CASE REPORT

Wyburn-Mason Syndrome with Retinal and Orbital Arteriovenous Malformations: A Diagnostic Challenge and Clinical Course

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ABSTRACT

Introduction: Wyburn-Mason Syndrome (WMS), also known as racemose angioma, is a rare congenital, nonhereditary neurocutaneous disorder characterized by arteriovenous malformations (AVMs). The syndrome exhibits heterogeneous clinical manifestations and predominantly involves the retina, orbit, and brain. This report presents a case of Wyburn-Mason Syndrome with retinal and orbital involvement and long-term follow-up.

Case illustration: A 19-year-old woman presented with a long-standing history of progressive visual impairment in the left eye since childhood, which worsened following menarche. Ophthalmic examination revealed no light perception in the left eye, unilateral proptosis, and tortuous, dilated retinal vessels extending from the optic disc to the peripheral retina, consistent with retinal arteriovenous malformation. Imaging studies demonstrated an intraconal orbital arteriovenous malformation without evidence of intracranial involvement. The diagnosis of Wyburn-Mason Syndrome was established based on retinal and orbital AVMs.

Discussion: Visual loss in this patient was likely caused by mechanical compression of the optic nerve by the orbital arteriovenous malformation and retinal ischemia associated with extensive retinal vascular involvement. Interventional treatment options were considered; however, management decisions were guided by the absence of acute neurological symptoms and the non-progressive course observed during follow-up.

Conclusion: This case highlights the variable clinical course of Wyburn-Mason Syndrome and underscores the importance of comprehensive evaluation, individualized management, and careful long-term monitoring to guide appropriate clinical decision-making.

Keywords: Wyburn-Mason syndrome; arteriovenous malformation; retinal arteriovenous malformation; orbital vascular malformation

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INTRODUCTION

Wyburn-Mason Syndrome is a rare non-hereditary congenital neurocutaneous condition characterized by vascular lesions known as arteriovenous malformations (AVMs) in the retina, brain, and/or skin[1]. Wyburn-Mason Syndrome also known as Bonnet-Bechaume-Blanc syndrome, congenital unilateral retinocephalic vascular malformation syndrome, racemose angiomatosis[2]. Wyburn-Mason syndrome is frequently included in the phakomatoses syndromes, which result in tumors or masses in the brain, spinal cord, and other organs.

Wyburn-Mason Syndrome's clinical presentation varies greatly, depending mostly on the location and size of the AVMs, with symptoms ranging from vision impairment to neurological symptoms such as stroke and focal neurological impairments[1]. The ophthalmoscopic appearance of the AVM in Wyburn-Mason Syndrome is characterized by dilated and tortuous vessels that span the optic disc and extend to the retinal periphery. Presenting vision might range from normal to no light perception, depending on the ophthalmic problems[3].

Wyburn-Mason Syndrome is extremely rare, with fewer than 100 cases documented in the literature. There is no distinguishable gender or ethnic preference. It is estimated that 30% of patients with retinal findings have brain abnormalities, whereas 8% of patients with brain findings have retinal findings[4]. The aim of this case is to report a case of Wyburn-Mason Syndrome in adolescent girl.

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Case presentation

A 19-years old girl came with a complaint of blurred vision in the left eye. The patient complained of blurry vision in the left eye since 11 years ago, which worsened seven years ago. The patient has been unable to see light since a year ago. The patient has been experiencing symptoms since her first menstrual period. She felt that the left eye was more prominent than the right one.



Figure 1. (A) Front view, exotropia of the left eye is noticeable. (B) Left view. (C) Worm's eye view, proptosis of the left eye is noticeable. ***Pictures taken with patient's consent. Courtesy: Outpatient Eye Clinic RSUD Dr. Soetomo.***

There were no complaints of red eyes, eye discharge, or throbbing eye pain. There are no tumor on other body parts or organ deformities. The patient had no complaints of nausea or vomiting, headaches, seizures, or limb weakness. The patient had no complaints about bloody urine, brown urine, lower back pain, vomiting blood, bleeding gums, nosebleeds, or any body bleeding. The patient has no previous history of illness. There is no family with a history of the same as the patient. There is no previous history of wearing glasses. There were no medical problems during the pregnancy, birth, or growth process. The patient has no history of trauma.

The visual acuity of the right eye is 5/5 and the left eye is no light perception. Intraocular pressure measurements in both eyes were within normal limits of 11 mmHg and 14 mmHg. Examination with a Hertel exophthalmometer revealed 17-110-22. It is concluded that there is proptosis of the left eye. There are limitations on left eye ocular motility examination, including -1 to nasal, superonasal, and inferonasal as seen in **figure 2**. There is a 15 degree exotropia in the left eye. The left eye pupil measures 5 mm in diameter, positive light reflex, and shows positive RAPD as shows in **figure 3**. Funduscopic examination on the left eye shows tortuous dilated retinal vessels over the optic disc and extends to the retinal periphery in accordance with retinal arteriovenous malformation (AVM) as seen in **figure 4**. The anterior segment and fundus of the right eye were within normal limits.

