Radiology Case Reports 19 (2024) 3693–3700

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Cerebritis, optic ischemia, and cavernous sinus thrombosis arising from sinonasal mucormycosis ☆

Eliseo Picchi, PhD, MD[a,b], Noemi Pucci, MD[a,c,], Alessia Amatruda, MD[a], Francesca Fu, MD[a], Paola Leomanni, MD[a], Valentina Ferrazzoli, MD[c,d], Francesca Di Giuliano, PhD, MD[c,d], Francesco Garaci, PhD, MD[a,c,d]*

[a] *Diagnostic Imaging Unit, University Hospital of Rome "Tor Vergata", Viale Oxford 81 00133, Rome, Italy*
[b] *Department of Systems Medicine, University of Rome "Tor Vergata", Via Montpellier 1 00133 Rome, Italy*
[c] *Department of Biomedicine and Prevention, University of Rome "Tor Vergata", Via Montpellier 1 00133 Rome, Italy*
[d] *Neuroradiology Unit, Department of Biomedicine and Prevention, University of Rome "Tor Vergata", Viale Oxford 81 00133, Rome, Italy*

ARTICLE INFO

Article history:
Received 3 May 2024
Revised 16 May 2024
Accepted 19 May 2024

Keywords:
Sinonasal mucormycosis
Cerebritis
Cavernous sinus thrombosis
Orbital cellulitis
Optic ischemia
MRI

ABSTRACT

Brain and ocular infections can be the worst and fatal consequences of sinonasal infections in immunomodulated or immunocompromised patients.

We report a case of a 35-year-old female who received an allogenic hematopoietic stem cell transplantation for acute myeloid leukemia, suffering from maxillo-spheno-ethmoidal rhinosinusitis which was complicated by cavernous sinus thrombosis, orbital cellulitis, optic ischemia and cerebritis.

Introduction

Mucormycosis is an infection caused by various fungal organisms in the order mucorales, including those belonging to the Rhizopus, Rhizomucor, and Mucor species, which can involve several organs [1]; in this contest the involvement of sinonasal cavities can be related to potentially lethal consequences due to the fungal angio-invasive properties with necrotic lesions spreading to brain and orbital tissues.

The most common site of infection is rhinocerebral, followed by cutaneous, lung, disseminated, and gastrointestinal tract [2].

Some aggravating features, such as immunosuppression of patients undergoing hematopoietic stem cell transplant (HSCT), can cause nasal mucosa inflammation to persist for a

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

* Corresponding author.
E-mail address: noemi.pucci@students.uniroma2.eu (N. Pucci).

