

Rhino-Orbito-Paranasal and Cavernous Sinus Mucormycosis Presenting with Extensive Cranial Nerve Palsies in a Patient with Diabetes Mellitus: A Case Report

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ABSTRACT

Rhino-orbito-paranasal and cavernous sinus mucormycosis is a rapidly progressive angioinvasive fungal infection caused by moulds of the order *Mucorales*. It occurs predominantly in patients with poorly controlled diabetes mellitus and carries high mortality when diagnosis is delayed, particularly in South and South-East Asia. Here, the authors report a case of 56-year-old male with long-standing poorly controlled type 2 diabetes mellitus (Glycated Haemoglobin (HbA1c) 13.6%) and a prior episode of myocardial infarction presented with one month of progressive left nasal obstruction. Over the next two months, he developed complete left visual loss, total external ophthalmoplegia, proptosis, ptosis, trigeminal sensory loss involving branches V1-V3, and ipsilateral lower motor neuron facial nerve palsy. Histopathology of nasal crusting showed broad pauci-septate hyphae consistent with *Mucorales*, and fungal culture grew *Rhizopus* species. Magnetic Resonance Imaging (MRI) orbit demonstrated involvement of the left frontal, ethmoidal, sphenoid, and maxillary sinuses, with extension into the superomedial extraconal orbital space, orbital apex, and left cavernous sinus. Associated oedema of the extraocular muscles and pterygoid muscles, diffuse thickening of the left optic nerve, and mild proptosis were also noted. The patient was treated with left functional endoscopic sinus surgery, medial orbital decompression, and liposomal amphotericin B 250 mg daily for 21 days. The present case represents an unusually extensive pattern of cranial nerve involvement in Rhino-Orbito-Cerebral Mucormycosis (ROCM), affecting cranial nerves II-VII, and highlights the need for early tissue diagnosis, prompt antifungal therapy, and multidisciplinary management.

Keywords: Amphotericin B, Cavernous sinus thrombosis, Cranial nerve palsy

CASE REPORT

A 56-year-old male had presented to the Otorhinolaryngology Department with a one-month history of progressive left-sided nasal obstruction. What had begun as a vague sensation of fullness on the left-side had evolved into what he described as a wall of concrete inside his nose. The patient was a known case of type 2 diabetes mellitus diagnosed eight years back, managed with oral hypoglycaemic agents (metformin and glimepiride), but he had been inconsistent in taking his medications. His glycated haemoglobin on admission had been 13.6%, which suggested long-standing uncontrolled glycaemic control. He had a history of an anterior wall myocardial infarction treated medically four months back.

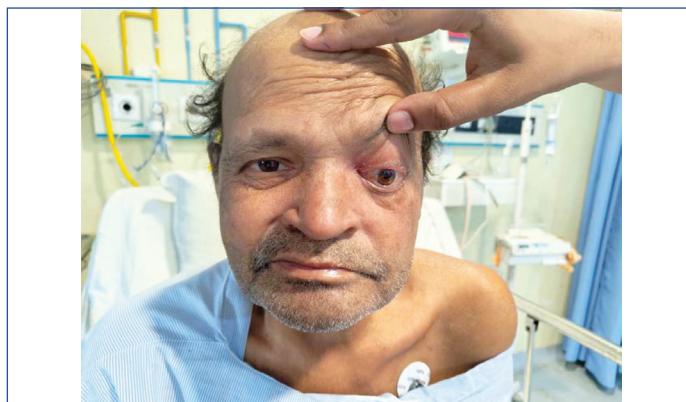
Ten days after the onset of nasal symptoms, he had noticed blurring, followed by progressive loss of vision in the left eye, accompanied by periorbital swelling and a dull frontoparietal headache. Over the next one month, after the onset of ocular symptoms, the visual loss had become complete; extraocular movements in the left eye had been entirely abolished; the upper lid had drooped; and the globe had been displaced forward. [Table/Fig-1,2].

The patient had been unable to raise the left eyebrow or puff up his cheeks on the left-side. The angle of his mouth had pulled to the right with every attempt at expression. He could not whistle, could not puff out his cheeks. The absence of any movement across the left-side of the face, combined with the proptotic fixed eye [Table/Fig-1,2].

