Pons Infection Due To Disseminated Aspergillus

María Paula Rojas-Arrieta, MD[1,2], Josue Moreno-Moreno[3] , Ricardo Buitrago-Bernal, MD[2,4] and Julian Orlando Casallas-Barrera MD[2]*

[1]*Resident in Critical Care Medicine, Universidad de la Sabana, Chía, Colombia*

[2]*Department of Critical Care and Intensive Care Medicine, Fundación Clínica Shaio, Bogotá, Colombia*

[3]*Department of Neurology, Fundación Clínica Shaio, Bogotá, Colombia*

[4]*Exploratorium group, Fundación Clínica Shaio, Bogotá, Colombia*

***Correspondence:** Julian Orlando Casallas-Barrera, M.D., 2Department of Critical Care and Intensive Care Medicine, Fundación Clínica Shaio, Bogotá, Colombia

julian.casallas@shaio.org

Radiology Case. 2024 September; 18(9):63-70 :: DOI: 10.3941/jrcr.5274

ABSTRACT

A 61-year-old patient with a history of hypertension and diabetes mellitus was admitted with newly developed heart failure, acute kidney injury, and nephrotic syndrome. The patient is suspected of being immunocompromised by a hematologic malignancy. Subsequently, developed pulmonary sepsis and neurological deterioration. Neuroimaging revealed diffuse signal abnormalities in the pons with symmetric involvement, but no signs of restricted diffusion. Despite targeted antibiotic therapy, the patient's condition did not improve, and systemic inflammation persisted. *Aspergillus fumigatus* was isolated from bronchoalveolar lavage and blood cultures, confirming hematogenous dissemination. Cerebrospinal fluid showed *hypoglycorrhachia*, hyperproteinorrhachia and a positive galactomannan. Follow-up brain Magnetic Resonance Imaging showed progression of edema and restricted diffusion, indicating disseminated aspergillosis involving the pons. The patient's condition worsened, leading to multiple organ dysfunction syndrome and death.

Journal of Radiology Case Reports

www.RadiologyCases.com

CASE REPORT

CASE REPORT

A 61-year-old patient with a medical history of hypertension and diabetes mellitus was admitted due to newly developed heart failure, acute kidney injury, and a nephrotic syndrome. The patient was suspected to be immunocompromised by a hematologic malignancy. Subsequently, developed pulmonary sepsis. Additionally, there is neurological involvement. Initial neuroimaging, including a brain magnetic resonance imaging (MRI) (Figure 1), revealed diffuse areas of increased signal intensity in the pons with symmetric involvement. There were no signs of restricted diffusion or significant involvement of any specific structure (Figure 2). Despite the patient received targeted antibiotic treatment, there was no improvement, and systemic inflammatory response persisted. A fungal screening was conducted. *Aspergillus fumigatus* was isolated from both bronchoalveolar lavage and blood cultures, indicating hematogenous dissemination. The analysis of cerebrospinal fluid (CSF) showed normal glycorrhachia (60 mg/dL), elevated protein (159 mg/dL), and acellularity. Microbiological tests, including culture, Cryptococcus antigen, and multiplex PCR, were negative. However, galactomannan was positive in CSF, serum, and BAL, suggesting systemic fungal involvement, likely Aspergillus.

A follow-up contrast brain MRI showed the progression of edema in the pons, extending to the midbrain, the diencephalic-mesencephalic junction, cerebellar peduncles, deep regions of both hemispheres, the bulb, and the medullary cord (Figure 3). Multiple areas of restricted diffusion were also observed in the cerebellum and brainstem (Figure 4). Disseminated aspergillosis with involvement of the pons was diagnosed. Despite receiving appropriate antifungal treatment with voriconazole and amphotericin B, the patient's clinical condition continued to deteriorate, leading to multiple organ dysfunction syndrome and death.

DISCUSSION

Etiology and Demographics

This report describes a case of a patient with multiple comorbidities in whom aspergillosis of the central nervous system (CNS) was diagnosed, with an unusual location in the brainstem. Invasive aspergillosis is the most common mold infection [1]. Immunocompromised conditions, such as leukemia, stem cell transplantation, HIV/AIDS, and corticosteroid use, are common risk factors [2–5]. CNS involvement of *Aspergillus* is a rare presentation, more frequently observed in immunocompromised patients [6]. In immunocompetent individuals, predisposing factors such as type 2 diabetes mellitus, sinusitis, gingivitis, mastoiditis, or recent neurosurgeries have been described [7]. Achieving a complete diagnosis of CNS aspergillosis is not easy [2]. The clinical suspicion from imaging should be correlated with a tissue biopsy or biomarker testing, although the latter

(PCR, galactomannan, 1,3-beta-D-glucan) is only approved for use in serum [1,2]. In our knowledge, this is the first case report of *Aspergillus fumigatus* as the etiology of pontine infection.

