

Ga-68 DOTATATE PET/CT in a 19-Year-Old Male with Left Optic Nerve Sheath Meningioma: Diagnostic Utility in Differentiating Inflammatory Perineuritis

Winsome Chan, MD, SCHP, FRACGP^{1,2*}, Karan Bir Singh, MBBS SCHP FRACP FAANMS¹, Mahtab Ghadiri, BMedSc MBBS (Hons) FRACP PhD³, Ramy Nour, MBBS, FRACP¹

¹Department of Nuclear Medicine, St George Hospital, Kogarah, NSW, Australia

²Faculty of Medicine & Health, University of New South Wales, Kensington, NSW, Australia

³Department of Neurology, St George Hospital, Kogarah, NSW, Australia

*Correspondence: Winsome Chan, Department of Nuclear Medicine, St George Hospital, Gray St, Kogarah, NSW 2217, Australia, Tel: 02 9113 1111

 winsomekakiu.chan@health.nsw.gov.au

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AUTHOR CONTRIBUTIONS

- Dr Winsome Chan was involved in reviewing the patient prior to imaging, summarising the clinical case, drafting the manuscript, writing the discussion, the question-and-answer section and tables, as well as preparing the images included in this report.
- Dr Karan Bir Singh reviewed the draft manuscript and provided critical input, including advice on imaging interpretation, publication strategy, and techniques for publishing imaging-related case reports.
- Dr Mahtab Ghadiri reviewed the draft manuscript, provided clinical details and patient follow-up, advised on relevant literature, and assisted in selecting and reviewing the imaging sequences included in the report.
- Dr Ramy Nour proposed the case for publication, reviewed the manuscript, and provided expert oversight and a general clinical overview of optic nerve sheath meningioma

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BACKGROUND

Optic nerve sheath meningioma is an uncommon cause of progressive unilateral visual loss and can be difficult to distinguish from inflammatory optic neuropathies on conventional magnetic resonance imaging, particularly in young patients. Delayed or incorrect diagnosis may result in inappropriate management and irreversible vision loss. This case highlights the diagnostic value of Ga-68 DOTATATE PET/CT in a young adult with atypical clinical features and equivocal MRI findings, demonstrating its utility in confirming somatostatin receptor-expressing optic nerve sheath meningioma and avoiding invasive biopsy. The case contributes to the growing evidence supporting somatostatin receptor PET as an important adjunct in the diagnostic pathway for optic nerve lesions.

CONSENT

Yes.

CONFLICT OF INTEREST/DISCLOSURE

The authors have no competing interests to declare.

HUMAN AND ANIMAL RIGHTS

Not applicable. This manuscript describes a single-patient clinical case report and does not involve human experimentation. Written informed consent for publication was obtained from the patient.

ABSTRACT

Background: Optic nerve sheath meningioma is an uncommon tumour arising from the meningotheial cells of the optic nerve sheath. Diagnostic challenges can arise when differentiating optic nerve sheath meningioma and optic neuritis and perineuritis due to similar clinical presentation and magnetic resonance imaging findings. A delay in diagnosis can lead to a delay in appropriate therapy thereby increasing the risk of irreversible vision loss.

Case: A 19-year-old male presented with painless visual loss in the left eye, with optic atrophy noted on clinical examination. Initial Magnetic Resonance Imaging of brain and orbits with gadolinium demonstrated increased T2 signal in and enhancement of the left optic nerve with the diagnostic impression being left optic neuritis. Initial laboratory testing excluded infectious and inflammatory aetiologies. Repeat Magnetic Resonance Imaging three months later demonstrated persistent enhancement of the left optic nerve. The possibility of optic nerve sheath meningioma was raised and therefore, a Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography was arranged. This demonstrated linear fusiform uptake (Standardised uptake value maximum 6.4) along the left optic nerve extending to the orbital apex, consistent with a somatostatin receptor expressing lesion such as an optic nerve sheath meningioma.

Conclusion: This case highlights the diagnostic value of Gallium-68 DOTA-octreotide Positron Emission Tomography/Computed Tomography in confirming optic nerve sheath meningioma and avoiding invasive biopsy in young patients with atypical or equivocal Magnetic Resonance Imaging findings and negative inflammatory markers.

CASE REPORT

CASE PRESENTATION

Clinical Presentation

A 19-year-old male presented with painless visual loss in the left eye, with optic atrophy noted on clinical examination. He denied headache, diplopia or other neurological or systemic symptoms. Past medical history was unremarkable. There was no family history of neurofibromatosis. Examination was notable for reduced corrected visual acuity of 6/36 on the left and normal visual acuity of 6/5 on the right. There was left optic disc pallor and atrophy. He had a left-sided relative afferent pupillary defect. The remainder of the neurologic examination was unremarkable.