https://doi.org/10.1016/j.radcr.2024.05.051

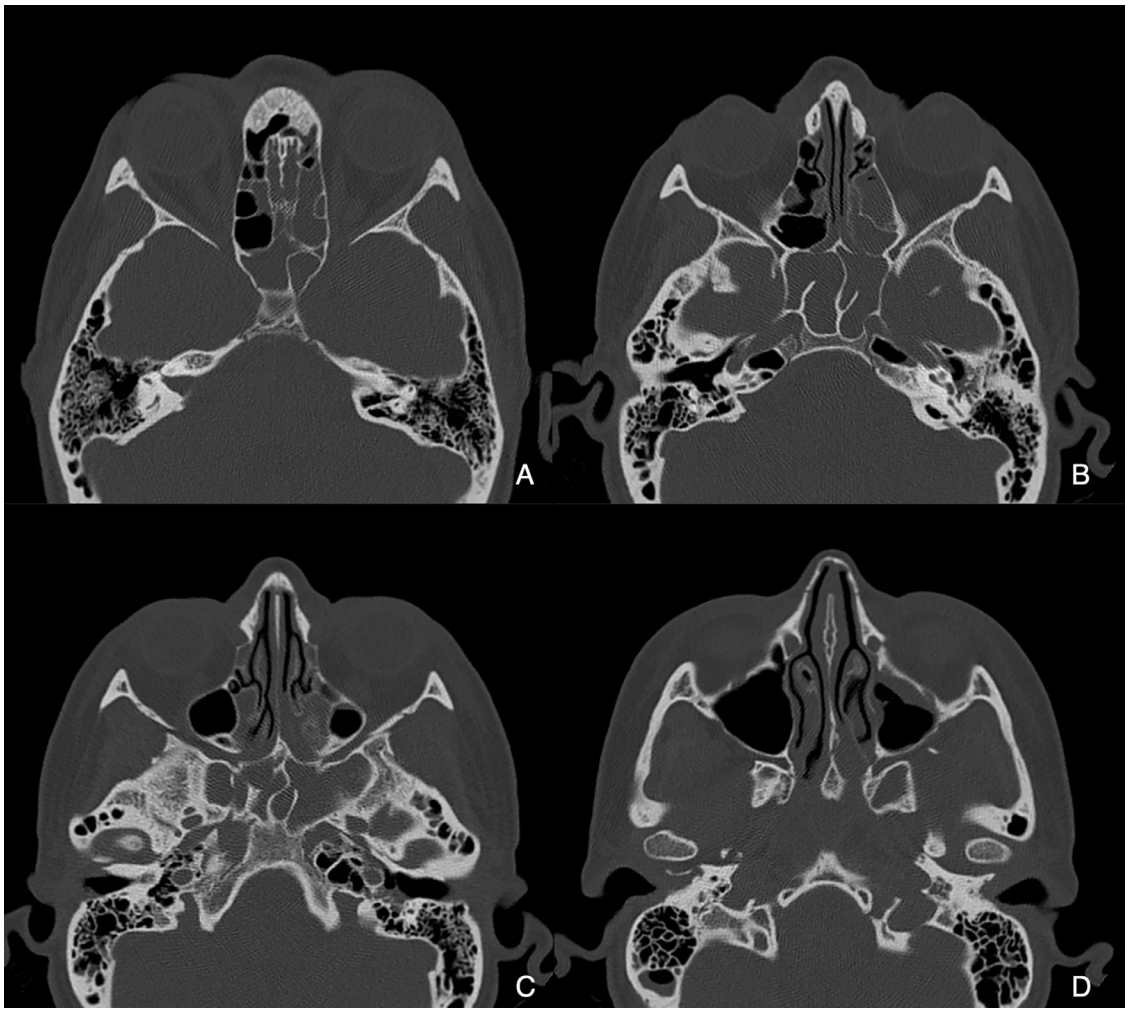

Fig. 1 – CT of the paranasal sinuses showed acute inflammation of the maxillary, ethmoidal, and sphenoidal sinuses without sign of bone erosion.

long time, resulting in chronic or recurrent episodes [3]. Other causes are trauma and neurosurgical intervention.

In this paper, we reported a case of cerebritis, optic ischemia, and cavernous sinus thrombosis arising from sinonasal mucormycosis in an immunosuppressed patient.

Case presentation

A 35-year-old woman with acute myeloid leukemia (AML), treated with allogeneic hematopoietic stem cell transplantation (HSCT) in January 2023, presented 1 week after graft at University Hospital of Rome Tor Vergata, with neutropenia and fever, despite antimicrobial therapy with meropenem. On examination she was alert and oriented toward the 3 spheres. She underwent computed tomography (CT) scan of the chest which did not reveal pathological conditions; otherwise, CT scan of the head and sinuses revealed acute inflammation of the maxillary, ethmoidal, and sphenoidal sinuses (Fig. 1). Patient continued intensive intravenous treatment with meropenem, vancomicina, and voriconazolo according to international protocols without clinical benefits. She was neutropenic and febrile up to 40°C daily, with the addition of episodes of frontal headache, occasional nausea, and a feeling of facial "fullness". After a few days, the fever had reduced significantly, with improvement in symptoms leading to the patient's discharge.

A couple of months later, during an outpatient follow-up, symptoms persisted, along with worsening mental status, increasing drowsiness, and left dull eye pain and swelling, exacerbated by eye movements, and associated with tearing and redness. During physical examination, the left eye showed significant eyelid edema with purulent discharge, proptosis, and chemosis, but clear cornea. She also presented ophthalmoplegia and the left pupil was not reactive to light. Examination of the right eye was unremarkable, with the right pupil reactive to light. Laboratory investigations showed a white blood cell count of 4000/mm^3, an absolute neutrophil count of 90 x $10^3/\mu L$, a hemoglobin of 10 g/dL, a platelet count of 25 $\times 10^9$ /L and elevated Lactate Dehydrogenase (LDH). A CT scan of the brain with contrast medium was performed showing a large area of cortico-subcortical hypodensity of the left frontal white matter, without hemorrhagic areas; left