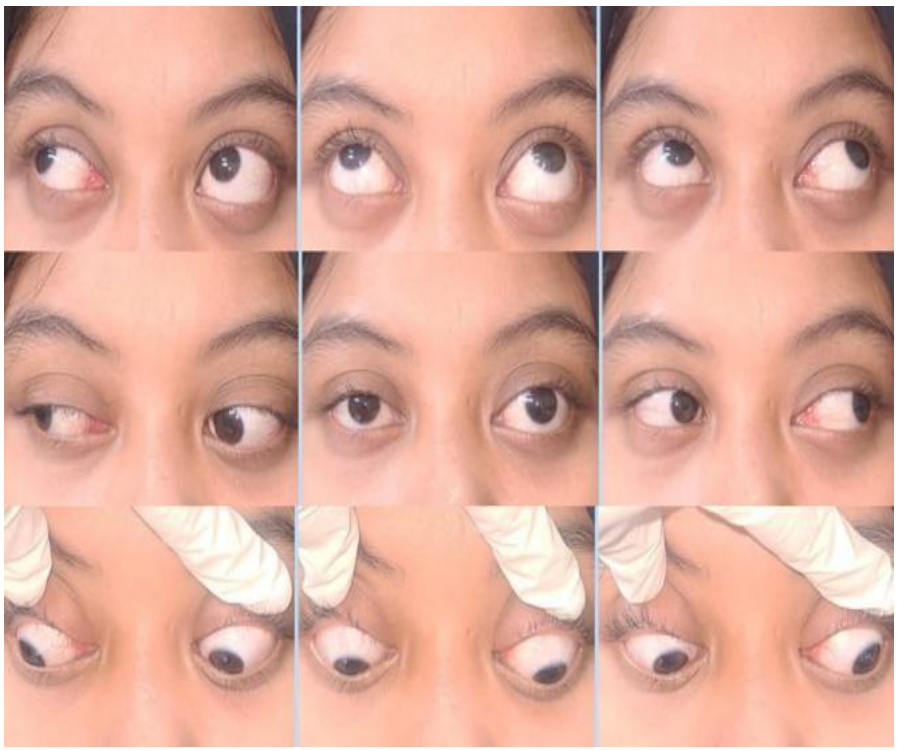


Figure 2. Limitations on left eye ocular motility examination: -1 to nasal, superonasal, and inferonasal. ***Pictures taken with patient's consent. Courtesy: Outpatient Eye Clinic RSUD Dr. Soetomo.***

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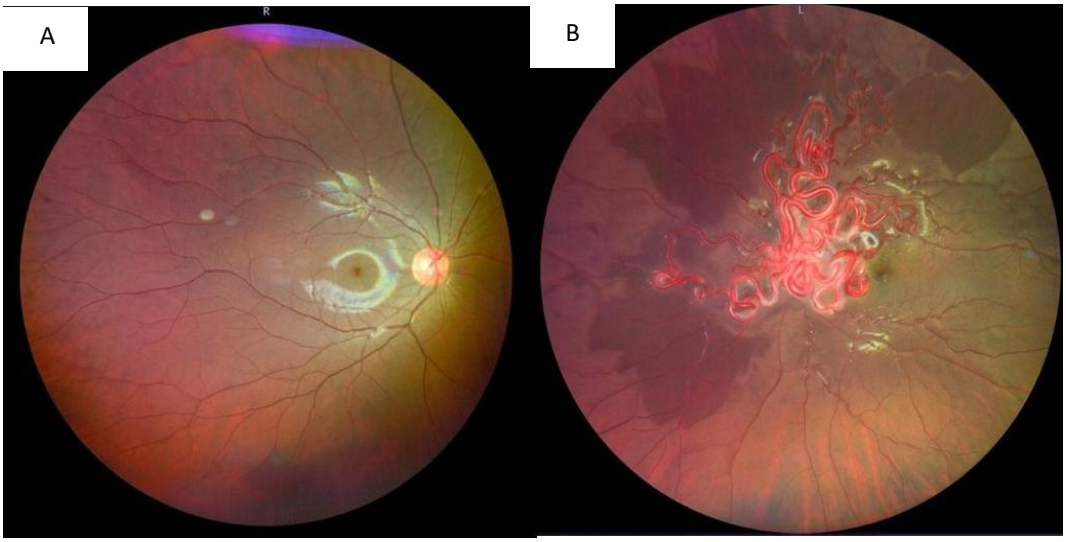


Figure 3. (A) Anterior segment of the right eye, within normal limits. (B) Anterior segment of the left eye, pupil anisocoria, 5 mm in diameter, positive light reflex, and positive RAPD. (C, D) Deep anterior chamber in both eyes. ***Pictures taken with patient's consent. Courtesy: Outpatient Eye Clinic RSUD Dr. Soetomo***