Imaging and Pathology Findings

Initial Gadolinium enhanced Magnetic Resonance Imaging (MRI) of the orbits (Figure 1) demonstrated T2 hyperintensity of the left optic nerve with peri-optic enhancement, with the initial diagnostic impression being optic neuritis. An MRI spine (Figure 2) showed no spinal cord demyelination. Full blood count, renal and hepatic panels were unremarkable. The C-reactive protein (CRP) was 5.7mg/L (N < 5.0) and Erythrocyte Sedimentation Rate (ESR) was normal. Serologic studies including serum electrophoresis and immunofixation, antinuclear antibodies (ANA), extractable nuclear antigen (ENA), antineutrophil cytoplasmic antibodies (ANCA), anti-double-stranded DNA antibodies (DsDNA), antiphospholipid antibodies, complement C3 and C4, angiotensin-converting enzyme (ACE), IgG, IgA, IgM, IgG subclasses, Serum myelin oligodendrocyte glycoprotein (MOG) and, neuromyelitis optica (NMO) negative. A broad infectious screen was negative including hepatitis B, hepatitis C, syphilis, HIV and QuantiFERON Gold. Lumbar puncture showed normal cerebrospinal fluid biochemistry, oligoclonal bands, ACE, and cytology. A Computed Tomography (CT) chest (Figure 3) showed no evidence of sarcoidosis. Given the unclear aetiology of the symptoms, no treatment was initiated and the patient was observed with intention for serial imaging.

Repeat MRI (Figure 4) three months later demonstrated persistent mild thickening and enhancement of the left optic nerve sheath with optic nerve hyperintensity and atrophy. The possibility of Optic nerve sheath meningioma (ONSM) was raised and therefore, a Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography (Ga-68 DOTATATE PET/CT) was arranged. This demonstrated linear fusiform tracer uptake (Standardised uptake value maximum (SUVmax) 6.4) along the left optic nerve extending to the orbital apex; consistent with a somatostatin receptor-expressing lesion such as an optic nerve sheath meningioma (Figure 5).

Management

Given imaging findings consistent with optic nerve sheath meningioma and the absence of evidence for inflammatory or

infectious pathology, biopsy and surgical intervention were avoided due to the risk of irreversible visual loss. The patient was referred to radiation oncology for assessment, where management options including stereotactic radiotherapy were discussed. In view of the patient's young age and relatively stable visual function, a conservative approach with close surveillance and multidisciplinary team review was recommended.

Follow-up

At follow-up, visual acuity in the left eye remained relatively stable over a six-month period, measuring approximately 6/24. The option of fractionated stereotactic radiotherapy over six weeks was discussed. The patient was counselled regarding a high probability of tumour control of approximately 85–90% and informed of an estimated 1–3% lifetime risk of radiation-induced malignancy. Referral for multidisciplinary team review was arranged, and clinical genetics referral was suggested given the association with neurofibromatosis type 2.

DISCUSSION

Aetiology & Demographics

ONSM is a benign neoplasm arising from meningotheial (arachnoid cap) cells of the optic nerve sheath and accounts for approximately 1–2% of all intracranial meningiomas [1]. It most commonly affects middle-aged adults, with a marked female predominance, and reported female-to-male ratios of up to 5:1 [2]. ONSM is uncommon in children and young adults, and presentation in this age group may delay diagnosis because demographics are atypical and imaging findings may overlap with inflammatory optic neuropathies [3]; recognised risk factors include neurofibromatosis type 2 and, rarely, prior cranial irradiation [2].

Clinical & Imaging Findings

Clinically, ONSM typically presents with slowly progressive, painless unilateral visual loss, often accompanied by optic disc pallor and relative afferent pupillary defect [1]. On MRI, the characteristic appearance is circumferential thickening of the optic nerve sheath with avid enhancement, producing the classic tram-track sign on post-contrast axial T1-weighted images, while the optic nerve itself is relatively spared [4]. Chronic cases may demonstrate optic nerve atrophy.

However, MRI findings may be indeterminate, particularly early in the disease course or in younger patients, and may overlap with optic neuritis, optic perineuritis, sarcoidosis, or infiltrative neoplastic processes [2,3]. Somatostatin receptor-based molecular imaging has therefore emerged as a valuable adjunct. Meningiomas demonstrate high expression of somatostatin receptor subtype-2, enabling sensitive detection using Ga-68-labelled somatostatin analogues. Ga-68 DOTATATE PET/CT typically demonstrates intense linear or fusiform uptake along the optic nerve sheath, a characteristic pattern for optic nerve

sheath meningioma that provides high diagnostic confidence and resolves diagnostic uncertainty when MRI findings are equivocal [5-7].

Plain radiography and scintigraphy have no established role in the routine evaluation of optic nerve sheath meningioma [2]. Orbital ultrasound may demonstrate enlargement of the optic nerve sheath; however, it is not routinely used for definitive diagnosis [2,3].