A complete lack of light perception on the left, a fixed dilated pupil, absolute ophthalmoplegia, about 6 mm of proptosis on Hertel exophthalmometry, and no corneal reflex had all been verified by an ophthalmological examination. Examining the fundus had been

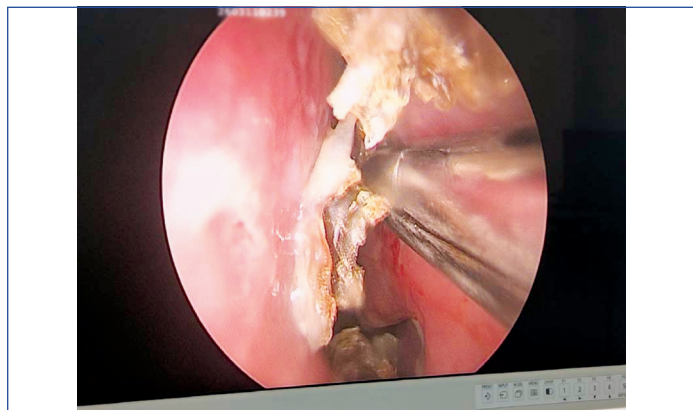


[Table/Fig-1]: Clinical image demonstrating severe left periorbital oedema with complete ptosis, suggestive of orbital involvement with deviation of mouth to right-side and loss of nasolabial fold of the left-side.



[Table/Fig-2]: Clinical photograph showing left orbital involvement with ptosis, ophthalmoplegia, and restricted ocular movements.

challenging due to significant ocular hypotony. A nasal endoscopy had shown widespread crusting, frank black discolouration of the left middle turbinate, and pale, nearly bloodless mucosa throughout the left nasal cavity [Table/Fig-3].



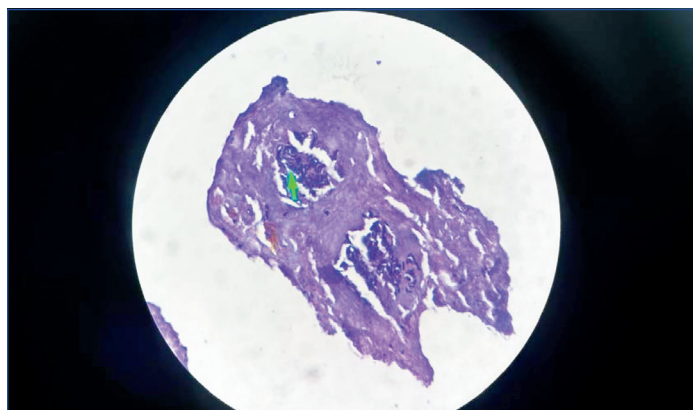
[Table/Fig-3]: Intraoperative endoscopic image during functional endoscopic sinus surgery showing black necrotic crusting and eschar within the nasal cavity in mucormycosis, with active endoscopic debridement using forceps.

The cranial nerve deficiencies had been mapped as follows by neurological testing. Complete visual loss (cranial nerve II), absence of globe adduction, elevation, and depression with ptosis (cranial nerve III), absence of intorsion (cranial nerve IV), sensory loss in all trigeminal nerve branches with a reduced jaw jerk (cranial nerve V), absence of globe abduction (cranial nerve VI), and lower motor neuron facial palsy affecting the forehead, orbicularis oculi, and orbicularis oris (cranial nerve VII) were all present on the left side. Meningism, elevated intracranial pressure, and altered consciousness were all absent.

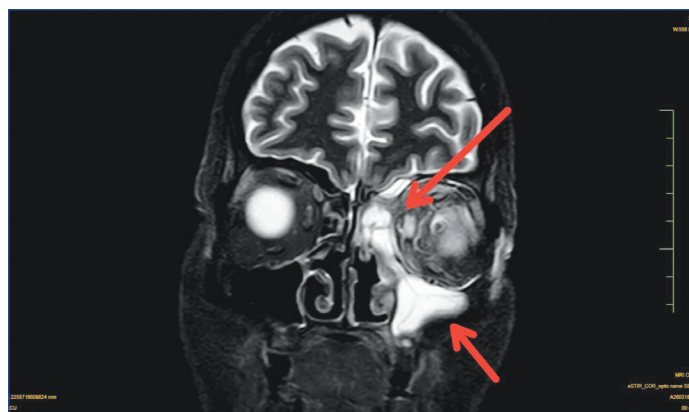
Laboratory investigations had shown a fasting plasma glucose of 18.4 mmol/L, HbA1c of 13.6%, C-reactive protein of 78 mg/L, and erythrocyte sedimentation rate of 64 mm/h. Serum creatinine had been 0.6 mg/dL (done as a baseline in view of the anticipated nephrotoxic treatment regimen). A full blood count, coagulation screen, and serum iron studies had been performed; iron indices had shown low transferrin saturation.