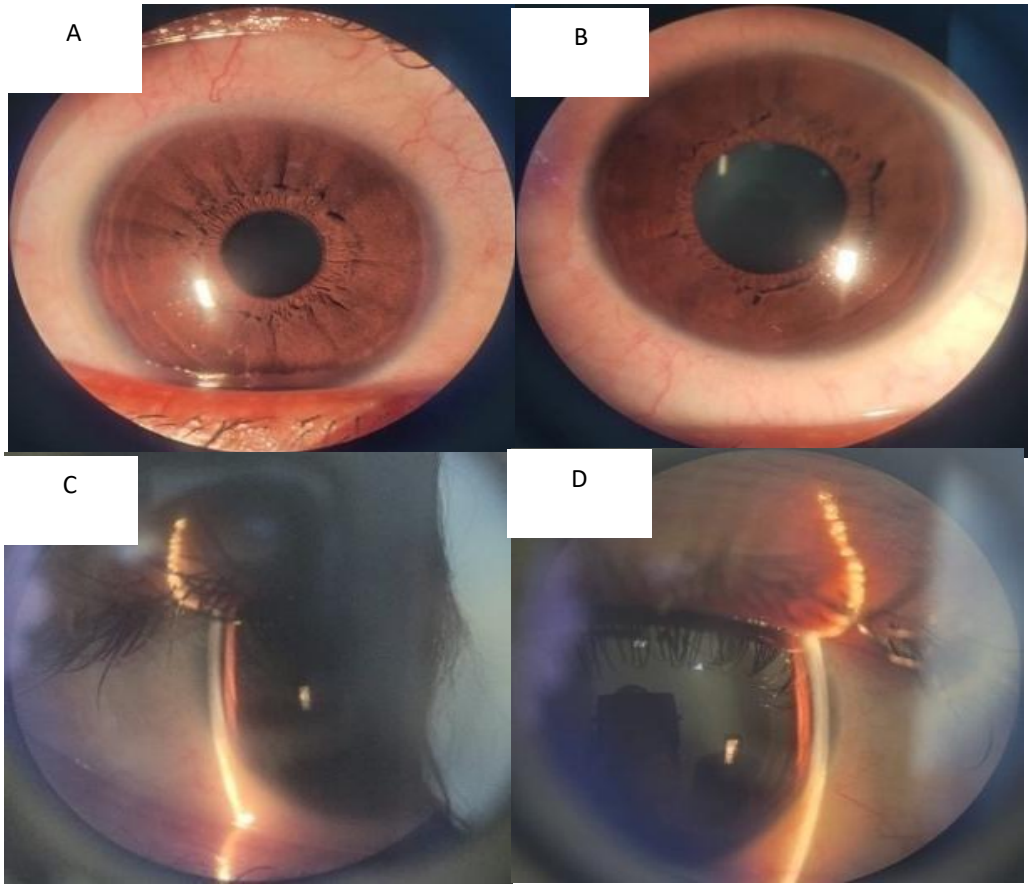
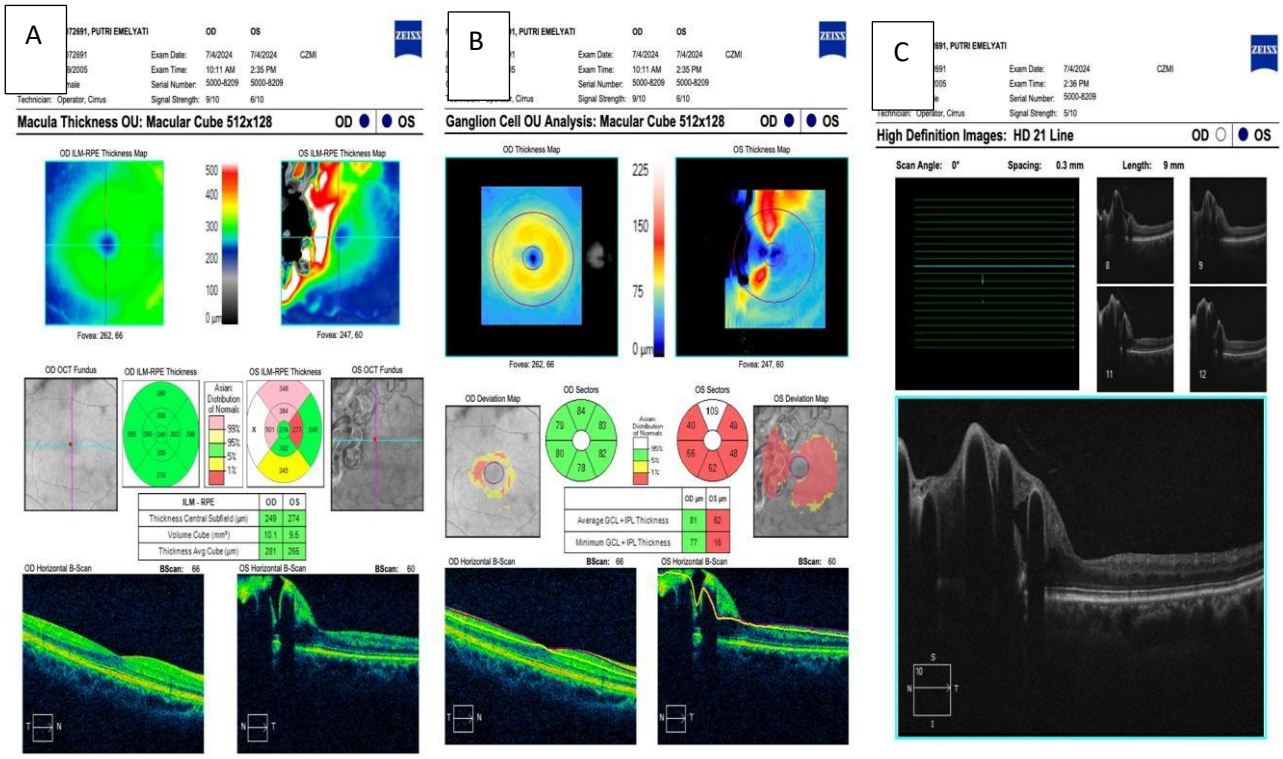