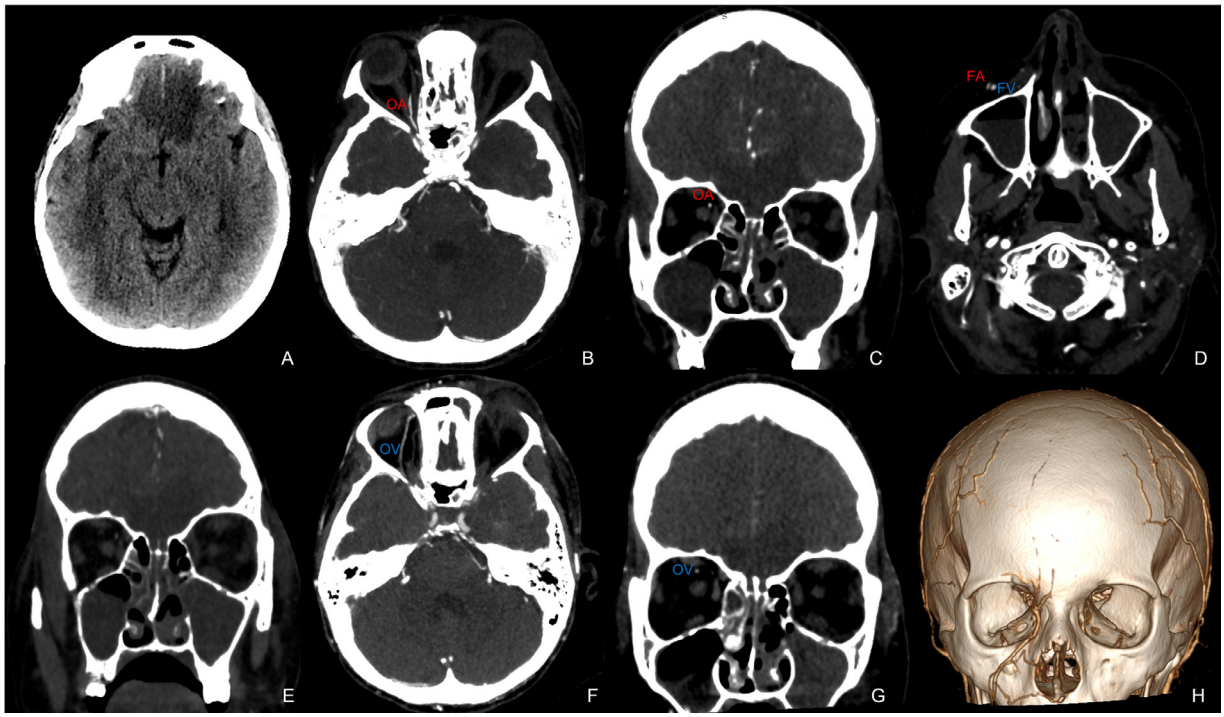

Fig. 2 – Skull CT showed large area of hypodensity of the left frontal white matter (A) along with maxillary sinusitis (E). Brain multiphasic Angio-CT scans highlighted opacification of only the right ophthalmic and facial artery and vein (B-H). *OA*, ophthalmic artery; *OV*, ophthalmic vein; *FA*, facial artery; *FV*, facial vein.

proptosis, orbital cellulitis, mild "globe tenting" and coexistence of left maxillary, ethmoidal and sphenoidal sinusitis were noted. Demineralization of ethmoid bone septae, nasal conchae, nasal septum and turbinates as well as medial wall of the maxillary synuses were also detected.

Brain multiphasic Angio-CT showed lack of opacification of the left ophthalmic and facial artery and vein (Fig. 2); no abnormal findings were detected along intracranial vessels. No area of contrast enhancement was revealed in brain CT delayed acquisition.

This examination was immediately followed by brain MRI with contrast agent performed using a high-field magnetic scanner (3 Tesla). The study revealed bilateral fronto-basal cortico-subcortical areas of restricted diffusion, more extensive on the left, involving the orbital and ipsilateral superior frontal gyri; a further area of restricted diffusion was revealed at cortico-subcortical left temporo-polar, extending to the level of the temporo-mesial hippocampal region and left hemiportion of pons. Restricted diffusion was also appreciated at the level of the left internal capsule, anteroventral nuclei of the ipsilateral thalamus and left fornix column. These changes were associated with swollen appearance of the cerebral gyri and white matter T2 hypersignal in whose context were detected multiple hypointensities in susceptibility weighted imaging (SWI). Abnormal signal was also detected along the left optic nerve, in keeping with ischemia, and it was associated with marked edema of the left orbital soft tissues, resulting in proptosis of the ipsilateral eyeball (Fig. 3). After contrast medium administration, minimal pachymeningeal thickening and enhancement was observed along the left frontal and temporal lobes. Extensive leptomeningeal enhancement was also appreciated at the frontal and left temporal lobes with involvement also of the left midbrain and ipsilateral pons. No intra-axial enhancement was observed, neither anular intra-axial lesions nor signs of perineural spread.

The left cavernous sinus did not show enhancement after contrast agent administration, findings in keeping with cavernous sinus thrombosis (Fig. 4).

These neuroradiological findings were highly suggestive for the diagnosis of brain angioinvasive fungal infection thus ambisome therapy was set up. Despite appropriate medical therapy and intensive medical treatment, the patient was unable to recover and led to her death. Essential for the diagnosis was the finding in the infected tissue, through autopsy examination, of hyphae of the mucormycosis fungus.

Postmortem diagnosis was obtained by analyzing brain tissue samples extracted from multiple restricted diffusion areas, as shown by MRI. The results of these samples were consistent with the presence of an invasive fungus, most likely a genus of Rhizopus spp; from the histopathological point of view, the harvested tissue showed the presence of pauciseptate hyphae with right angle branching, that had infiltrated both blood vessels and nerves and were surrounded by a strong neutrophilic inflammatory response.

Discussion

Invasive fungal infections are a major cause of morbidity and mortality in immunocompromised patients with hematologic