Scrapings and crusting from the left nasal cavity had been submitted for direct microscopy and fungal culture. Microscopy with the Periodic Acid-Schiff (PAS) stain had shown *Mucor* fungi [Table/Fig-4]. Histopathological examination of biopsied sinonasal mucosa on PAS stain had confirmed the presence of *Mucor*.

MRI orbit [Table/Fig-5] had been done and was suggestive of polypoidal thickening and mucosal collection in the left frontal, ethmoidal, sphenoid and maxillary sinuses. An ill-defined soft-tissue collection had been noted in the superomedial extraconal space in the orbit, which was extending to the orbital apex and left cavernous sinus. The extraocular muscles and left medial and lateral pterygoid muscles were oedematous with T2-weighted imaging/Short Tau Inversion Recovery (T2/STIR) hyperintensity. The distal intraorbital segment of the left optic nerve showed T2/STIR rim hyperintensity,



[Table/Fig-4]: Microscopic image showing mucor fungi (PAS) (100x).



[Table/Fig-5]: MRI of the orbit and paranasal region showing coronal T2/STIR demonstrating left Rhino-Orbito-Cerebral Mucormycosis (ROCM).

with the left optic nerve diffusely thickened, and mild proptosis was seen.

After urgent blood glucose optimisation with an intravenous insulin infusion and a multidisciplinary review involving otorhinolaryngology, ophthalmology, neurosurgery, and the infectious diseases service, the patient had been taken to theatre. He had undergone left full-house functional endoscopic sinus surgery incorporating complete sphenoethmoidectomy, total left middle turbinectomy, maxillary antrostomy, and frontal sinus drainage-combined with right middle meatal antrostomy and left medial orbital decompression through the lamina papyracea.

The left middle turbinate had been frankly necrotic; there had been complete mucosal necrosis across all left paranasal sinuses; fungal debris and necrotic tissue had extended into the left posterior nasal space; and the posterior wall of the right maxillary sinus had also appeared unhealthy. Intraoperative histopathology had confirmed the diagnosis. Adequate surgical clearance had been achieved within the accessible sinonasal compartment, but the degree of orbital apex and cavernous sinus involvement had precluded complete resection without an unacceptable risk of haemorrhage or further cranial nerve injury.

Liposomal Amphotericin B 250 mg per day had been given for 21 days as part of medical management. Consequently, step-down therapy with oral posaconazole (300 mg twice daily) had been administered. However, the patient had subsequently developed acute kidney injury, following which posaconazole had been discontinued.

Based on the culture and sensitivity findings from the nasal cavity, which had revealed growth of *Klebsiella pneumoniae* (sensitive to meropenem), intravenous meropenem had been used to treat secondary bacterial sinusitis for 10 days along with Amphotericin B. By the end of the first week of admission, fasting blood glucose had dropped below 7.5 mmol/L under a basal-bolus insulin regimen for glycaemic control, and the patient had had a slight improvement in drooping of the left eyelid before discharge, but unfortunately, the patient had been lost to follow-up.

DISCUSSION

Mucormycosis is a disease of the modern period in many ways. Saprophytic moulds from the class Zygomycetes and order Mucorales are the cause. Diabetes mellitus has supplanted haematological malignancy as the most common predisposing condition in Asia, particularly in the Indian subcontinent [1,2]. According to some institutional series from northern India, the prevalence of mucormycosis can reach 0.14 cases per 1000 diabetic admissions—a figure that most European doctors would consider extraordinary [1-3].