Clinical and Imaging Findings

For topographic characterization in the CNS, neuroimaging is required, which may include a cranial CT scan or contrast-enhanced MRI. Advanced imaging techniques, such as diffusion-weighted (DWI) MRI, can help in assessing the extent of infection and guiding treatment decisions for patients with suspected CNS involvement. Although there are no specific radiological findings for diagnosis, dural enhancement secondary to extension from the paranasal sinuses and bone erosion are the most common signs [6]. The lesions are usually intraxial and supratentorial. However, when the source of dissemination is hematogenous, multiple lesions may appear at the gray-white matter junction at the lobar level, knowing that vascular involvement may lead to infarctions or intraparenchymal hemorrhages [6]. Mardari et al. described a patient with pontocerebellar angle aspergillosis with an MRI showing a ring-enhancing left pontocerebellar mass [8]. Also, a pontine hemorrhage was described in an older patient with meningitis by *Aspergillus* [9]. In immunocompetent patients, CNS aspergillosis may present with distinct imaging features such as hypointense T2-weighted lesions and hyperdense masses on CT scans, which can help differentiate it from other conditions [10]. In this case, MRI showed a FLAIR sequence with edema in the midbrain, diencephalic-mesencephalic junction, cerebellar peduncles, deep region of both hemispheres, bulb, and medullary cord. The DWI showed multiple foci of high signal intensity in the cerebellum and stem. To our knowledge, this is the first case reporting an involvement of midbrain, diencepahalic-mesencephalic junction, cerebellar peduncles, bulb and medullary cord.

Differential Diagnosis

CNS aspergillosis is a rare but often fatal fungal infection. It presents with non-specific clinical features, making its diagnosis challenging. Common neurological symptoms of this illness include headaches, altered mental status, seizures, and focal neurological deficits [2–4]. A crucial diagnostic procedure for etiological identification of infections of the CNS is lumbar puncture (LP). As described above, there are no typical neuroimaging findings, so this entity can mimic other conditions such as tumors or bacterial abscesses [3, 10, 11]**.** Key diagnostic findings for CNS aspergillosis in LP include pleocytosis and elevated protein levels in CSF [12]. Positive results from a galactomannan assay, which identifies a component of the Aspergillus cell wall, are significant for early diagnosis [13]. Polymerase chain reaction (PCR) assays can also detect Aspergillus DNA in the CSF, providing a definitive diagnosis [14]. Additionally, microscopic examination of the CSF may reveal fungal hyphae, which indicate the presence of Aspergillus infection [13, 14]. Table 2 summarizes the differential diagnosis of this entity.

Treatment and Prognosis

Treatment of cerebral aspergillosis includes surgical management to remove infected tissue (bone, paranasal sinuses, and brain abscess) and antifungal therapy [15]. Voriconazole has been described as the primary treatment for CNS aspergillosis [16–18]. Studies have shown that voriconazole alone or in combination with surgery could have radiological resolution and improved survival rates [17,18]. Treatment efficacy can be increased by combining voriconazole with other antifungal medications such as liposomal amphotericin B or echinocandins; nevertheless, voriconazole and amphotericin B together appear to be the most promising combination [19,20]. In addition to antifungal therapy, reducing or reversing immunosuppression should be considered whenever possible.

Despite effective antifungal treatment, the mortality rate can reach up to 33% in immunocompromised patients [21]. In this case, other negative outcome predictors present were galactomannan positivity, disseminated disease, renal failure, and CNS involvement [22].

TEACHING POINT

Aspergillosis of the CNS is a rare condition with a high mortality rate. Given the challenges in diagnosis, this case underscores the importance of considering topographic involvement through MRI neuroimaging and highlights distinct findings compared to those previously reported in the literature, to facilitate timely diagnosis and treatment.

QUESTIONS

Question 1: Which of the following is a common risk factor for central nervous system aspergillosis?