Treatment & Prognosis

Surgical resection and biopsy are generally avoided in optic nerve sheath meningioma because of high risk of iatrogenic optic nerve injury and irreversible visual loss [2]. Observation is associated with progressive visual loss in up to 86% of patients but may be considered in selected individuals with normal baseline vision [3]. In cases of progressive visual decline, fractionated stereotactic radiotherapy is the preferred first-line treatment, achieving high local tumour control with acceptable preservation of visual function [2]. Management options after radiotherapy remain limited, as further radiation is often contraindicated and systemic therapies have not demonstrated proven efficacy [2]. Alternative medical therapies, including chemotherapy and hormonal manipulation, have been reported only in isolated cases, while hydroxyurea has shown potential benefit in a small number of patients; however, overall evidence remains limited and these approaches are not established treatments [3]. Somatostatin receptor-targeted Positron emission tomography (PET) imaging may support consideration of theranostic approaches, including peptide receptor radionuclide therapy, in selected progressive or unresectable meningiomas [8]. Hybrid Positron emission tomography/ Magnetic resonance imaging (PET/MRI) may further improve anatomical localisation and delineation of meningiomas at the orbital apex and skull base and assist in treatment planning [9].

Radiotherapy has been associated with stability or improvement of visual function in more than 80% of patients, with outcomes influenced by baseline visual acuity and timing of treatment [2].

Differential Diagnoses

The principal differential diagnoses for ONSM include optic neuritis, optic perineuritis, sarcoidosis, optic nerve glioma, lymphoma, leukemia, and metastatic disease [2,3]. Inflammatory optic neuropathies typically present with more acute visual loss and demonstrate clinical, serologic, or cerebrospinal fluid evidence of inflammation, with absent or minimal uptake on somatostatin receptor PET imaging [5]. Awareness of physiological Ga-68 DOTATATE uptake in normal tissues, including the pituitary gland, salivary glands, liver, spleen, and kidneys, is essential to avoid misinterpretation [10]. The combination of characteristic MRI features and intense somatostatin receptor uptake on PET allows reliable differentiation of ONSM from these alternative diagnoses.

CONCLUSION

This case highlights the critical diagnostic value of Ga-68 DOTATATE PET/CT in evaluating ambiguous optic nerve pathology. In a young patient with persistent MRI enhancement and negative inflammatory and infectious workup, somatostatin receptor PET enabled confident diagnosis of optic nerve sheath meningioma, directly influencing management and avoiding biopsy in a vision-critical region. This case demonstrates the emerging role of nuclear medicine as an essential adjunct for differentiating meningioma from inflammatory optic neuropathies when conventional imaging such as MRI remains equivocal.

TEACHING POINT

Optic nerve sheath meningioma classically demonstrates tram-track perineural enhancement on contrast-enhanced MRI, with corresponding somatostatin receptor-avid uptake on Ga-68 DOTATATE PET/CT. This multimodality imaging pattern is particularly valuable when MRI findings are equivocal, allowing differentiation from inflammatory optic neuropathies and potentially obviating the need for biopsy in vision-critical regions.

QUESTIONS

1. Which imaging feature is most characteristic of optic nerve sheath meningioma on contrast-enhanced Magnetic Resonance Imaging?

- A. Homogeneous enhancement of the optic nerve parenchyma
- B. Tram-track enhancement surrounding the optic nerve (applies)
- C. Central necrosis with peripheral enhancement
- D. T2 hyperintensity with restricted diffusion
- E. Optic nerve enlargement without enhancement

Correct Answer: B

Applies to article: *Chavhan GB, Shroff MM. Twenty classic signs in neuroradiology: A pictorial essay. Indian J Radiol Imaging. 2009;19(2):135-145. PMID: 19881070*

Explanation:

A. Homogeneous enhancement of the optic nerve parenchyma
This finding is more typical of intrinsic optic nerve pathology such as optic neuritis or optic nerve glioma. In optic nerve sheath meningioma, the optic nerve itself is usually relatively spared.

B. Optic nerve sheath meningioma classically demonstrates tram-track enhancement on contrast-enhanced Magnetic Resonance Imaging, representing enhancement of the meningioma encasing a relatively non-enhancing optic nerve, which is a characteristic imaging feature of this entity [*“ONSM typically shows the classic ‘tram-track’ sign on post-contrast MRI due to enhancement of the tumor surrounding the optic nerve” [4]*].

C. Central necrosis with peripheral enhancement

Central necrosis is not a feature of optic nerve sheath meningioma and is more suggestive of aggressive neoplasms or high-grade malignancies.

D. T2 hyperintensity with restricted diffusion

Restricted diffusion is not a characteristic feature of optic nerve sheath meningioma and is more commonly seen in hypercellular tumors such as lymphoma or in acute ischemic processes.

E. Optic nerve enlargement without enhancement

Optic nerve sheath meningioma characteristically enhances avidly; lack of enhancement would argue against this diagnosis.

2. Which patient demographic is most commonly affected by optic nerve sheath meningioma?

- A. Paediatric patients
- B. Young females
- C. Middle-aged to older females (applies)
- D. Elderly males
- E. Patients with a history of malignancy

Correct Answer: C

Applies to article: *Turbin RE, Pokorny K. Diagnosis and treatment of orbital optic nerve sheath meningioma. Cancer Control. 2004;11(5):334–341. PMID: 15377993*

Explanation:

A. Paediatric patients
Optic nerve sheath meningioma is rare in children and adolescents.

B. Young females

Although there is a female predominance, optic nerve sheath meningioma typically presents in middle-aged or older adults rather than young females.

