CASE REPORT

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Rupture of a Basilar Aneurysm Secondary to Wyburn-Mason Syndrome: A Case Report

Nelson Antonio Milanés-González[1], Jose Enrique Velázquez-Amador[2], Luis Alejandro Carrillo-Santillán[3], Maria Pamela Contla-Armengol[1], Eder Fernando Ríos-Bracamontes[4]

[1]Department of Research, Universidad de Colima Faculty of Medicine, General Hospital No. 1, Mexican Social Security Institute, Colima, Mexico
[2]Department of Research, Universidad Autónoma de Guadalajara Faculty of Medicine, General Hospital No. 1, Mexican Social Security Institute, Zapopan, Mexico
[3]Department of Internal Medicine, Universidad de Colima Faculty of Medicine, General Hospital No. 1, Mexican Social Security Institute, Colima, Mexico
[4]Clinic of Internal Medicine, General Hospital No. 1, Mexican Social Security Institute, Colima, Mexico

Abstract

Wyburn-Mason syndrome (WMS) is an extremely rare, non-hereditary congenital disorder characterized by arteriovenous malformations (AVMs) that primarily affect the retina and central nervous system and, less commonly, other structures. Its clinical presentation is highly variable, and management is often challenging due to the high risk of morbidity and mortality, particularly in deep lesions that are not amenable to surgical, endovascular, or radiosurgical treatment. We present the case of a young woman with relevant neurologic and ophthalmologic history who developed a massive subarachnoid hemorrhage secondary to rupture of a basilar artery aneurysm associated with a prepontine AVM, a complication that is poorly documented in the literature. The clinical course and fatal outcome are described. This case highlights the severe neurological complications associated with WMS and underscores the importance of early recognition and long-term monitoring in patients with high-risk vascular malformations.

Keywords: Wyburn-Mason syndrome, arteriovenous malformations, subarachnoid hemorrhage, basilar aneurysm, cerebrovascular malformations, neurocutaneous syndromes

INTRODUCTION

Wyburn-Mason Syndrome (WMS), also known as Bonnet-Dechaume-Blanc Syndrome, is a rare non-hereditary neurocutaneous disorder classified among the phakomatoses. It results from abnormal embryological development of the primitive vascular mesoderm shared by the optic vesicle and anterior neural tube, leading to the formation of arteriovenous malformations (AVMs).[1]

These malformations may involve the retina, optic pathway, and central nervous system, resulting in potentially severe ophthalmologic and neurologic complications.

Approximately 30% of patients with retinal involvement also present intracranial AVMs, whereas only 8% of patients with cerebral AVMs demonstrate retinal involvement.[2] Fewer than 100 cases have been reported worldwide over the past six decades, most presenting with

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ORCID IDs of the authors: N.A.M.G. 0009-0009-1170-5966; J.E.V.A. 0009-0006-7373-5181; L.A.C.S. 0009-0009-0801-0629; M.P.C.A. 0009-0007-6250-1758; E.F.R.B. 0000-0002-4660-7372.

Corresponding author: Nelson Antonio Milanés-González
E-mail: nelsonantonio971@gmail.com
ORCID ID: orcid.org/0009-0009-1170-5966

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ophthalmologic manifestations and only a minority presenting with severe neurological complications.

Cerebrofacial arteriovenous metameric syndromes (CAMS) represent a group of congenital vascular disorders characterized by AVMs affecting embryologically related regions of the brain and face. WMS is typically classified as CAMS type II due to the involvement of the optic nerve, optic chiasm, diencephalon, and retina. However, recent literature suggests it may present with overlapping characteristics of other CAMS subtypes.[3,4]

Clinical manifestations depend on the anatomical location and size of the vascular malformations. Ophthalmologic findings may include retinal racemose hemangioma, decreased visual acuity, afferent pupillary defects, and proptosis.[5-7] Neurological manifestations include intracranial hemorrhage, seizures, headache, and progressive neurological deficits.[1] Among these, subarachnoid hemorrhage (SAH) secondary to aneurysm rupture represents one of the most severe and life-threatening complications.

CASE REPORT

A 27-year-old woman presented to a private hospital with progressive loss of consciousness, dysarthria, vomiting, and two generalized tonic-clonic seizures following an episode of intense emotional stress. Her condition was accompanied by quadriparesis, rapid neurological deterioration, pallor, and diaphoresis.

Her medical history included congenital aniridia and left ocular proptosis, which ultimately required enucleation during the neonatal period, as well as epilepsy diagnosed at six years of age.

She had a prepontine AVM associated with WMS, diagnosed 20 years earlier by cerebral angiography, and classified as unsuitable for surgical, endovascular, or radiosurgical treatment because of its anatomical location. Although the original angiographic images were unavailable, computed tomography angiography performed one year earlier confirmed the vascular malformation (Figure 1).

Neuroimaging revealed Fisher grade IV SAH with intraventricular extension and acute hydrocephalus (Figure 2).[8] Imaging findings were consistent with rupture of a basilar artery aneurysm associated with the prepontine AVM, which was determined to be the primary source of the hemorrhage.

The patient developed severe neurological deterioration requiring endotracheal intubation, deep sedation, and ventriculoperitoneal shunt placement. She was transferred to a secondary-level public hospital for comprehensive management.

During her ICU stay, she developed severe hypernatremia, acute renal failure with anuria, hemodynamic instability requiring vasopressors, and respiratory failure requiring mechanical ventilation.

Despite aggressive supportive care, her condition progressed to refractory bradycardia and ultimately to asystole. Advanced cardiopulmonary resuscitation was unsuccessful, and death was declared.