1. Hypertension
2. Immunocompromised conditions (e.g., leukemia, HIV/AIDS) [applies]
3. Hyperlipidemia
4. Chronic obstructive pulmonary disease (COPD)
5. Vitamin D deficiency

Explanation:

1. Hypertension is not a risk factor. [Immunocompromised conditions, such as leukemia, stem cell transplantation, HIV/AIDS, and corticosteroid use, are common risk factors]

2. Immunocompromised conditions (e.g., leukemia, HIV/AIDS) [Immunocompromised conditions, such as leukemia, stem cell transplantation, HIV/AIDS, and corticosteroid use, are common risk factors].

3. Hyperlipidemia is not a risk factor. [Immunocompromised conditions, such as leukemia, stem cell transplantation, HIV/AIDS, and corticosteroid use, are common risk factors].

4. Chronic obstructive pulmonary disease (COPD) is not a risk factor. [Immunocompromised conditions, such as leukemia, stem cell transplantation, HIV/AIDS, and corticosteroid use, are common risk factors]

5. Vitamin D deficiency is not a risk factor. [Immunocompromised conditions, such as leukemia, stem cell

Journal of Radiology Case Reports
www.RadiologyCases.com

transplantation, HIV/AIDS, and corticosteroid use, are common risk factors].

Question 2: What is a significant imaging finding in CNS aspergillosis related to hematogenous dissemination?

1. Bone erosion [applies]
2. Multiple lesions at the gray-white matter junction [applies]
3. Single large lesion in the cerebellum [applies]
4. Diffuse dural enhancement [applies]
5. Hypontense lesions on T1-weighted MRI

Explanation:

1. Bone erosion. [Dural enhancement secondary to extension from the paranasal sinuses and bone erosion are the most common signs].
2. Multiple lesions at the gray-white matter junction when the source of dissemination is hematogenous. [Multiple lesions may appear at the gray-white matter junction at the lobar level].
3. Restricted diffusion in the cerebellum. [Multiple areas of restricted diffusion were also observed in the cerebellum and brainstem].
4. Diffuse dural enhancement. [Dural enhancement secondary to extension from the paranasal sinuses and bone erosion are the most common signs].
5. Hypointense lesions on T1-weighted MRI has not been described. [T2-fluid-attenuated inversion recovery (FLAIR) with confluent hyperintensities in pons with symmetric involvement]

Question 3: Which diagnostic method is considered crucial for confirming CNS aspergillosis?

1. Complete blood count.
2. Electroencephalogram.
4. Lumbar puncture with CSF analysis [applies].
4. Skin biopsy
5. Cardiac ultrasound

Explanation:

1. Complete blood count has not specific findings. [Key diagnostic findings for CNS aspergillosis in LP include pleocytosis and elevated protein levels in CSF. Positive results from a galactomannan assay, which identifies a component of the Aspergillus cell wall, are significant for early diagnosis].
2. Electroencephalogram has not described as a diagnosis tool of SNC aspergillosis. [Key diagnostic findings for CNS aspergillosis in LP include pleocytosis and elevated protein levels in CSF. Positive results from a galactomannan assay, which identifies a component of the Aspergillus cell wall, are significant for early diagnosis].
4. Lumbar puncture with CSF analysis [applies]. [Key diagnostic findings for CNS aspergillosis in LP include pleocytosis and elevated protein levels in CSF. Positive results from a galactomannan assay, which identifies a component of the Aspergillus cell wall, are significant for early diagnosis].
4. Skin biopsy is not a diagnosis tool. [Key diagnostic findings for CNS aspergillosis in LP include pleocytosis and elevated protein levels in CSF. Positive results from a galactomannan assay, which identifies a component of the Aspergillus cell wall, are significant for early diagnosis].
5. Cardiac ultrasound is not used for CNS aspergillosis. [Key diagnostic findings for CNS aspergillosis in LP include pleocytosis and elevated protein levels in CSF. Positive results from a galactomannan assay, which identifies a component of the Aspergillus cell wall, are significant for early diagnosis].

Question 4: Which antifungal medication is primarily used for treating CNS aspergillosis?