Figure 4. (A) Fundus of the right eye, within normal limit. (B) Fundus of the left eye, tortuous dilated retinal vessels over the optic disc and extends to the retinal periphery. ***Pictures taken with patient's consent. Courtesy: Outpatient Eye Clinic RSUD Dr. Soetomo***

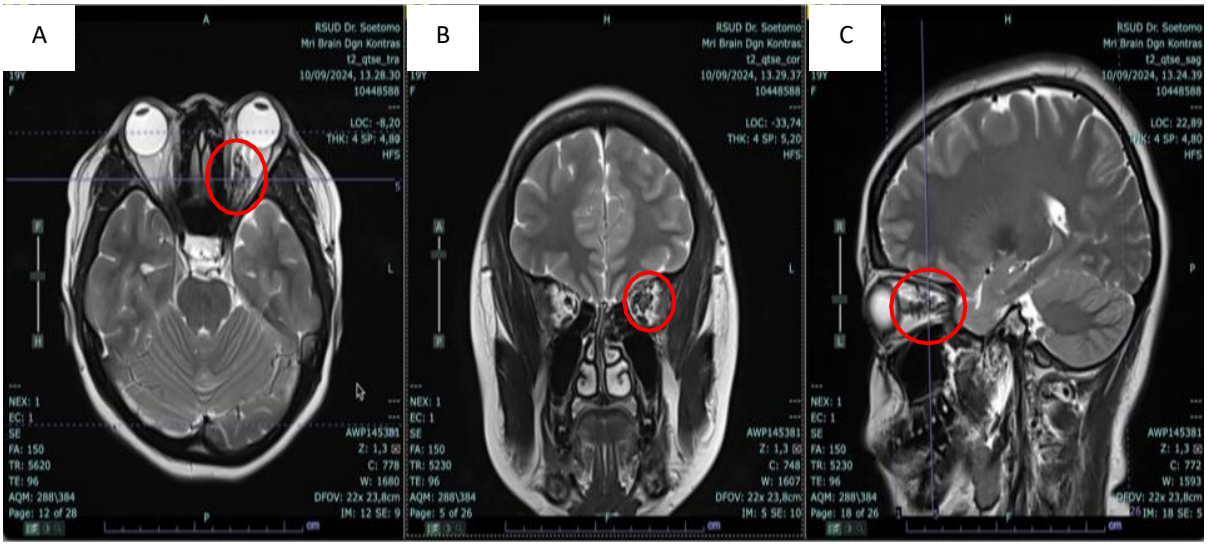
The patient underwent optical coherence tomography (OCT) examination of the right and left eyes. Macular OCT revealed no macular edema in both eyes. The left eye showed a decrease in macular thickness and macular ganglion cells. OCT HD21 on the left eye revealed multiple large inner retinal vessels create a prominent shadowing artifact. The retina is diffusely thickened. The left eye's optic nerve papillae was difficult to evaluate during an Optic Disc OCT examination because it was covered by tortuous dilated retinal vessels.



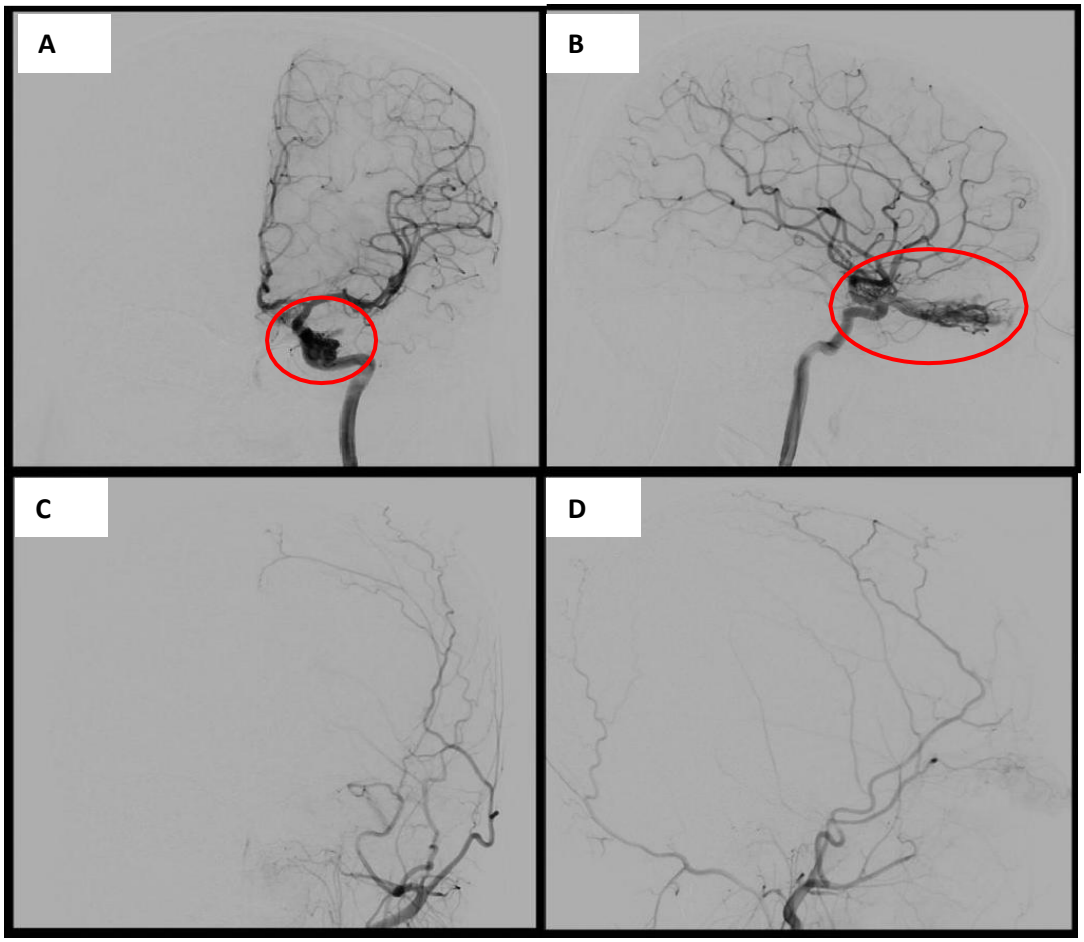
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Figure 5. (A) Macular thickness OCT, left eye's macular thickness was decrased (B) Macular ganglion cell OCT, left eye's macular ganglion cell was decreased C) Left eye's HD21, multiple large inner retinal vessels create a prominent shadowing artifact and no macular edema, retina is diffusely thickened. ***Pictures taken with patient's consent. Courtesy: Outpatient Eye Clinic RSUD Dr. Soetomo***