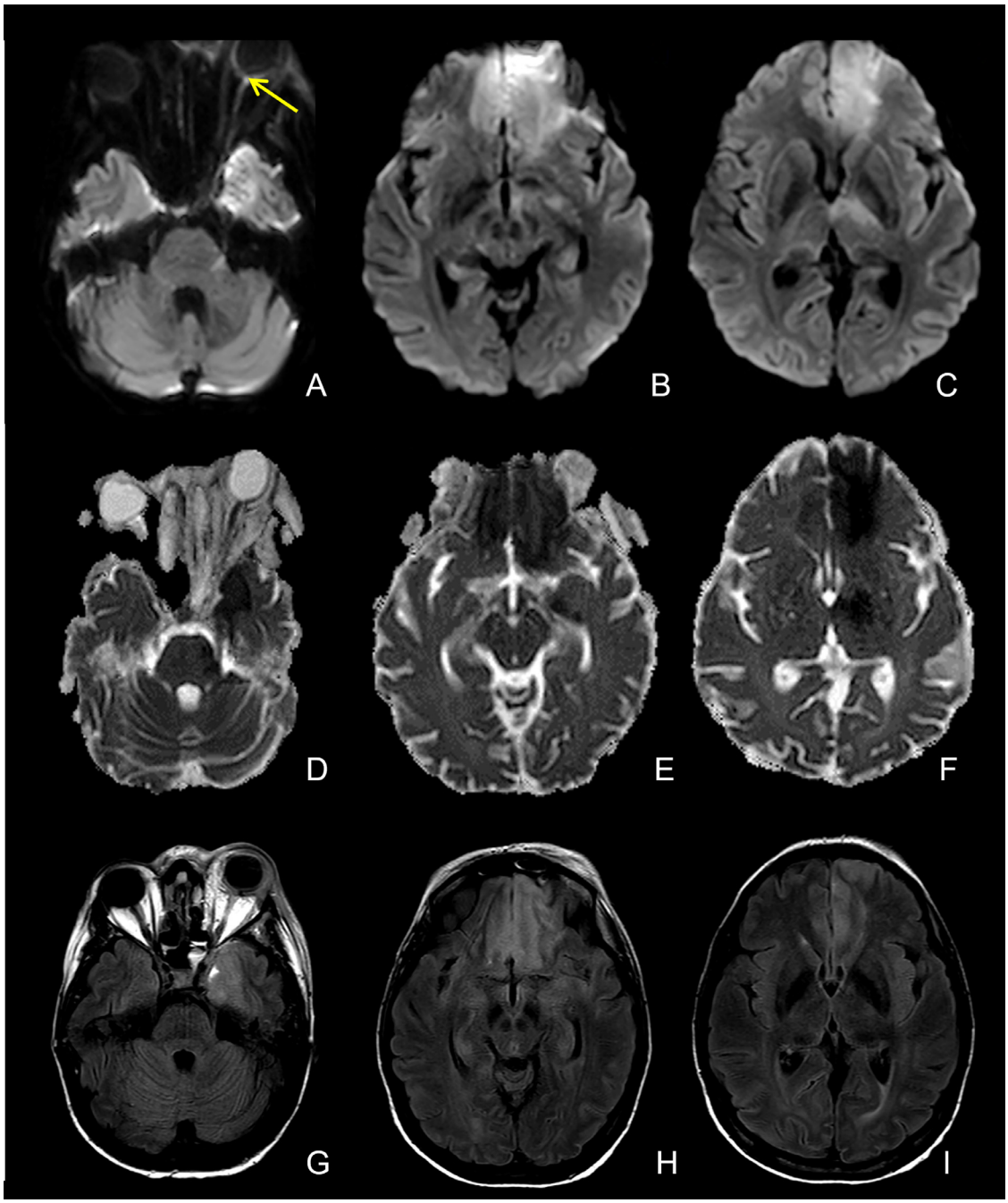

Fig. 3 – Axial DWI and apparent diffusion coefficient (ADC) sequences showed cortico-subcortical restricted diffusion at the fronto-basal site, more extensive on the left, extending to the orbital gyrus and superior frontal gyrus (B, C and E, F); at the left temporo-polar site, up to temporo-mesial hippocampal region, and sectorially at the left hemiportion of pons (A and D); restricted diffusion was also shown at the left internal capsule and anteroventral nuclei of the ipsilateral thalamus (C and F). Swollen appearance of the cerebral gyri and flattening of the furrows was appreciable at these sites. Ischemia was also detected along the left optic nerve, and it was associated with marked edema of the left orbital soft tissues, resulting in proptosis of the ipsilateral eyeball (A and D). These changes were associated with white matter T2 hypersignal in axial FLAIR images (G-I).

malignancies (HM), including those undergoing to HSCT in which central nervous system involvement is more likely [4,5].

Patients with HM and/or HSCT showed higher infection rates of mucormycosis than patients with solid organ transplant in particular if treated for graft versus-host disease (GVHD), too [2,6–10]. Even though the lung is the most common site of Mucormycosis, in hematologic patients the brain, skin, and digestive tract might be involved [11,12]; moreover, multi-organ infection has also been described as potentially fatal. Only few epidemiological studies allow estimation of