1. Fluconazole
2. Voriconazole [applies].
3. Itraconazole
4. Anphotericin B [applies].
5. Echinocandins [applies].

Explanation:

1. Fluconazole is not a treatment option. [Voriconazole has been described as the primary treatment for CNS aspergillosis]
2. Voriconazole. [Voriconazole has been described as the primary treatment for CNS aspergillosis]
3. Itraconazole not a treatment option. [Voriconazole has been described as the primary treatment for CNS aspergillosis]
4. Anphotericin B. [Treatment efficacy can be increased by combining voriconazole with other antifungal medications such as liposomal amphotericin B or echinocandins].
5. Echinocandins. [Treatment efficacy can be increased by combining voriconazole with other antifungal medications such as liposomal amphotericin B or echinocandins].

Question 5: What factor is associated with poor prognosis in patients with CNS aspergillosis?

1. Immunocompetence
2. Absence of comorbidities
3. Positive galactomannan assay results. [applies]
4. Disseminated disease. [applies]
5. Limited CNS involvement

Explanation:

1. Immunocompetence is not a poor prognosis factor. [Other negative outcome predictors present were galactomannan positivity, disseminated disease, renal failure, and CNS involvement]
2. Absence of comorbidities is not a poor prognosis factor. [Other negative outcome predictors present were galactomannan positivity, disseminated disease, renal failure, and CNS involvement]
3. Positive galactomannan assay results. [Other negative outcome predictors present were galactomannan positivity, disseminated disease, renal failure, and CNS involvement]
4. Disseminated disease. [Other negative outcome predictors present were galactomannan positivity, disseminated disease, renal failure, and CNS involvement]
5. Limited CNS involvement is not a poor prognosis factor. [Other negative outcome predictors present were galactomannan positivity, disseminated disease, renal failure, and CNS involvement].

Journal of Radiology Case Reports

www.RadiologyCases.com

ACKNOWLEDGMENTS

To the CRitical carE investigATors IN Group (CREATING)

AUTHOR CONTRIBUTIONS

All authors have read and approved of the final version of manuscript. MPRA and JMM: Conceptualization, writing–review and editing. RBB: Conceptualization, writing–original draft, visualization, supervision, and project administration. JOCB: Conceptualization, investigation, writing–review and editing, supervision, and project administration.

DISCLOSURES AND FUNDINGS

Open access funding provided by Fundación Clínica Shaio, Bogotá-Colombia.

INFORMED CONSENT STATEMENT

Written informed consent has been obtained from the involved patient.

ETHICAL APPROVAL

This study adhered to ethical requirements. It was approved by the Ethics committee in research of the Fundación Abood Shaio (ID approval DIB-24-05).