The patient /underwent contrast-enhanced MRI and MRA head examinations. From the MRI examination, there was a tubular lesion in the left intraconal region that is attached to and surrounds the left optic nerve measures +/- 2.7 x 1.1 x 1.0 cm and is isointense on T1WI, hypointense on T2WI, unrestricted diffusion area on DWI, and tubular contrast enhancement is visible on contrast administration. The lesion appears to emerge from the left optic canal and spread to the left cavernous sinus. The angioma appears to be receiving arterial feeding from the left ophthalmic artery branch. According to the MRA, the Circle of Willis is patent, and the left ophthalmic artery appears to feed the angioma in the left orbital cavity.



. Figure 6. Contrast-enhanced MRI-MRA of the brain (A) Axial (B) Coronal (C) Sagittal. There is a intraconal angioma of the left eye. ***Pictures taken with patient's consent. Courtesy: Radiology RSUD Dr. Soetomo***



.Figure 7. Transfemoral Cerebral Angiography (A) ICA antero-posterior (B) ICA lateral (C) ECA antero-posterior (D) ECA lateral. An AVM measuring 2.9 cm x 1 x 1 cm, feeding artery from the left ophthalmic artery with a small feeder from the distal internal maxillary artery, with a drainer to the left jugular vein. ***Pictures taken with patient's consent. Courtesy: Outpatient Neurosurgery Clinic RSUD Dr. Soetomo***

We diagnosed the patient with retinal arteriovenous malformation and intraconal angioma. We consulted the patient to the neurosurgery department for treatment. The patient underwent a Transfemoral Cerebral Angiography (TFA) examination. The patient underwent a TFA examination before the procedure, the left internal carotid artery (ICA) angiogram revealed an AVM measuring 2.9 cm x 1 x 1 cm, feeding artery from the left ophthalmic artery with a small feeder from the distal internal maxillary artery, with a drainer to the left jugular vein. Left vertebral artery angiogram showed no vascular malformations or aneurysms. Because of the dangerous anastomosis, embolization cannot be performed.

Given the absence of acute neurological or ocular emergency manifestations, the long-standing nature of the visual impairment, and the indolent clinical course of the lesion, an initial conservative approach was considered appropriate. Therefore, active intervention was deferred, and the patient was managed with close clinical observation. Regular follow-up was recommended to monitor for potential progression, with instructions to seek earlier evaluation should new symptoms or clinical deterioration arise.

At one-year follow-up, the patient reported no new ocular or neurological complaints. The left eye remained without light perception, with no ocular pain. On ophthalmic examination, visual acuity in the left eye remained no light perception. Intraocular pressure in the left eye was 12 mmHg. Hertel exophthalmometry revealed measurements of 18–110–21, indicating persistent but stable left-sided proptosis. The left eye remained exotropic.

Pupillary examination of the left eye revealed a round pupil measuring 5 mm in diameter, with preserved direct and consensual light reflexes and a persistent relative afferent pupillary defect. Funduscopic examination of the left eye demonstrated a clear fundus reflex; however, the optic disc margin remained obscured by overlying vascular structures. Marked retinal vascular tortuosity and vascular enlargement persisted, consistent with retinal arteriovenous malformation. No retinal hemorrhage or exudation was observed.