C. Middle-aged to older females

This is the most commonly affected demographic, with a female-to-male ratio reported to be as high as 5:1 [*“female predominance with ratios reported as high as 5:1” [2]*].

D. Elderly males

While optic nerve sheath meningioma can occur in males, elderly males are not the most commonly affected group.

E. Patients with a history of malignancy

There is no strong association between optic nerve sheath meningioma and prior systemic malignancy.

3. Which imaging modality is most useful for detecting optic canal involvement in optic nerve sheath meningioma?

- A. Non-contrast Computed Tomography
- B. Contrast-enhanced Computed Tomography (applies)
- C. Non-contrast Magnetic Resonance Imaging
- D. Contrast-enhanced Magnetic Resonance Imaging
- E. Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography

Correct Answer: B

Applies to article: *Turbin RE, Pokorny K. Diagnosis and*

treatment of orbital optic nerve sheath meningioma. Cancer Control. 2004;11(5):334–341. PMID: 15377993

Explanation:

A. Non-contrast Computed Tomography

Non-contrast Computed Tomography may demonstrate calcification but is less sensitive for assessing optic canal narrowing or hyperostosis.

B. Contrast-enhanced Computed Tomography

Contrast-enhanced Computed Tomography is particularly useful for demonstrating optic canal hyperostosis, calcification, and bony canal narrowing, which are characteristic features of optic nerve sheath meningioma and may be less obvious on Magnetic Resonance Imaging [*“CT is superior in demonstrating optic canal hyperostosis and calcification associated with ONSM” [2]*].

C. Non-contrast Magnetic Resonance Imaging

Non-contrast MRI provides limited evaluation of bony structures and optic canal involvement.

D. Contrast-enhanced Magnetic Resonance Imaging

While MRI is excellent for soft tissue assessment, it is less sensitive than CT for detecting subtle bony changes such as optic canal hyperostosis.

E. Gallium-68 DOTA-octreotate Positron

Emission Tomography/Computed Tomography

Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography is valuable for confirming meningioma through somatostatin receptor expression but does not provide sufficient spatial resolution for detailed assessment of bony optic canal involvement.

4. What is the preferred first-line management for optic nerve sheath meningioma in patients with progressive visual loss?

- A. Surgical resection of nerve sheath meningioma
- B. Systemic chemotherapy
- C. Observation by serial Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography studies
- D. Fractionated radiotherapy (applies)
- E. Steroid therapy

Correct Answer: D

Applies to article: *Turbin RE, Pokorny K. Diagnosis and treatment of orbital optic nerve sheath meningioma. Cancer Control. 2004;11(5):334–341. PMID: 15377993*

Explanation:

A. Surgical resection of nerve sheath meningioma

Surgical resection carries a high risk of optic nerve injury and permanent vision loss and is generally avoided.

B. Systemic chemotherapy

Chemotherapy has no established role in the management of optic nerve sheath meningioma.

C. Observation by serial Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography studies
Observation may be appropriate in stable disease but is not sufficient when there is progressive visual decline.

D. Fractionated radiotherapy

Fractionated radiotherapy is considered the preferred first-

line treatment for optic nerve sheath meningioma in patients with progressive visual loss, as surgical resection carries a high risk of irreversible optic nerve injury [*“Radiotherapy has become the treatment of choice for ONSM with progressive visual loss, providing tumor control while preserving vision”* [2]].

E. Steroid therapy

Steroids are used for inflammatory optic neuropathies but are not effective for treating meningioma.

5. Which property of optic nerve sheath meningioma underlies the diagnostic utility of Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography imaging?

- A. Increased glucose uptake in inflammatory lesions
- B. High expression of somatostatin receptor subtype 2 on tumour cells (applies)
- C. Increased blood–brain barrier permeability in meningioma
- D. Preferential uptake of Ga-68 by optic nerve axons
- E. High tumour vascularity leading to increased tracer delivery

Correct Answer: B

Applies to article: *Palmisciano P, Watanabe G, Conching A, Ogasawara C, Ferini G, Bin-Alamer O, et al. The Role of [⁶⁸Ga]Ga-DOTA-SSTR PET Radiotracers in Brain Tumors: A Systematic Review. Cancers (Basel). 2022;14(12). PMID: 35740591*

Explanation:

- A. Increased glucose uptake in inflammatory lesions
This describes FDG PET rather than somatostatin receptor-based imaging and is not specific for meningioma.
- B. High expression of somatostatin receptor subtype 2 on tumour cells
The diagnostic utility of Gallium-68 DOTA-octreotate Positron Emission Tomography/Computed Tomography imaging in optic nerve sheath meningioma is based on the high expression of somatostatin receptor subtype 2 on meningioma cells, resulting in intense radiotracer uptake [*“Meningiomas demonstrate high expression of somatostatin receptor subtype 2, enabling sensitive detection with Ga-68-labelled somatostatin analogues”* [6]].
- C. Increased blood–brain barrier permeability in meningioma
Blood–brain barrier permeability does not account for DOTATATE uptake, which is receptor-mediated.
- D. Preferential uptake of Ga-68 by optic nerve axons
The tracer binds to somatostatin receptors on tumour cells, not to optic nerve axons.