REFERENCES

1. Boucher HW, Patterson TF. Aspergillosis. 2023; 183–196. [10.1007/978-3-031-35803-6_11]
2. Meena DS, Kumar D, Bohra GK, Kumar G. Clinical manifestations, diagnosis, and treatment outcome of CNS aspergillosis: A systematic review of 235 cases. *Infect Dis Now.* 2021; 51(8): 654-660. PMID: 33964485.
3. Shariati A, Didehdar M, Rajaeih S, et al. Aspergillosis of central nervous system in patients with leukemia and stem cell transplantation: a systematic review of case reports. *Ann Clin Microbiol Antimicrob.* 2021; 20(1): 44. PMID: 34130699.
4. Kleinschmidt-DeMasters BK. Central nervous system aspergillosis: A 20-year retrospective series. *Hum Pathol.* 2002; 33(1): 116–124. PMID: 11823982.
5. Mylonakis E, Paliou M, Sax PE, Skolnik PR, Baron MJ, Rich JD. Central nervous system aspergillosis in patients with human immunodeficiency virus infection. Report of 6 cases and review. *Medicine.* 2000; 79(4): 269–280. PMID: 10941356.
6. Miceli MH. Central nervous system infections due to aspergillus and other hyaline molds. *J Fungi (Basel).* 2019; 5(3): 79. PMID: 31480311.
7. Ma Y, Li W, Ao R, et al. Central nervous system aspergillosis in immunocompetent patients. *Medicine.* 2020; 99(44): e22911.
8. Mardari R, Puppa A Della, Rotilio A, et al. Pontocerebellar angle aspergillosis: Clinical and radiological findings. *Neurologist.* 2011; 17(2): 75–78. PMID: 21364357.
9. Adunsky A, Rubinstein E, Goldsmith A. Aspergillus flavus meningitis and pontine hemorrhage in an older patient. J Am Geriatr Soc. 1996; 44: 739-740/ PMID: 8642177
10. Kumar D, Nepal P, Singh S, et al. CNS aspergilloma mimicking tumors: Review of CNS aspergillus infection imaging characteristics in the immunocompetent population. *J Neuroradiol.* 2018; 45(3): 169–176. PMID: 29273531.
11. Carrazana EJ, Rossitch E, Morris J. Isolated central nervous system aspergillosis in the acquired immunodeficiency syndrome. *Clin Neurol Neurosurg.* 1991; 93(3): 227–230. PMID: 1660377.
12. Winterholler M, Coras R, Geißdörfer W, et al. Fatal mycotic aneurysm of the basilar artery caused by Aspergillus fumigatus in a patient with pituitary adenoma and meningitis. *Front Med (Lausanne.)* 2017; 4: 113. PMID: 28770205.
13. Bolla C, Lupia T, Schimmenti A, et al. A rare case of meningoencephalitis due to Aspergillus fumigatus. *Working Paper of Public Health.* 2023; 11(1).
14. Canning B, Senanayake V, Burns D, Moran E, Dedicoat M. Post-influenza aspergillus ventriculitis. *Clin Infect Pract*. 2020; 7–8.
15. Schwartz S, Thiel E. Cerebral aspergillosis: tissue penetration is the key. *Med Mycol*. 2009; 47 Suppl 1: S387–S393. PMID: 19255905.
16. Schwartz S, Thiel E. CNS-aspergillosis: are there new treatment options? *Mycoses.* 2003; 46 Suppl 2(2): 8–14. PMID: 15055138.
17. Gupta N, Kodan P, Mittal A, et al. Role of voriconazole in the management of invasive central nervous system aspergillosis: A case series from a tertiary care centre in India. *J Fungi (Basel).* 2020; 6(3): 139. PMID: 32824829.
18. Schwartz S, Ruhnke M, Ribaud P, et al. Improved outcome in central nervous system aspergillosis, using voriconazole treatment. *Blood.* 2005; 106(8): 2641–2645. PMID: 15998833.
19. Coleman JM, Hogg GG, Rosenfeld JV, Waters KD. Invasive central nervous system aspergillosis: cure with liposomal amphotericin B, itraconazole, and radical surgery--case report and review of the literature. *Neurosurgery.* 1995; 36(4): 858–863. PMID: 7596522.
20. Clemons K V., Espiritu M, Parmar R, Stevens DA. Comparative efficacies of conventional amphotericin b, liposomal amphotericin B (AmBisome), caspofungin, micafungin, and voriconazole alone and in combination against experimental murine central nervous system aspergillosis. *Antimicrob Agents Chemother*. 2005; 49(12): 4867–4875. PMID: 16304147.
21. Shariati A, Didehdar M, Rajaeih S, et al. Aspergillosis of central nervous system in patients with leukemia and stem cell transplantation: a systematic review of case reports. *Ann Clin Microbiol Antimicrob*. 2021; 20(1):44. PMID: 34130699.
22. Koehler P, Salmanton-García J, Gräfe SK, et al. Baseline predictors influencing the prognosis of invasive aspergillosis in adults. *Mycoses.* 2019; 62(8): 651–658. PMID: 31066092.