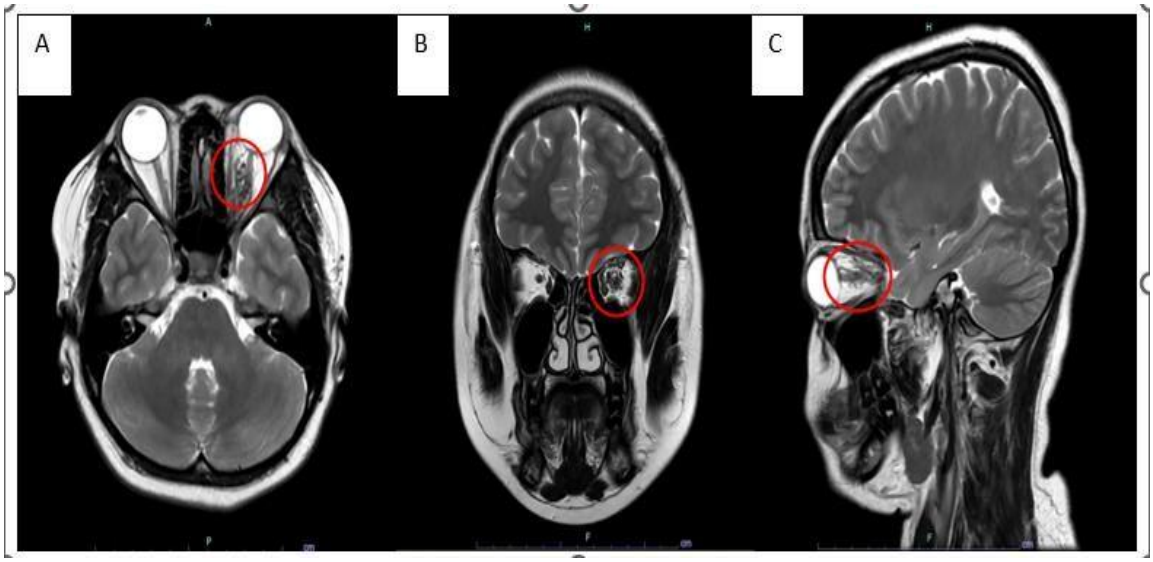


Figure 8. Contrast-enhanced MRI of the brain and orbit at one-year follow-up. (A) Axial, (B) Coronal, and (C) Sagittal views demonstrate a persistent intraconal angioma of the left orbit, with lesion size comparable to the previous MRI examination and no significant interval progression. ***Pictures taken with patient's consent. Courtesy: Radiology RSUD Dr. Soetomo***

Magnetic resonance imaging (MRI) of the brain and orbit demonstrated a persistent tubular vascular lesion in the left retrobulbar intraconal region, encasing the left optic nerve. The lesion measured approximately 2.2 × 1.0 × 0.8 cm, representing a reduction in size compared to the previous examination (previously 2.7 × 1.1 × 1.0 cm). The lesion appeared isointense on T1-weighted images, hypointense on T2-weighted images, demonstrated unrestricted diffusion on diffusion-weighted imaging, and showed tubular contrast enhancement following contrast administration. The lesion remained confined to the intraconal orbital space without extraorbital extension and continued to receive arterial supply from a branch of the left ophthalmic artery.

Based on these findings, the patient was assessed as having a stable left retrobulbar vascular lesion with interval size reduction and no evidence of disease progression. Conservative management with continued observation was recommended, with regular follow-up every 3–4 months or earlier if new symptoms developed**.**

DISCUSSION

We diagnosed the patient in this case report with retinal arteriovenous malformation and intraconal angioma. Retinal AVMs are congenital, nonhereditary, and sporadic vascular anomalies that develop in the posterior pole. Archer et al subdivided these anomalies into different groups, depending on their severity [1,5]. Group 1 is defined as retinal arteriovenous communication with an arteriolar or aberrant capillary plexus. Group 2 has one or more direct arteriovenous communications without the presence of capillary or arteriolar elements. Group 3 has extensive and complex arteriovenous communication with large caliber vessels and without the interposition of a capillary plexus. To diagnose Wyburn Mason Syndrome, group 3 AVMs and concomitant intracranial AVMs must be present[1]. Group 3 retinal AVMs usually have large caliber anomalous vessels with poor visual acuity and eyes may be blind from birth [1,6]. This patient had large caliber anomalous vessels in his left eye, no light perception visual acuity, and complains of blurring since childhood, which indicated a group 3 retinal AVM.

The differential diagnosis of retinal arteriovenous malformation includes Wyburn Mason Syndrome, Sturge Weber syndrome, Von Hippel Lindau disease, vasoproliferative tumors (VPT), retinal cavernous hemangioma, Rendu-Osler-Weber Disease (Hereditary hemorrhagic telangiectasis), Familial Retinal Arteriolar Tortuosity (FRAT) [3,7]. Sturge-Weber syndrome should be suspected if there are port wine stains (facial angioma) and/or ipsilateral juvenile or congenital open-angle glaucoma [3,7]. In this patient, there were no reddish birthmark on the face, so this diagnosis can be excluded. Von Hippel-Lindau (VHL) disease is linked to retinal capillary hemangioma (RCH), cerebral aneurysms, renal cell carcinoma, and pheochromocytoma. VHL is an autosomal dominant. RCH presents clinically as a circular, circumscribed, orange to red retinal lesion in the peripapillary or peripheral region [7]. Laboratory examination of kidney function in this patient was within normal limits, no complaints of hematuria, no family history, and no circular retinal lesions, so this diagnosis can be excluded. Vasoproliferative tumors (VPT) are distinguished by retinal nodules made up of glial cells interlaced with a fine capillary network and dilated hyalinised blood vessels. VPT can be primary or subsequent to retinal disorders such retinitis pigmentosa, coat's disease, and post-vitreoretina surgery [8]. There were no retinal nodules or other retinal abnormalities discovered in this patient, so the diagnosis is ruled out. Retinal cavernous hemangioma is characterized by a dark grape-like cluster of venous intraretinal