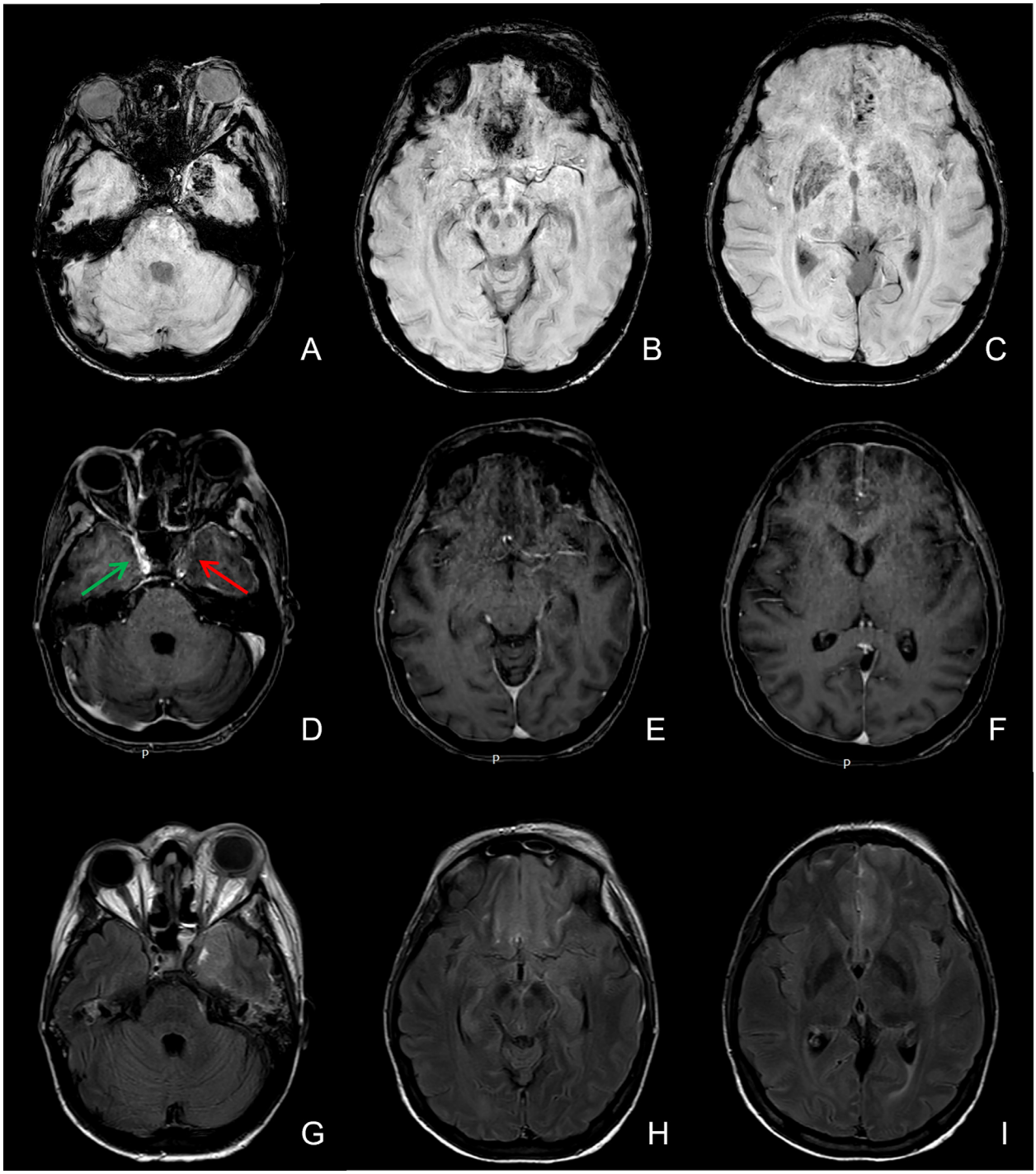

Fig. 4 – Axial SWI sequences identified multiple hypointensities at fronto-basal site and left temporo-polar site, suspicious for localization of fungal hyphae within brain tissue (A-C). Axial T1-weighted images and FLAIR sequences obtained after administration of Gadobutrolo denoted area of altered signal intensity in the context of the left cavernous sinus of suspected thrombotic nature (D and G) as well as pachymeningeal and leptomeningeal enhancement at the frontal and left temporal lobes (E-I).

the incidence of mucormycosis reporting higher risk in patients with AML [13,14]. Skiada et al reported lower incidence of mucormycosis in other acute or chronic HM; moreover, patients having HSCTs had a lower incidence of mucormycosis than those with AML [15–17]. Even though immunosuppression is a major factor in the condition, there are currently insufficient adequate denominators, making it impossible to accurately evaluate any trends in the disease's incidence. Essentially, invasive fungal rhinosinusitis, which has a lesser prevalence than bacterial rhinosinusitis, is more likely to occur in patients undergoing HSCT due to immunosuppression [18–21].

During the last COVID-19 pandemic a growing incidence of mucormycosis has been reported which could be related to steroid medications, elevated ferritin levels, mechanical ventilation, reusable oxygen humidifiers, and inadequate sanitation practices [22,23].

The prompt diagnosis of the disease, before intracranial and ocular dissemination, is necessary to avoid fatal evolution. Considering the severity and the rapid progression of angioinvasive fungal infections, the identification of early radiological changes is essential to set up the suspicion before the onset of clinical symptoms and to address the adequate therapy.

Several fungal and nonfungal neurological diseases might have similar neuroimaging appearances making differential diagnosis complex; however, the knowledge of specific predisposing factors could help the presumptive neuroradiological diagnosis.

In our report, the acute maxillary, ethmoidal, and sphenoidal sinusitis had been followed by cavernous sinus thrombosis resulting in cerebritis and optic ischemia in patient with AML after allogeneic HSCT. In this clinical *scenario*, the sinusitis and the selective involvement of the anterior cranial fossa structures could be related to Mucormycosis and/or aspergillosis infection due to angio-invasive properties of these fungal rhinosinusitis that allow direct extension to the central nervous system.

Otherwise, the hematogenous spread of the fungal infection in the CNS have wider brain involvement along the gray–white junction and basal ganglia while the fungal cerebrospinal fluid seeding, common with Cryptococcus and Aspergillus, show meningeal, ependymal, and choroidal structures engagement [5]. Although the well-know angioinvasive properties of Aspergillus and Mucormycoses allow large cerebral vessel their growth pattern make impossible the small peripheral or deep vessels involvement resulting in different neuroradiological findings [24,25].

Even though CT allows for the prompt diagnosis of rhinosinusitis the contrast media can add further information: as a matter of fact, the absence of mucosal contrast-enhancement might suggest the angio-invasive proprieties of the infection [26]. The lack of mucosal contrast-enhancement along the nasal turbinates is defined as “the black turbinate sign” and it can be found in immunocompetent and immunosuppressed patients without invasive fungal rhinosinusitis [27]; however, in patients with invasive fungal rhinosinusitis the infiltrative nonenhancement is not confined to the adjacent structures without smooth and thin enhancing margin.

On the other hand, orbital complications are more common than cerebral ones in sinusitis [28]. Usually, infections spread from the ethmoidal sinus to the orbit via the lamina papyracea, a slender bone structure that contains nerve endings and perforating arteries. Unenhanced CT reconstruction using the bone algorithm are necessary to look for bony dehiscence of the sinus walls, that may represent a red flag sign for intracranial involvement and suggest the need of further investigation with brain MR, which has a greater sensitivity for meningeal and intra-axial localization compared to brain CT.