Journal of Radiology Case Reports

www.RadiologyCases.com

FIGURES

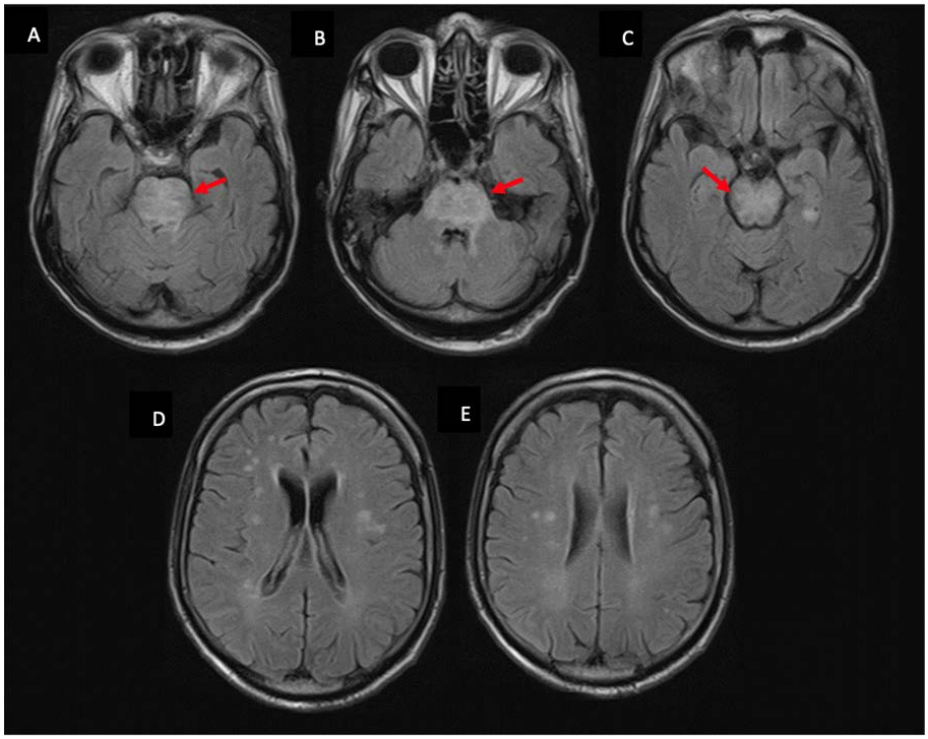

Figure 1: 61-year-old male with SNC aspergillosis.

FINDINGS: Brain magnetic resonance image (MRI) with confluent hyperintensities in pons (A, B and C) with symmetric involvement (red arrows). Cortico-subcortical hyperintensities (D and E).

TECHNIQUE: MRI Tesla 1.5 TOSHIBA Vantage Titan. Axial section of the brain acquired using T2-weighted fluid-attenuated inversion recovery (FLAIR) imaging.

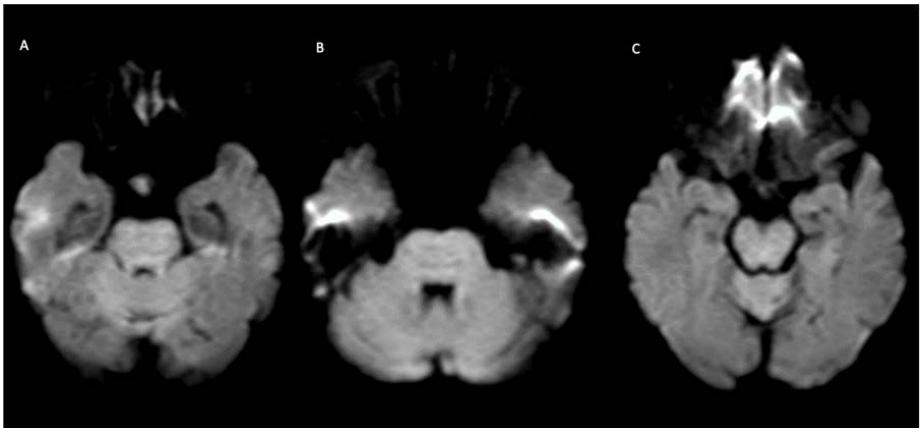

Figure 2: 61-year-old male with SNC aspergillosis.

FINDINGS: Brain magnetic resonance image (MRI) with no restriction in diffusion sequences or predominance by any structure (A, B and C).

TECHNIQUE: MRI Tesla 1.5 TOSHIBA Vantage Titan. Axial section of the brain acquired using diffusion-weighted (DWI) imaging.