E. High tumour vascularity leading to increased tracer delivery

Tracer uptake is determined by receptor expression rather than vascularity alone.

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FIGURES

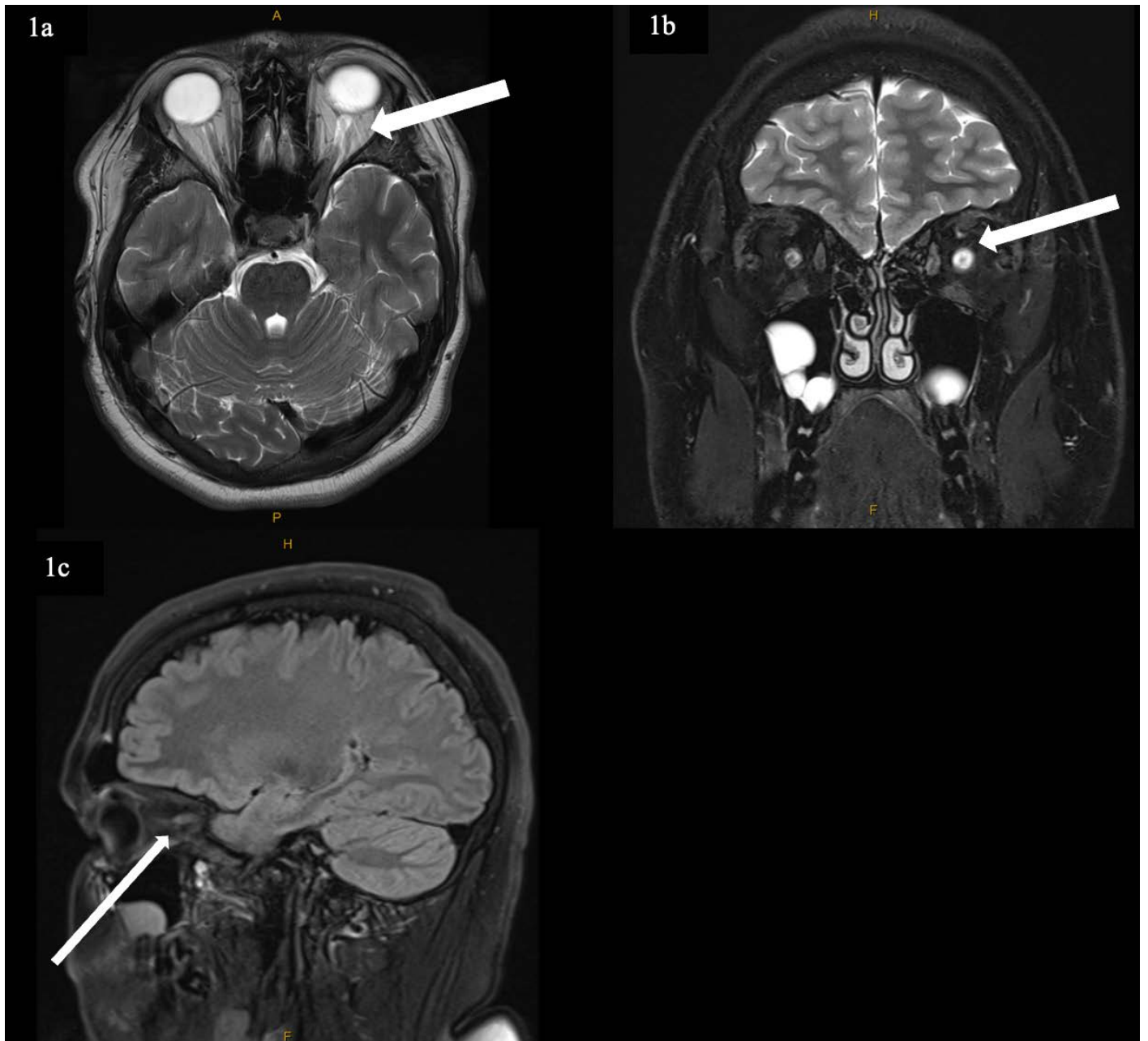


Figure 1: MRI Orbits (a. T2 axial view, b. T2 coronal view, c. Fluid- Attenuated Inversion Recovery (FLAIR) sagittal view)

Age, gender, diagnosis: 19-year-old male with left optic nerve sheath meningioma.