Orbital cellulitis is more easily identified at CT, but when diagnosed it represents already a severe complication and it may rapidly progress to fatal implications if it is not treated effectively, since the venous drainage system in this region is less valved and permits connection between the sinuses and orbit [29]. The progression of sinonasal orbital infections was categorized into five phases following Chandler classification [30,31]:

- preseptal cellulitis (stage I),
- orbital cellulitis (stage II),
- subperiosteal abscess (stage III),
- orbital abscess (stage IV),
- cavernous sinus thrombosis (stage V).

The features of orbital infection, such as proptosis, intramuscular and orbital fat oedema, lateral displacement, and thickness of the medial rectus muscle, are better depicted by MRI. Cavernous sinus thrombosis is typically related to retrograde thrombophlebitis during aggressive sinusitis and can lead to cerebritis. Cavernous sinus thrombosis might bring to retinal artery filling deficiency with optic nerve ischemia [32]. Spike fevers, orbital discomfort, and worsening headache may be followed by seizures, proptosis, ophthalmoplegia, chemosis, impaired visual acuity, and cranial nerve palsies, including III, IV, V1, V2, and VI nerves, which are characteristic symptoms of cavernous sinus thrombosis. The most involved nerve is the VI since it is entirely enclosed within the sinus [33–35]. Although CT and CT angiography are useful in the initial work up of suspected cavernous sinus thrombosis, MRI is indicated to confirm the diagnosis. Findings of cavernous sinus thrombosis include the absence of venous flow in the affected cavernous sinus, and narrowing or occlusion of the intracavernous segment of the internal carotid artery [32].

Cerebritis may occur for hematogenous spread, direct posterior sinonasal spread, perineural spread, after trauma or as neurosurgical complication; however, from 15% to 30% of brain infections are classified as cryptogenic if the source cannot be found [36–39]. Cerebritis presents as an indistinct region of acute inflammation in the brain tissue characterized by enhanced blood vessel permeability in the absence of neoangiogenesis [40,41].

Many microorganisms, such as pyogenic bacteria, can cause cerebritis, which, if left untreated, can lead to the development of a pyogenic brain abscess [42]. MRI might suggest some features which are able to differentiate pyogenic from fungal brain abscess with the last characterized by more irregular walls, intracavitary projections, more heterogenous and higher apparent diffusion coefficient in the abscess cavity. Moreover, the wall of fungal abscess might have a more pronounced T2 hypointensity which is related to the increased iron molecules. Magnetic resonance spectroscopy seems to provide additional informations and features of fungal abscess which beside the well-known lactate, lipid, and amino acids metabolism revealed several peaks between 3.6 and 3.8 ppm suitable with trehalose which has been reported in some fungal abscess, findings best appreciated with 3T MR scanner [43–45].

CT and MRI have become essential diagnostic tools for the evaluation and management of complicated sinusitis [46]. Although MRI is required if intracranial extension is suspected, CT remains the standard initial method for diagnosing sinusitis or complicated sinusitis [46].

Conclusion

Mucormycosis is a rare emerging fungal infection with a high mortality rate; the variability of clinical manifestations and its elusive presentation are often the cause of a delayed diagnosis that may result in fatal outcomes. A high index of suspicion for mucormycosis, based on appropriate risk stratification, neuroradiological findings, and improved laboratory diagnosis are important to treat the natural history of this devastating disease. Contrast-enhanced imaging studies (CT/MRI) play a critical role in the early diagnostic process of rhinocerebral mucormycosis, contributing to the evaluation of vascular complications, which are at the roots of the lethality of this disease.

Patient consent

An informed consent was obtained for publication of this case report.

REFERENCES

[1] Chung JH, Godwin JD, Chien JW, Pipavath SJ. Case 160: pulmonary mucormycosis. Radiology 2010;256(2):667–70. doi:10.1148/radiol.10081907.

[2] 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.

[3] Ortiz E, Altemani A, Vigorito AC, Sakano E, Nicola EMD. Rhinosinusitis in hematopoietic stem cell-transplanted patients: Influence of nasosinus mucosal abnormalities? Stem Cell Res Ther 2014;5(6):6–10. doi:10.1186/scrt523.

[4] Neofytos D, Horn D, Anaissie E, Steinbach W, Olyaei A, Fishman J, et al. Epidemiology and outcome of invasive fungal infection in adult hematopoietic stem cell transplant recipients: analysis of multicenter prospective antifungal therapy (PATH) alliance registry. Clin Infect Dis 2009;48(3):265–73. doi:10.1086/595846/2/48-3-265-FIG004.GIF.

[5] Starkey J, Moritani T, Kirby P. MRI of CNS fungal infections: review of aspergillosis to histoplasmosis and everything in between. Clin Neuroradiol 2014;24(3):217–30. doi:10.1007/S00062-014-0305-7/METRICS.

[6] Park BJ, Pappas PG, Wannemuehler KA, D'Alessandro B, Anaissie EJ, Andes DR, et al. Invasive non-aspergillus mold infections in transplant recipients, United States, 2001-2006. Emerg Infect Dis 2011;17(10):1855–64. doi:10.3201/eid1710.110087.