aneurysms [7]. This diagnosis can be ruled out because the AVM in this patient is tortuous dilated retinal vessels. Rendu-Osler-Weber syndrome can be diagnosed with three of the following criteria: telangiectasia on the face, hands and oral cavity; recurrent epistaxis; arteriovenous malformations with visceral involvement; family history [9]. This patient has no family history, no red spots on the face and fingers, no nosebleeds, no gastrointestinal complaints, so the diagnosis can be ruled out. Familial retinal arteriolar tortuosity (FRAT) is an inherited disorder characterized by increased tortuosity of the second and higher order retinal arterioles in the posterior pole. The first-order arteries and venous system are usually not affected [10]. This patient has tortuous dilated first-order retinal vessels over the optic disc and no family history, thereby ruling out this diagnosis. Based on the explanation above, this patient's diagnosis suggests Wyburn Mason Syndrome.

Intraorbital AVMs are rare, slowly progressive congenital lesions, and their management represents a challenge for multidisciplinary teams. Unilateral exophthalmos can be caused by both intraorbital and extraorbital vascular malformations. The differential diagnosis includes anterior cranial fossa dural fistulas, cirsoid aneurysms of the scalp, traumatic carotid-cavernous fistulas, and congenital vascular malformations like Wyburn-Mason Syndrome. A congested orbit produces mechanical restriction of ocular motility without cranial nerve dysfunction. However, the presence of a dilated pupil would suggest a third cranial nerve palsy. Arteriography is essential for confirming the diagnosis and defining the arterial supply and venous drainage pathways [26]. Our patient had an intraconal angioma in the left eye, resulting in unilateral proptosis. Aside from that, ocular motility problems were discovered, as well as pupil anisocoria (5 mm in diameter) and positive RAPD. MRA and TFA revealed that the feeding artery was from the left ophthalmic artery, while the drainer to the left jugular vein.

Wyburn Mason Syndrome (WMS) is an exceedingly rare, non-hereditary, congenital neurocutaneous disorder characterized by AVMs in the retina, brain, and occasionally the skin [1,3]. The majority of AVMs described in WMS are unilateral [11]. Because cutaneous signs are uncommon, the disease is typically diagnosed later in childhood [3]. The exact etiology of Wyburn-Mason syndrome is currently unknown. Sporadic abnormalities in the formation of blood vessels during embryonic or fetal growth are hypothesized to be the cause of WMS [3]. The disease has no racial or gender preference, and females and males are affected equally [1,3]. AVMs in the retina grow slowly, although pregnancy, menarche, and trauma might accelerate their progression [12]. WMS happened unilaterally in this patient, causing blurry complaints that had been noticed from childhood (11 years ago) and worsened since 7 years ago, when the patient had menarche. Currently, the left eye has no light perception. This is consistent with the hypothesis that WMS is a congenital AVM condition that progresses as menarche occurs. In this patient there is no cutaneous symptoms.

Wyburn Mason Syndrome is diagnosed through a clinical examination. Ophthalmoscopy is typically used to identify retinal arteriovenous malformations, while fluorescein angiography may be required for minor lesions. The ophthalmoscopic appearance of the AVM in WMS typically appear as irregular, tortuous, and dilated vessels, with minimal observable difference between arteries and veins. The AVM range in size but often tend to be large. The current eyesight may range from normal to no light perception, depending on the associated ophthalmic complications. Retinal AVMs typically do not cause symptoms, but vision loss can result from ocular complications including, intraretinal or vitreous hemorrhage, exudation, retinal artery occlusion, retinal vein occlusion, mechanical compression of the optic nerve, or neovascular glaucoma [1,3,6,13]. This patient displays AVM characteristics similar to those described for WMS, which is massive tortuous and dilated vessels over the optic disc and spreading to the retinal periphery. Mechanical compression of the optic nerve might cause the patient's complete loss of vision.

Ocular findings of Wyburn Mason Syndrome included decreased visual acuity or total blindness, proptosis (occasionally pulsatcile), blepharoptosis, ocular nerve palsies, nystagmus, strabismus, ocular motility disorder, abnormally dilated vessels of the conjunctiva, rubeosis iridis, secondary glaucoma (neovascular glaucoma), pupillary defects, vitreous hemorrhage, retinal detachment, vein occlusions, optic disc edema, optic atrophy, and visual field defects [2,14,15]. In this patient there is total blindness, proptosis, exotropia, impaired ocular motility, positive RAPD in the left eye.