Findings: MRI of the orbits demonstrates increased T2 signal in the optic nerve sheath and within the optic nerve from the level of the mid orbit to the apex with increased signal also extending into the left prechiasmatic nerve (arrow). Associated gadolinium enhancement of the left optic nerve. Radiological impression was left optic neuritis.

Technique: MRI was performed on a 3T system, including sagittal FLAIR; axial T2-weighted, T1-weighted, FLAIR, and diffusion-weighted imaging; and dedicated orbital T1-weighted, T2 fat-suppressed, and post-contrast sequences.



Figure 2: MRI spine (a. T2 axial view, b. Short Tau Inversion Recovery (STIR) sagittal view, T1 post contrast sagittal view)

Age, gender, diagnosis: 19-year-old male with left optic nerve sheath meningioma.

Findings: MRI of the spine shows no intrinsic cord lesions and no abnormal enhancement.

Technique: 3T MRI of the cervical and thoracic spine was performed, including sagittal T1-weighted, T2-weighted, and STIR sequences; axial T2-weighted imaging; and post-contrast sequences.



Figure 3: CT Chest (a. Axial view, b. Coronal view, c. Sagittal view)

Age, gender, diagnosis: 19-year-old male with left optic nerve sheath meningioma.

Findings: CT Chest shows no features of sarcoidosis

Technique: High resolution CT



Figure 4: MRI Orbits 3 months later (a. T2 axial view, b. T2 coronal view, c. Fluid- Attenuated Inversion Recovery (FLAIR) sagittal view, d. T1 post gadolinium axial view)

Age, gender, diagnosis: 19-year-old male with left optic nerve sheath meningioma.

Findings: MRI of the orbits demonstrates circumferential mild thickening and persistent enhancement of the left optic nerve sheath with an associated left optic neuropathy (arrow), raising suspicion of an optic nerve sheath meningioma rather than perioptic inflammatory change.

Technique: High-resolution 3T MRI was performed before and after contrast administration. Sequences included T2 Dixon, diffusion-weighted imaging (DWI), T1-weighted Magnetization-Prepared Rapid Gradient Echo (MPRAGE), 3D T1-weighted Volumetric Interpolated Breath-hold Examination (VIBE), 3D FLAIR, T1-weighted black-blood imaging, and susceptibility-weighted imaging (SWI).

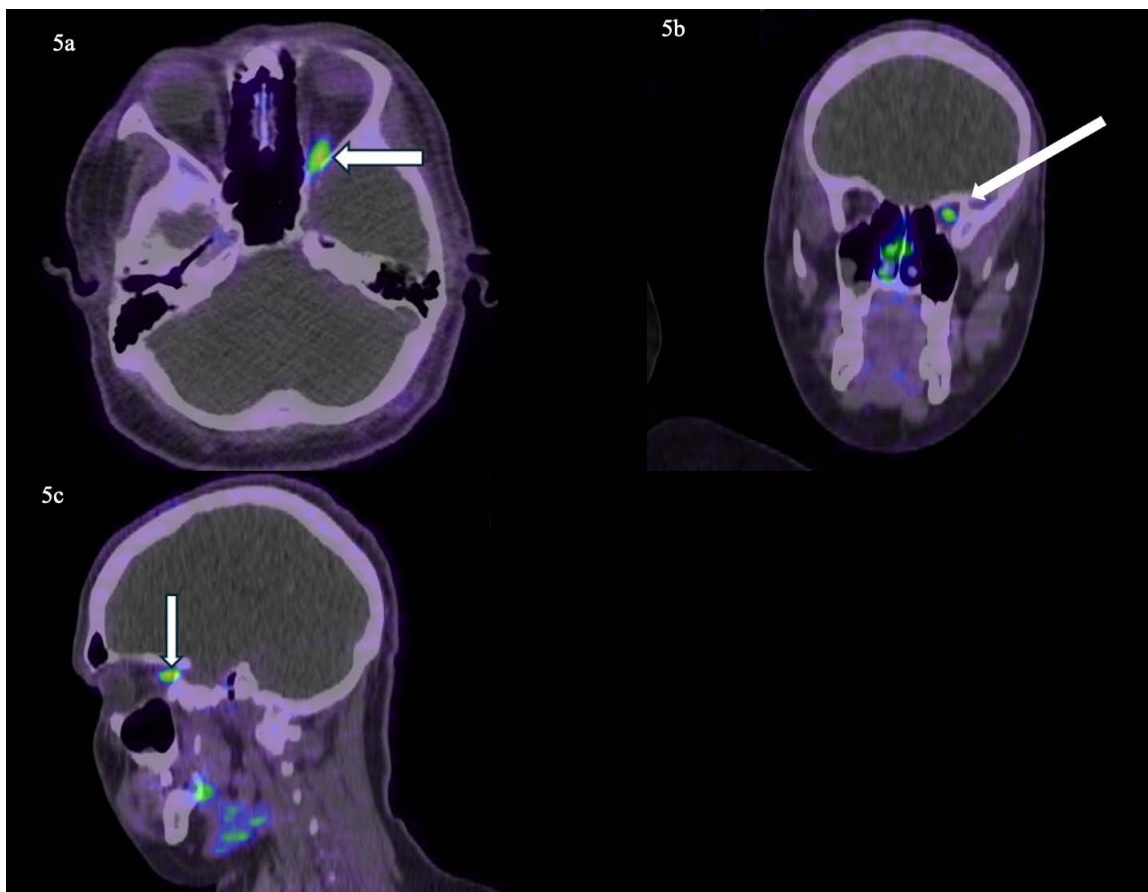


Figure 5: Ga-68 DOTATATE PET/CT (a. Axial view, b. Coronal view, c. Sagittal view)

Age, gender, diagnosis: 19-year-old male with left optic nerve sheath meningioma.