[7] Baddley JW, Stroud TP, Salzman D, Pappas PG. Invasive mold infections in allogeneic bone marrow transplant recipients. Clin Infect Dis 2001;32(9):1319–24. doi:10.1086/319985.

[8] Almyroudis NG, Sutton DA, Linden P, Rinaldi MG, Fung J, Kusne S. Zygomycosis in solid organ transplant recipients in a tertiary transplant center and review of the literature. Am J Transplant 2006;6(10):2365–74. doi:10.1111/j.1600-6143.2006.01496.x.

[9] Pappas PG, D' Alessandro B, Andes DR, Hadley S, Kauffmann CA, Freifeld A, et al. Invasive fungal infections among organ transplant recipients: results of the transplant-associated infection surveillance network (Transnet). Clin Infect Dis 2010;50(8):1101–11. doi:10.1086/651262.

[10] Lanternier F, Sun HY, Ribaud P, Singh N, Kontoyiannis DP, Lortholary O. Mucormycosis in organ and stem cell transplant recipients. Clin Infect Dis 2012;54(11):1629–36. doi:10.1093/cid/cis195.

[11] Rüping MJGT, Heinz WJ, Kindo AJ, Rickerts V, Lass-Flörl C, Beisel C, et al. Forty-one recent cases of invasive zygomycosis from a global clinical registry. J Antimicrob Chemother 2010;65(2):296–302. doi:10.1093/JAC/DKP430.

[12] Skiada A, Pagano L, Groll A, Zimmerli S, Dupont B, Lagrou K, et al. Zygomycosis in Europe: analysis of 230 cases accrued by the registry of the European Confederation of Medical Mycology (ECMM) Working Group on Zygomycosis between 2005 and 2007. Clin Microbiol Infect 2011;17(12):1859–67. doi:10.1111/j.1469-0691.2010.03456.x.

[13] Pagano L, Ricci P, Tonso A, Nosari A, Cudillo L, Montillo M, et al. Mucormycosis in patients with haematological malignancies: a retrospective clinical study of 37 cases. Br J Haematol 1997;99(2):331–6. doi:10.1046/j.1365-2141.1997.3983214.x.

[14] Lanternier F, Dannaoui E, Morizot G, Elie C, Garcia-Hermoso D, Huerre M, et al. A global analysis of mucormycosis in France: the RetroZygo study (2005-2007). Clin Infect Dis 2012;54(1):35–43. doi:10.1093/cid/cir880.

[15] Marr KA, Carter RA, Crippa F, Wald A, Corey L. Epidemiology and outcome of mould infections in hematopoietic stem cell transplant recipients. Clin Infect Dis 2002;34(7):909–17. doi:10.1086/339202.

[16] Kontoyiennis DP, Marr KA, Park BJ, D'Alessandro B, Anaissie EJ, Walsh TJ, et al. Prospective surveillance for invasive fungal infections in hematopoietic stem cell transplant recipients, 2001-2006: overview of the transplant-associated infection surveillance network (TRANSNET) database. Clin Infect Dis 2010;50(8):1091–100. doi:10.1086/651263.

[17] Skiada A, Lanternier F, Groll AH, Pagano L, Zimmerli S, Herbrecht R, et al. Diagnosis and treatment of mucormycosis in patients with hematological malignancies: guidelines from the 3rd European Conference on Infections in Leukemia (ECIL 3). Haematologica 2013;98(4):492–504. doi:10.3324/haematol.2012.065110.

[18] Won YW, Yi SY, Jang JH, Kihyun K, Seok JK, Seog K, et al. Retrospective analysis of paranasal sinusitis in patients receiving hematopoietic stem cell transplantation. Int J Hematol 2011;93(3):383–8. doi:10.1007/s12185-011-0797-8.

[19] Verschraegen CF, Van Besien KW, Dignani C, Hester JP, Andersson BS, Anaissie E. Invasive aspergillus sinusitis during bone marrow transplantation. Scand J Infect Dis 1997;29(4):436–8. doi:10.3109/00365549709011849.

[20] Parikh SL, Venlkatraman G, Del Gaudio JM. Invasive fungal sinusitis: a 15-year review from a single institution. Am J Rhinol 2004;18(2):75–81. doi:10.1177/194589240401800202.

[21] Johnson PJ, Lydiatt WM, Huerter JV, Ogren FP, Vose JM, Stratta RJ, et al. Invasive fungal sinusitis following liver or bone marrow transplantation. Am J Rhinol 1994;8(2):77–83. doi:10.2500/105065894781874485.

[22] Sarda R, Swain S, Ray A, Wig N. COVID-19-Associated mucormycosis: an epidemic within a pandemic. QJM 2021;114(6):355–6. doi:10.1093/qjmed/hcab165.

[23] John TM, Jacob CN, Kontoyiannis DP. When uncontrolled diabetes mellitus and severe covid-19 converge: the perfect storm for mucormycosis. J Fungi 2021;7(4):298. doi:10.3390/jof7040298.

[24] Mathur M, Johnson CE, Sze G. Fungal infections of the central nervous system. Neuroimaging Clin N Am 2012;22(4):609–32. doi:10.1016/J.NIC.2012.04.004.