The reasons underlying the increased vulnerability of diabetic patients have been extensively investigated. Chronic hyperglycaemia reduces neutrophil chemotaxis, impairs phagocytosis, and perhaps most importantly raises serum levels of available iron,

Parameters	Sachdeva K 2013, India (6 cases) [14]	Munasinghe KVP and Silva FHDS, 2023, Sri Lanka [16]	Sadiq F et al., 2025, Pakistan/Kyrgyzstan [17]	Nagendra V et al., 2020, India [9]	Present case
Age/Gender	42-65 years (mean 52) Male: Female=5:1	70 years/Female	47 years/Female	60 years/Male	56 years/Male
Predisposing factor (s)	Uncontrolled diabetes mellitus (6/6); diabetic ketoacidosis (2/6); recent surgery/trauma (4/6)	Poorly controlled type 2 diabetes mellitus×15 years; no diabetic ketoacidosis	Uncontrolled diabetes mellitus + iatrogenic Cushing syndrome (2 years steroid use)	Poorly controlled diabetes mellitus + nephropathy; Hepatitis B positive	Long-standing poorly controlled type 2 diabetes mellitus (Haemoglobin A1c 13.6%) + recent myocardial infarction; no diabetic ketoacidosis
Cranial nerves involved	II, III, IV, V, VI, VII (each of 6 patients, unilateral)	I-VII (left-side)	VI, VII only	III, IV, V, VI, VII, IX, X, XII (Garcin syndrome-8 nerves)	II, III, IV, V (V1-V3); VI, VII (6 nerves, left-side)
Diagnostic confirmation	Histopathology - aseptate hyphae (6/6); culture positive (3/6)	Direct microscopy + histopathology + culture (Rhizopus species)	Potassium hydroxide wet mount + histopathology (buccal biopsy); culture not reported	Tissue culture (Rhizopus oryzae); bedside debridement swab	Potassium hydroxide microscopy + histopathology + culture (Rhizopus species)-all 3 positive
Treatment	Intravenous Amphotericin B (6 weeks) + Caldwell-Luc surgery/debridement (4/6)	Liposomal amphotericin B 3 mg/kg/day×85 days + functional endoscopic sinus surgery + orbital decompression (no exenteration)	Amphotericin B 0.7 mg/kg/day-medical management only, no surgery	Liposomal amphotericin B 5 mg/kg/day + posaconazole + meropenem; bedside debridement only (surgery precluded)	Liposomal amphotericin B 250 mg/day×21 days + step-down posaconazole (stopped for acute kidney injury) + functional endoscopic sinus surgery + orbital decompression
Outcome	2/6 died (<72 hours); 4/6 survived with residual Cranial Nerve VI/VII palsy; 1 recurrence at 6 months	Survived; residual blindness & Cranial Nerve palsies; slight ptosis improvement	Survived; Cranial Nerve VI palsy fully resolved by 1 month; facial palsy improved	Fatal - died within 8 hours of multiorgan dysfunction; bilateral anterior cerebral artery infarct	Survived; residual blindness and Cranial Nerve palsies; slight ptosis improvement

[Table/Fig-6]: Comparative summary of published cases of Rhino-Orbito-Cerebral Mucormycosis (ROCM) with extensive cranial nerve involvement [9,14,16,17].

which Mucorales species avidly acquire through high-affinity iron permeases and cell surface reductases [4-6].

Cranial nerve involvement is well described as a consequence of cavernous sinus infiltration, typically affecting cranial nerve II through VI [7]. Facial nerve (cranial nerve VII) involvement is considerably rarer and therefore less familiar to clinicians, as it requires the infection to extend along the skull base or through the petrous temporal bone to reach the stylomastoid foramen [8]. Even rarer still is concomitant thrombosis of the lateral dural sinuses in the absence of otogenic disease a finding that, to our knowledge, has been reported only in isolated cases [7-9].

Mucorales germination and tissue invasion are strongly favoured by persistent hyperglycaemia and the subsequent alteration in transferrin- iron dynamics in uncontrolled diabetes [7]. Once hyphae penetrate vessel walls, the resulting thrombosis and ischaemia not only kill tissue but also impede the delivery of antifungal drugs and host immune effectors to the site of infection [8]. This observation helps explain both the clinical aggressiveness of the disease and the difficulty of treating it once it is established [7,10].

Orbital apex syndrome, as seen here, reflects the anatomical reality that an extraordinary density of critical neurovascular structures- the optic nerve, three ocular motor nerves, and the first division of the trigeminal nerve- must traverse a very confined bony corridor [10]. Even modest fungal infiltration at this site produces profound deficits that, in the case of the optic nerve, are almost always irreversible by the time the diagnosis is established [7,10,11].

The most plausible route of spread in the present case was along the skull base from the sphenoid sinus or the petrous apex, with the infection reaching the facial nerve either at or near the stylomastoid foramen [10].