Findings: Axial Ga-68 DOTATATE PET/CT demonstrates intense linear fusiform radiotracer uptake along the left optic nerve extending to the orbital apex (arrow), with a maximum standardised uptake value (**SUV_{max} 6.4**), consistent with somatostatin receptor-expressing meningioma.

Technique: PET/CT performed **59 minutes** after intravenous administration of **Ga-68 DOTATATE (149 MBq)**.

Summary table

Feature	Description
Etiology	Benign tumour arising from meningothelial (arachnoid cap) cells of the optic nerve sheath
Incidence	Accounts for approximately 1–2% of all intracranial meningiomas and ~1–2% of all orbital tumours
Gender ratio	Female predominance, with reported female-to-male ratio up to 5:1
Age predilection	Typically affects middle-aged adults (4th–6th decade); paediatric and young-adult cases are uncommon
Risk factors	Neurofibromatosis type 2 (NF2); prior cranial irradiation (rare); female sex
Treatment	Observation for stable disease; fractionated stereotactic radiotherapy is first-line for progressive visual loss; surgery generally avoided due to high risk of optic nerve injury
Prognosis	Generally indolent tumour with high local control rates; visual outcome depends on baseline visual acuity and timing of treatment
Findings on imaging	MRI: Circumferential optic nerve sheath thickening with classic <i>tram-track</i> enhancement; optic nerve atrophy in chronic cases. Ga-68 DOTATATE PET/CT: Intense linear or fusiform uptake along the optic nerve reflecting somatostatin receptor subtype-2A overexpression

Table legend: Summary of key epidemiologic, clinical, and imaging features of optic nerve sheath meningioma

Differential Diagnosis of Optic Nerve Sheath Meningioma with Imaging Findings

Differential diagnosis	Clinical features	CT findings (incl. X-ray/US/Scintigraphy where applicable)	MRI features (T1, T2, DWI, enhancement pattern)	Ga-68 DOTATATE PET/CT
Optic nerve sheath meningioma (ONSM)	Slowly progressive, painless unilateral visual loss; optic disc pallor; Relative afferent pupillary defect ; typically middle-aged females but can occur in young adults	X-ray: Limited diagnostic role. US: Limited role; may show optic nerve sheath thickening CT: Optic nerve sheath calcification; optic canal hyperostosis or narrowing; soft-tissue sheath thickening. Scintigraphy: No routine role.	T1: Iso- to hypointense sheath-based enhancing lesion. ‘tram- track’ sign on post- contrast T1 with fat suppression T2: Variable; hyperintense but may also be hypointense DWI: Usually no marked diffusion restriction; may show mild restriction depending on cellularity.	Intense linear or fusiform SSTR-avid uptake along the optic nerve sheath
Optic neuritis	Acute or subacute visual loss; often painful eye movements; frequently associated with demyelinating disease	X-ray: Limited role. US: Limited role. CT: Limited role. May show non- specific findings such as subtle optic nerve thickening, swelling, or contrast enhancement Scintigraphy: No role.	T1: Gadolinium contrast enhancement of the optic nerve on T1-weighted fat-suppressed sequences T2: Intrinsic optic nerve hyperintensity with segmental enlargement, most commonly involving the retrobulbar intra-orbital segment DWI: Typically no significant diffusion restriction; mild diffusion abnormality may be seen in the acute phase.	Low or absent SSTR uptake
Optic perineuritis	Subacute visual loss; may be painful;	X-ray: Limited role. US: Limited role. CT: Usually normal or demonstrates mild circumferential perioptic soft tissue thickening; no calcification or osseous hyperostosis. Scintigraphy: No role.	T1: Smooth circumferential perineural enhancement (“tram-track” appearance) on T1-weighted fat-suppressed sequences. T2/STIR: Perioptic sheath hyperintensity and thickening; optic nerve itself typically preserved. DWI: Typically no significant diffusion restriction; may show mild diffusion abnormality in active inflammation.	No significant or intense SSTR uptake; uptake typically low or non-specific.
Sarcoidosis (optic nerve/sheath involvement)	May have systemic or constitutional symptoms; serum ACE may be elevated; abnormal chest imaging common; may involve multiple CNS sites.	X-ray: Chest radiograph may show hilar lymphadenopathy (systemic clue). US: Limited role for optic nerve assessment. CT orbit: may show optic nerve or sheath thickening. Chest CT: may demonstrate bilateral hilar and mediastinal lymphadenopathy. Scintigraphy: Limited role in optic nerve assessment.	T1: Variable enhancement of the optic nerve; may be nodular, diffuse, or perineural; often multifocal CNS involvement. T2: Variable signal abnormality of the optic nerve and/or sheath; may show T2 hyperintensity in active inflammatory disease. DWI: Not a defining feature; diffusion restriction is uncommon but may be seen in active inflammatory involvement.	May show mild to moderate SSTR uptake due to inflammatory activity, but typically lacks the intense linear fusiform sheath-tracking pattern characteristic of ONSM.