[25] Allen LM, Fowler AM, Walker C, Derdeyn CP, Nguyen BV, Hasso AN, et al. Retrospective review of cerebral mycotic aneurysms in 26 patients: focus on treatment in strongly immunocompromised patients with a brief literature review. Am J Neuroradiol 2013;34(4):823–7. doi:10.3174/AJNR.A3302.
[26] Safder S, Carpenter JS, Roberts TD, Bailey N. The "black turbinate" sign: an early MR imaging finding of nasal mucormycosis. Am J Neuroradiol 2010;31(4):771–4. doi:10.3174/ajnr.A1808.
[27] Han Q, Escott EJ, Han XQ, Escott XEJ. The black turbinate sign, a potential diagnostic pitfall: evaluation of the normal enhancement patterns of the nasal turbinates. Am J Neuroradiol 2019;40(5):855–61. doi:10.3174/AJNR.A6037.
[28] Germiller JA, Monin DL, Sparano AM, Tom LWC. Intracranial complications of sinusitis in children and adolescents and their outcomes. Arch Otolaryngol–Head Neck Surg 2006;132(9):969–76. doi:10.1001/ARCHOTOL.132.9.969.
[29] Lee S, Yen MT. Management of preseptal and orbital cellulitis. Saudi J Ophthalmol 2011;25(1):21–9. doi:10.1016/J.SJOPT.2010.10.004.
[30] Dankbaar JW, van Bemmel AJM, Pameijer FA. Imaging findings of the orbital and intracranial complications of acute bacterial rhinosinusitis. Insights Imaging 2015;6(5):509–18. doi:10.1007/S13244-015-0424-Y/FIGURES/14.
[31] Radovani P, Vasili D, Xhelili M, Dervishi J. Orbital complications of sinusitis. Balkan Med J 2013;30(2):151–4. doi:10.5152/BALKANMEDJ.2013.8005.
[32] Mazzai L, Anglani M, Giraudo C, Martucci M, Cester G, Causin F. Imaging features of rhinocerebral mucormycosis: from onset to vascular complications. Acta Radiol 2022;63(2):232–44. doi:10.1177/0284185120988828.
[33] Ziegler A, Patadia M, Stankiewicz J. Neurological complications of acute and chronic sinusitis. Curr Neurol Neurosci Rep 2018;18(2):1–8. doi:10.1007/S11910-018-0816-8/METRICS.
[34] Chaurasia D, Yadav B, Dhungana K. Dural venous sinus thrombosis: a case report. J Nepal Med Assoc 2021;59(244):1316–19. doi:10.31729/JNMA.7170.
[35] Singh V. Fungal rhinosinusitis: unravelling the disease spectrum. J Maxillofacial Oral Surg 2019;18(2):164–79. doi:10.1007/S12663-018-01182-W/FIGURES/5.
[36] Mamelak AN, Mampalam TJ, Obana WG, Rosenblum ML. Improved management of multiple brain abscesses: a combined surgical and medical approach. Neurosurgery 1995;36(1):76–86.
[37] Kragha KO. Multiple brain abscesses due to Streptococcus anginosus: prediction of mortality by an Imaging Severity Index Score. Case Rep Radiol 2016;2016:1–8. doi:10.1155/2016/7040352.
[38] Nam TK, Park YS, Kwon JT. Brain abscesses associated with asymptomatic pulmonary arteriovenous fistulas. J Korean Neurosurg Soc 2017;60(1):118–24. doi:10.3340/jkns.2015.0707.023.
[39] Lersy F, Royer-Leblond J, Lhermitte B, Chammas A, Schneider F, Hansmann Y, et al. Cerebral mucormycosis: neuroimaging findings and histopathological correlation. J Neurol 2022;269(3):1386–95. doi:10.1007/S00415-021-10701-8/METRICS.
[40] Manchanda S, Semalti K, Bhalla AS, Thakar A, Sikka K, Verma H. Revisiting rhino-orbito-cerebral acute invasive fungal sinusitis in the era of COVID-19: pictorial review. Emerg Radiol 2021;28(6):1063–72. doi:10.1007/s10140-021-01980-9.
[41] Hallak B, Bouayed S, Ghika JA, Teiga PS, Alvarez V. Management strategy of intracranial complications of sinusitis: our experience and review of the literature. Allergy Rhinol 2022;13(9). doi:10.1177/21526575221125031.
[42] Rath TJ, Hughes M, Arabi M, Shah G V. Imaging of cerebritis, encephalitis, and brain abscess. Neuroimaging Clin N Am 2012;22(4):585–607. doi:10.1016/j.nic.2012.04.002.
[43] Mueller-Mang C, Castillo M, Mang TG, Cartes-Zumelzu F, Weber M, Thurnher MM. Fungal versus bacterial brain abscesses: is diffusion-weighted MR imaging a useful tool in the differential diagnosis? Neuroradiology 2007;49(8):651–7. doi:10.1007/S00234-007-0242-0/METRICS.
[44] Luthra G, Parihar A, Nath K, Jaiswal S, Prasad KN, Husayn N, et al. Comparative evaluation of fungal, tubercular, and pyogenic brain abscesses with conventional and diffusion MR imaging and proton MR spectroscopy. AJNR 2007;28(7):1332. doi:10.3174/AJNR.A0548.
[45] Kamalapur MG, Patil PB, Joshi SK, Sateesh M, Patil SH, SubramaniamAG, et al. Role of magnetic resonance imaging in distinguishing fungal from nonfungal multiple brain abscesses. Egypt J Radiol Nucl Med 2011;42(3–4):397–404. doi:10.1016/J.EJRNM.2011.06.008.
[46] Younis RT, Anand VK, Davidson B. The role of computed tomography and magnetic resonance imaging in patients with sinusitis with complications. Laryngoscope 2002;112(2):224–9. doi:10.1097/00005537-200202000-00005.