Regarding treatment, medical management alone is not effective because of poor drug delivery to the infection due to extensive vascular thrombosis. Systemic antifungal therapy, including high-dose amphotericin B, is associated with an overall survival rate of 72% [12,13]. It is usually given in 5% dextrose in water intravenously at a dose of 1.0-1.5 mg/kg daily. Since its usage is associated with renal toxicity, it requires careful monitoring of serum urea nitrogen, blood urea nitrogen, potassium, creatinine, and creatinine clearance as an essential part of the therapy [5,12-14]. Recently, studies have shown that liposomal amphotericin B, fluconazole, and caspofungin, along with hyperbaric oxygen therapy (believed to improve neutrophil killing by increasing oxygen delivery), low-dose

heparin, and anti-inflammatory medications, can be used with good success [4,6,13,14].

Functional endoscopic sinus surgery has transformed the surgical management of ROCM over the past two decades, largely replacing the more disfiguring open approaches that were standard practice until the mid-1990s [13]. Functional endoscopic sinus surgery offers direct endoscopic visualisation of the sinuses and the skull base, allows repeat debridement under general or local anaesthesia with relatively low morbidity, and, crucially, provides the tissue access needed for definitive histopathological diagnosis [13,14]. The decision about whether to add orbital exenteration in cases with orbital involvement remains genuinely difficult [13]. There is evidence that exenteration improves survival in some series, but that evidence comes almost entirely from studies in which the indication was complete orbital soft-tissue involvement; the benefit in cases of orbital apex disease without frank globe or extraocular muscle necrosis is much less certain, and orbital decompression without exenteration, as was done here, represents a reasonable middle ground when optic nerve function is already lost but the extent of soft-tissue necrosis is relatively limited [8,13,14].

The outcome of ROCM is largely determined by three factors that interact in complex ways: the rapidity of diagnosis, the promptness of antifungal therapy initiation, and the reversal of the underlying predisposing condition [12,13]. A systematic analysis of published cases by Roden MM et al., reviewing nearly 930 cases spanning more than a century of published literature-found overall survival from rhinocerebral mucormycosis to be approximately 61% when amphotericin B was used, but substantially lower in patients with intracranial extension [13]. More recent data from the large Jeong W et al., systematic review confirm that overall mortality in published mucormycosis cases remains in the region of 46%, despite decades of advances in antifungal therapy-a sobering reminder that we have not yet solved this problem [12,13,15]. Comparative summary of published cases of ROCM with extensive cranial nerve involvement is given in [Table/Fig-6] [9,14,16,17].

CONCLUSION(S)

The present case illustrates, perhaps more completely than most single case reports can, the full range of cranial nerve destruction that rhinocerebral mucormycosis is capable of producing. The clinical presentation of an incorporating ophthalmoplegia, complete visual loss, trigeminal sensory and motor deficit, and lower motor neuron facial palsy, in a patient with poorly controlled diabetes, should prompt

immediate investigation for mucormycosis. Nasal endoscopy with tissue sampling for direct microscopy, histopathology, and fungal culture is the single most important diagnostic step; results must be acted on without delay. Injectable liposomal Amphotericin B at standard doses, initiated as soon as the histopathological diagnosis is made, combined with aggressive surgical debridement and urgent glycaemic optimisation, represents the treatment framework supported by the best available evidence. The co-occurrence of lateral sinus thrombosis, particularly on a background of a hypoplastic venous system, adds a further dimension to an already complex picture and may warrant consideration of anticoagulation in future similar cases, though this question remains unresolved in the literature. By documenting the present case in detail, the authors aim to contribute to the growing body of clinical evidence and experience that may help improve outcomes in this devastating disease.