<p>Optic nerve glioma</p>	<p>Common in children; associated with NF1; gradual visual decline; may have proptosis</p>	<p>X-ray: Limited role. US: Limited role. CT: Fusiform enlargement of the optic nerve with soft tissue density; typically lacks calcification. May cause smooth optic canal enlargement. Enhancement is variable. Scintigraphy: No routine role.</p>	<p>T1: Enlarged intrinsic nerve. T2: Hyperintense fusiform enlargement of the optic nerve. DWI: Typically no marked restricted diffusion; variable signal. Not a defining feature.</p>	<p>Low or no significant uptake.</p>
<p>Lymphoma / leukaemia infiltration</p>	<p>Subacute or rapid visual decline; may have systemic lymphoma/leukaemia; can be bilateral; possible constitutional symptoms or known hematologic malignancy</p>	<p>X-ray: Limited role. US: Limited role. CT: Soft-tissue infiltration of optic nerve and/or sheath; may appear hyperdense; possible associated orbital mass Scintigraphy: No role.</p>	<p>T1: Iso- to hypointense infiltrative thickening T2: Iso- to mildly hyperintense DWI: Restricted diffusion with low Apparent Diffusion Coefficient values</p>	<p>DOTATATE uptake typically low or variable; orbital lymphoma is more FDG-avid than SSTR-avid.</p>
<p>Metastatic disease</p>	<p>Known systemic malignancy; rapid visual decline; may have other metastatic sites</p>	<p>X-ray: Limited role. May demonstrate orbital mass effect, bone destruction or sclerosis, or optic canal widening or erosion US: Limited role. CT: Nodular or irregular enhancing lesion along optic nerve/sheath; may cause orbital mass effect or optic canal involvement. Bone destruction or sclerosis depending on primary tumour. Scintigraphy: Limited role.</p>	<p>T1: Iso- to hypointense nodular lesion; intrinsic nerve or sheath involvement. T2: Variable signal; often mildly hyperintense; may be heterogeneous. DWI: Variable; may show restricted diffusion in hypercellular metastases.</p>	<p>Uptake variable depending on tumour type; usually low or absent in non-neuroendocrine metastases</p>

Table legend: Differential diagnosis of optic nerve sheath meningioma comparing clinical features and multimodality imaging findings (X-ray, ultrasound, CT, MRI including T1, T2 and DWI sequences, contrast enhancement pattern, scintigraphy, and Ga-68 DOTATATE PET/CT).

KEYWORDS

Optic nerve sheath meningioma; Optic neuritis; Optic perineuritis; Optic nerve tumors; Orbital tumors; Ga-68 DOTATATE PET/CT; Somatostatin receptor imaging; Nuclear medicine; Magnetic resonance imaging

ABBREVIATIONS

ACE = Angiotensin-Converting Enzyme
 ANA = Anti Nuclear Antibodies
 ANCA = Antineutrophil Cytoplasmic Antibodies
 APQ4 = Aquaporin-4 Antibodies
 CRP = C-Reactive Protein
 DsDNA = Anti-Double-Stranded DNA Antibodies
 ENA = Extractable Nuclear Antigen
 ESR = Erythrocyte Sedimentation Ratio
 FLAIR = Fluid- Attenuated Inversion Recovery
 Ga-68 DOTATATE PET/CT = Gallium-68 DOTA-octreotate
 Positron Emission Tomography/Computed Tomography
 MOG = Serummyelin Oligodendrocyte Glycoprotein
 MPRAGE = Magnetization-Prepared Rapid Gradient Echo
 MRI = Magnetic Resonance Imaging
 NMO = Neuro Myelitis Optica
 ONSM = Optic Nerve Sheath Meningioma
 PET = Positron Emission Tomography
 PET/CT = Positron Emission Tomography/Computed Tomography
 PET/MRI = Positron Emission Tomography/ Magnetic Resonance Imaging
 PRRT = Peptide Receptor Radionuclide Therapy
 SSTR = Somato Statin Receptor
 SSTR2A = Somatostatin Receptor Subtype-2A
 STIR = Short Tau Inversion Recovery
 SUVmax = Standardised Uptake Value Maximum
 SWI = Susceptibility-Weighted Imaging
 VIBE = Volumetric Interpolated Breath-Hold Examination

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