and Legius syndrome: an international consensus recommendation. *Genet Med.*2021;23(8):1506-1513. <https://doi.org/10.1038/s41436-021-01170-5>
 - VI. Stewart DR, Korf BR, Nathanson KL, Stevenson DA, Yohay K. Care of adults with neurofibromatosis type 1: a clinical practice resource of the American College of Medical Genetics and Genomics (ACMG). *Genet Med.*2018;20:671-682. <https://doi.org/10.1038/gim.2018.28>
 - VII. Zedde M, Pascarella R. Neurovascular issues in neurofibromatosis type I: focus on intracranial stenosis. *Life (Basel).*2026;16(2):234. <https://doi.org/10.3390/life16020234>
 - VIII. Beladan CC, Geavlete OD, Botezatu S, Postu M, Popescu BA, Ginghina C, et al. Revascularization in a 17-year-old girl with neurofibromatosis and severe hypertension caused by renal artery stenosis. *Tex Heart Inst J.* 2017;44(1):50-54. <https://doi.org/10.14503/THIJ-15-5466>
 - IX. Srinivasan A, Krishnamurthy G, Fontalvo-Herazo L, Nijs E, Keller MS, Meyers K, Kaplan B, Cahill AM. Angioplasty for renal artery stenosis in pediatric patients: an 11-year retrospective experience. *J Vasc Interv Radiol.* 2010;21(11):1672-1680. <https://doi.org/10.1016/j.jvir.2010.07.012>
 - X. Ueda K, Awazu M, Konishi Y, Takenouchi T, Shimozato S, Kosaki K, Takahashi T. Persistent hypertension despite successful dilation of a stenotic renal artery in a boy with neurofibromatosis type 1. *Am J Med Genet A.* 2013;161A(5):1154-1157. <https://doi.org/10.1002/ajmg.a.35829>
 - XI. Chillura I, Restivo GA, Callari S, Cibella S, D'Alessandro MM, Corrado C, Vallone M, Antona V, Corsello G. A novel NF1 mutation in a pediatric patient with renal artery aneurysm. *Ital J Pediatr.* 2022;48(1):186. <https://doi.org/10.1186/s13052-022-01382-8>
 - XII. AlSheikh S. Renal artery aneurysm induced by neurofibromatosis type 1: a case report and review of the endovascular interventions for this rare vasculopathy. *Medicine (Baltimore).* 2023;102(27):e34216. <https://doi.org/10.1097/MD.00000000000034216>
 - XIII. Sheerin UM, Holmes P, London NF1 Research Group, Childs L, Roy A, Ferner RE. Neurovascular complications in adults with neurofibromatosis type 1: a national referral center experience. *Am J Med Genet A.* 2022;188(10):3009-3015. <https://doi.org/10.1002/ajmg.a.62931>
 - XIV. Wala SJ, Beebe M, Warren P, Kallash M, Nathan J, Rasmussen S. Early experience with renal autotransplant for renal artery stenosis in a 6-year-old patient with neurofibromatosis type 1: a case report. *J Med Case Rep.* 2025;19(1):196. <https://doi.org/10.1186/s13256-025-05208-z>
 - XV. Dang QL, Fairman AS. Endovascular salvage of renal artery bypass in pediatric a patient with neurofibromatosis type 1. *J Vasc Surg Cases Innov Tech.* 2025;11(1):101644. <https://doi.org/10.1016/j.jvscit.2024.101644>
 - XVI. Pytlos J, Majcher P, Skrzypczyk P, Maciąg R, Werner B, Furmanek M. A complex case of renal artery stenosis in a 3-year-old patient with neurofibromatosis type 1 and secondary hypertension. *Diagnostics (Basel).* 2026;16(7):1047. <https://doi.org/10.3390/diagnostics16071047>