REFERENCES

- [1] Prakash H, Chakrabarti A. Global epidemiology of mucormycosis. *J Fungi*. 2019;5(1):26. Doi: 10.3390/jof5010026
- [2] Patel A, Kaur H, Xess I, Michael JS, Savio J, Rudramurthy S, et al. A multicentre observational study on the epidemiology, risk factors, management and outcomes of mucormycosis in India. *Clin Microbiol Infect* 2020;26(7):944.e9-944.e15. Doi: 10.1016/j.cmi.2019.11.021.
- [3] Ribes JA, Vanover-Sams CL, Baker DJ. Zygomycetes in human disease. *Clin Microbiol Rev*. 2000;13(2):236-301. Doi: 10.1128/CMR.13.2.236.
- [4] Alqarihi A, Kontoyiannis DP, Ibrahim AS. Mucormycosis in 2023: An update on pathogenesis and management. *Front Cell Infect Microbiol*. 2023;13:1254919. Doi: 10.3389/fcimb.2023.1254919.
- [5] Ibrahim AS, Spellberg B, Walsh TJ, Kontoyiannis DP. Pathogenesis of mucormycosis. *Clin Infect Dis*. 2012;54 Suppl 1(Suppl 1):S16-S22. Doi: 10.1093/cid/cir865.
- [6] Spellberg B, Edwards J, Ibrahim A. Novel perspectives on mucormycosis: Pathophysiology, presentation, and management. *Clin Microbiol Rev*. 2005;18(3):556-69. Doi: 10.1128/CMR.18.3.556-569.2005.
- [7] Hosseini SMS, Borghei P. Rhinocerebral mucormycosis: Pathways of spread. *Eur Arch Oto-Rhino-Laryngol Head Neck*. 2005;262(11):932-38. Doi: 10.1007/s00405-005-0919-0
- [8] Thurtell MJ, Chiu ALS, Goold LA, Akdal G, Crompton JL, Ahmed R, et al. Neuro-ophthalmology of invasive fungal sinusitis: 14 consecutive patients and a review of the literature. *Clin Experiment Ophthalmol*. 2013;41(6):567-76. Doi: 10.1111/ceo.12055.
- [9] Nagendra V, Thakkar KD, Prasad Hrish A, Prathapadas U. A rare case of rhinocerebral mucormycosis presenting as garcin syndrome and acute ischemic stroke. *Indian J Crit Care Med Peer-Rev Off Publ Indian Soc Crit Care Med*. 2020;24(11):1137-38.
- [10] Petrikos G, Skiada A, Lortholary O, Roilides E, Walsh TJ, Kontoyiannis DP. Epidemiology and clinical manifestations of mucormycosis. *Clin Infect Dis*. 2012;54(suppl_1):S23-S34. Doi: 10.1093/cid/cir866.
- [11] Skiada A, Lass-Floerl C, Klimko N, Ibrahim A, Roilides E, Petrikos G. Challenges in the diagnosis and treatment of mucormycosis. *Med Mycol*. 2018;56(suppl_1):93-101. Doi: 10.1093/mmy/myx101.
- [12] Roden MM, Zaoutis TE, Buchanan WL, Knudsen TA, Sarkisova TA, Schaufele RL, et al. Epidemiology and outcome of zygomycosis: A review of 929 reported cases. *Clin Infect Dis*. 2005;41(5):634-53. Doi: 10.1086/432579.
- [13] Cornely OA, Alastruey-Izquierdo A, Arenz D, Chen SCA, Dannaoui E, Hochhegger B, et al. Global guideline for the diagnosis and management of mucormycosis: An initiative of the European Confederation of Medical Mycology in cooperation with the Mycoses Study Group Education and Research Consortium. *Lancet Infect Dis*. 2019;19(12):e405-e421. Doi: 10.1016/S1473-3099(19)30312-3.
- [14] Sachdeva K. Rhino-oculo cerebral mucormycosis with multiple cranial nerve palsy in diabetic patient: Review of six cases. *Indian J Otolaryngol Head Neck Surg*. 2013;65(4):375-79. Doi: 10.1007/s12070-013-0659-1.
- [15] Jeong W, Keighley C, Wolfe R, Lee WL, Slavin MA, Kong DCM, et al. The epidemiology and clinical manifestations of mucormycosis: A systematic review and meta-analysis of case reports. *Clin Microbiol Infect*. 2019;25(1):26-34. Doi: 10.1016/j.cmi.2018.07.011.
- [16] Munasinghe KVP, Silva FHDS. Rhinocerebral Mucormycosis with extensive cranial nerve palsies in a diabetic Patient. *Cureus*. 2023;15(12):e50451. Doi: 10.7759/cureus.50451.
- [17] Sadiq F, Gohar A, Ahmad MH, Ali M, Khan AU, Ahmed B, et al. A rare case report of mucormycosis presenting as cranial nerve palsies in a patient with diabetes and iatrogenic cushing syndrome. *Medicine (Baltimore)*. 2025;104(34):e43886. Doi: 10.1097/MD.00000000000043886.

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