Presentation of patients with cerebral lesions varied according to the site and extent of the lesion. The orbit was the most usually affected site, followed by the thalamus, hypothalamus, optic chiasm, and suprasellar region. Many cerebral AVMs remain asymptomatic throughout the life of the patient. Neurological symptoms of WMS include severe headaches, vomiting, seizures, paralysis (from cranial nerves), nuchal rigidity, epistaxis, subarachnoid or intracerebral hemorrhage, hydrocephalus, hemiparesis, or hemiplegia [14,15,2]. Cutaneous lesions, such as angiomas, can affect the facial skin in the trigeminal nerve distribution. These lesions can range from severe to mild, erythematous discolouration [11,15,16]. This patient has no neurological or cutaneous symptoms.

Optical coherence tomography (OCT) is a valuable tool for confirming the diagnosis and monitoring structural changes of the retina and optic nerve in Wyburn-Mason Syndrome, particularly for detecting macular edema and serous retinal detachment [3]. Previous reports have described OCT findings in WMS as round intraretinal structures with prominent shadowing artifacts, diffuse retinal thickening, and heterogeneous inner retinal layers caused by dilated anomalous vessels [17,18]. Multimodal imaging, including fluorescein angiography and OCT angiography, further demonstrates arterialization of retinal veins with minimal leakage and limited exudation [19]. Enhanced depth imaging OCT has also been shown to reveal choroidal thickening

and enlargement of luminal areas, suggesting choroidal vascular dilatation in affected eyes [20]. In the present case, OCT demonstrated multiple large inner retinal vessels with marked shadowing artifacts and diffuse retinal thickening without macular edema, while evaluation of the optic disc was limited due to overlying tortuous dilated retinal vessels. Intracranial arteriovenous malformations can be identified using computed tomography (CT), magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), or cerebral arteriography. MR imaging and CT are helpful in delineating the extent and the precise location of vascular lesions affecting the brain and orbit, and their relationship to neighboring structures. Catheter angiography can show the size, position, and features of the feeding arteries and draining veins, and it is thought to be the best method for determining the exact angioarchitecture of the lesion [3,6,14]. From the Contrast-enhanced MRI-MRA examination of the brain, an intraconal angioma of the left eye was found. The angioma appears to be receiving arterial feeding from the left ophthalmic artery branch. The patient underwent a Transfemoral Cerebral Angiography (TFA) examination. The TFA examination revealed an angioma measuring 2.9 cm x 1 x 1 cm, feeding artery from the left ophthalmic artery with a small feeder from the distal internal maxillary artery, with a drainer to the left jugular vein.

The management of Wyburn-Mason Syndrome is primarily determined by the location of the arteriovenous malformations and the presence or absence of related symptoms [3]. In asymptomatic or minimally symptomatic patients, conservative management with close clinical and radiological observation is generally recommended, given the typically slow-growing nature of these vascular lesions [1,3]. Surgical intervention may be considered for selected intracranial arteriovenous malformations, particularly in cases complicated by hemorrhage or progressive neurological deficits; however, surgical access to deep-seated lesions carries a substantial risk of neurological morbidity [3,14].

Alternative treatment modalities for arteriovenous malformations that are not amenable to surgical resection include endovascular embolization and radiation-based therapies [21]. Endovascular embolization is often preferred due to its less invasive nature and lower complication rates compared with open surgery, and it may be performed to reduce intraoperative bleeding, control symptomatic vessels, or achieve definitive treatment when feasible [22,23]. Radiation therapy, including stereotactic radiosurgery such as Gamma Knife or CyberKnife, has also been reported as a therapeutic option for orbital and intracranial arteriovenous malformations [21,24]. However, outcomes following radiation therapy are variable, and visual deterioration after treatment has been described, particularly in lesions involving critical orbital structures [24]. In the present case, endovascular embolization was contraindicated due to the presence of dangerous anastomoses identified on transfemoral cerebral angiography, which posed a high risk of ischemic complications [3,21]. Although referral for stereotactic radiosurgery was initially considered, the absence of acute neurological symptoms, the long-standing and non-progressive nature of the patient's visual impairment, and the indolent course of the lesion supported a conservative management strategy [1,3,14]. This approach is consistent with previously reported cases of Wyburn-Mason Syndrome in which patients without progressive neurological symptoms or lesion enlargement were managed conservatively with careful observation [1,3].

Furthermore, follow-up imaging in this patient demonstrated interval stability and slight reduction in lesion size, reinforcing the appropriateness of continued observation rather than immediate intervention [3,14]. Similar reports have emphasized that not all orbital or intracranial arteriovenous malformations associated with Wyburn-Mason Syndrome require active treatment, particularly in the absence of hemorrhage, progressive neurological deficits, or lesion growth [1,3,21]. Therefore, careful long-term monitoring with a multidisciplinary approach remains a reasonable and safe management strategy in selected patients [3,14].

CONCLUSION

Wyburn-Mason Syndrome is a rare congenital vascular disorder characterized by arteriovenous malformations involving the retina and orbit, with or without intracranial involvement. This case illustrates the heterogeneous clinical presentation and variable disease course of Wyburn-Mason Syndrome. Comprehensive clinical and radiological evaluation is essential to establish the diagnosis and assess the extent of involvement. During follow-up, the lesion in this patient remained stable without evidence of progression, highlighting the importance of individualized management and careful long-term monitoring. A multidisciplinary approach is crucial to support appropriate decision-making and timely intervention should clinical or radiological changes occur.

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