Patient consent was provided by a family member due to the patient's neurological state, it was signed on the 7th of January, 2026 and kept by the main author, a copy has also been provided to the publisher.

DISCUSSION

In this case, the prepontine AVM represented a significant therapeutic challenge due to its deep location and complex vascular anatomy, rendering surgical or endovascular treatment not feasible.[9] Although embolization and radiosurgery are therapeutic alternatives, not all

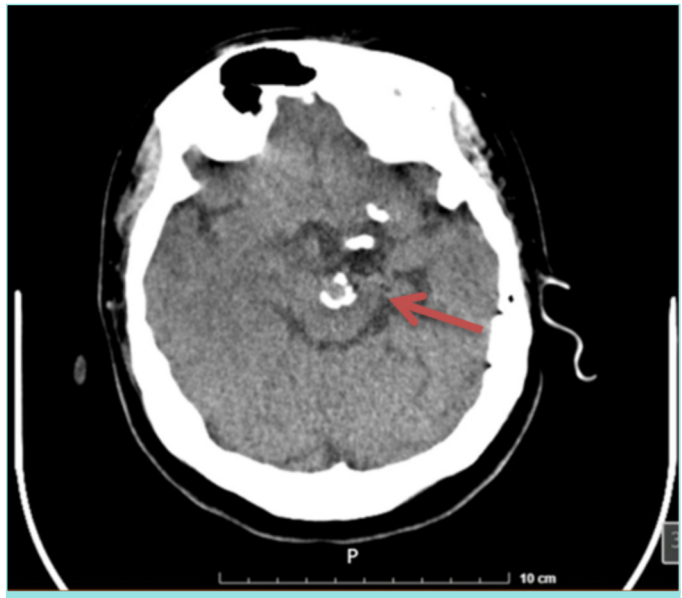

Figure 1. Brain computed tomography angiography demonstrating a prepontine and suprasellar arteriovenous malformation characterized by dilated serpiginous vascular structures arising from the basilar artery (red arrow), with associated calcifications and a nidus measuring 27×25 mm.

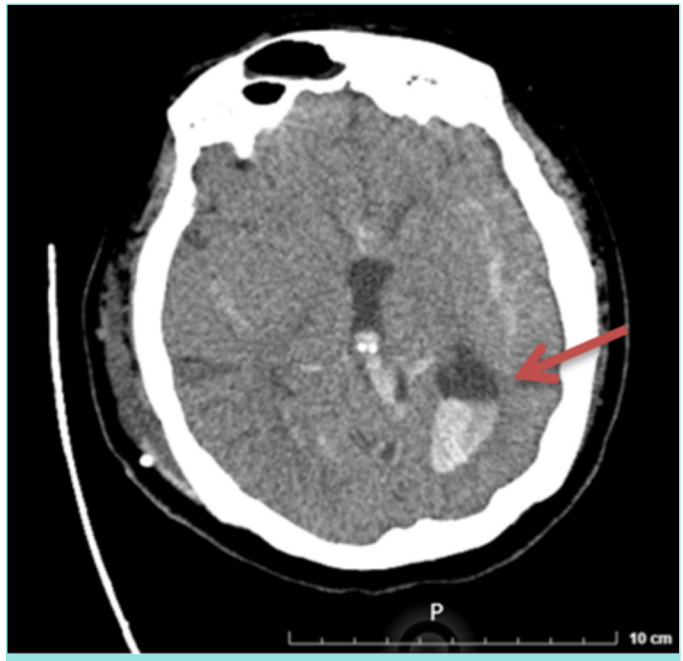

Figure 2. Non-contrast brain computed tomography demonstrates extensive subarachnoid hemorrhage with intraventricular extension (red arrow), ventriculomegaly, and erosion of the left clivus, suggestive of chronic structural changes.

AVMs are amenable to these interventions due to their size, anatomical location, or associated risk of neurological injury. SAH accounts for approximately 3% of all strokes but is associated with disproportionately high mortality and morbidity.[10,11]

The development of aneurysmal rupture associated with WMS represents a rare but devastating complication. Hemodynamic stress and abnormal vascular architecture likely contribute to aneurysm formation and rupture.

This case highlights the importance of long-term monitoring of patients with WMS, as vascular lesions may remain clinically stable for years before catastrophic deterioration.

CONCLUSION

This case report describes a rare, fatal complication of WMS: rupture of a basilar aneurysm associated with a prepontine AVM. WMS remains a poorly understood condition due to its rarity. Early recognition and long-term follow-up are essential to identify high-risk vascular malformations and prevent catastrophic neurological events. Further research is needed to better understand the natural history and optimal management of this condition.

MAIN POINTS

- Wyburn-Mason syndrome (WMS) can cause arteriovenous malformations (AVMs) primarily affecting the retina and central nervous system.
- Sometimes the complexity of their location makes this disease inoperable, turning it into a major therapeutic challenge.
- These AVMs can be asymptomatic throughout much of a patient's life and may present with aneurysmal rupture.
- As far as is known, WMS is a disease with a poor prognosis, and the medical community has not yet established a management protocol, making it a promising area for future research.

ETHICS

Informed Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Footnotes

Authorship Contributions

Surgical and Medical Practices: N.A.M.G., J.E.V.A., M.P.C.A., Concept: N.A.M.G., L.A.C.S., E.F.R.B., Design: N.A.M.G., J.E.V.A., L.A.C.S., M.P.C.A., E.F.R.B., Data Collection and/or Processing: J.E.V.A., L.A.C.S., M.P.C.A., Analysis and/or Interpretation: N.A.M.G., E.F.R.B., Literature Search: J.E.V.A., L.A.C.S., M.P.C.A., Writing: N.A.M.G., E.F.R.B.

DISCLOSURES

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