Journal of Radiology Case Reports

www.RadiologyCases.com

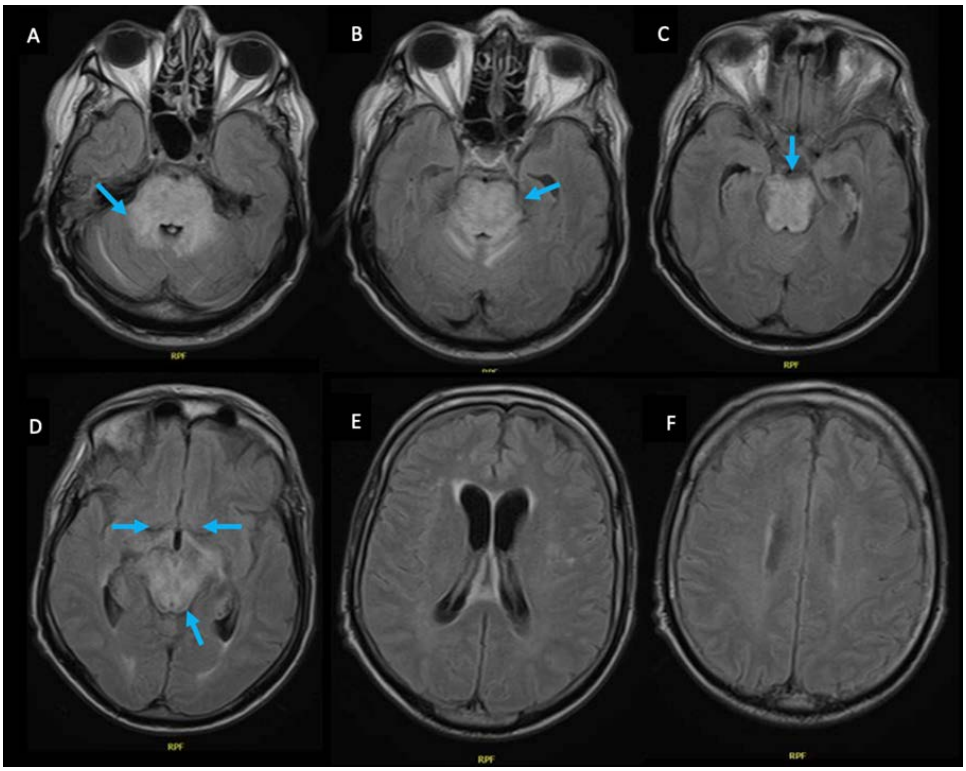

Figure 3: 61-year-old male with SNC aspergillosis.

FINDINGS: Follow-up brain magnetic resonance image (MRI) showing progression of pontine edema extending into the midbrain (A), diencephalic-mesencephalic junction (B), cerebellar peduncles, deep region of both hemispheres, bulb, and medullary cord (C and D) (Blue arrows). E and F showing images at the level of the lateral ventricles and upper with no alterations.

TECHNIQUE: MRI Tesla 1.5 TOSHIBA Vantage Titan. Axial section of the brain acquired using T2-weightened fluid-attenuated inversion recovery (FLAIR) imaging.

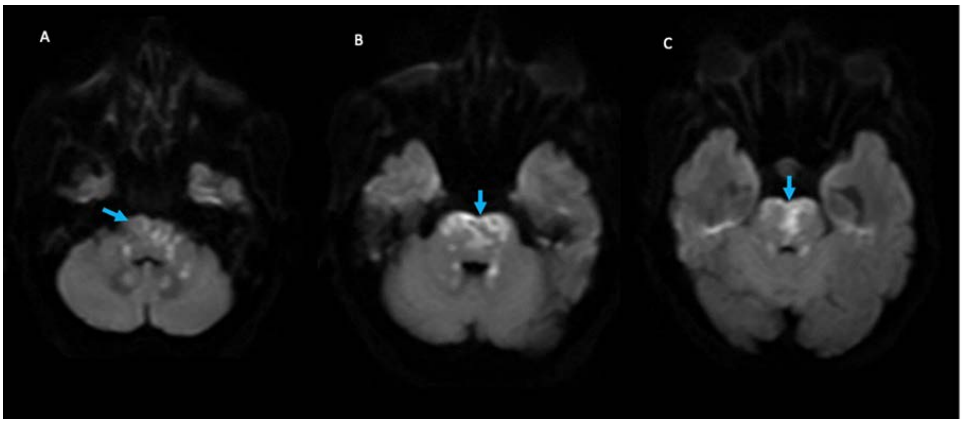

Figure 4: 61-year-old male with SNC aspergillosis.

FINDINGS: Follow-up brain magnetic resonance image (MRI) showing multiple foci of high signal intensity in cerebellum (A) and stem (B and C) (Blue arrows).

TECHNIQUE: MRI Tesla 1.5 TOSHIBA Vantage Titan. Axial section of the brain acquired using diffusion-weighted (DWI) imaging.

Journal of Radiology Case Reports

www.RadiologyCases.com

Table 1: Summary table

Category	Details
Etiology	Caused by *Aspergillus* species, predominantly *Aspergillus fumigatus*.
Incidence	Rare; occurs in 10-20% of invasive aspergillosis cases, with higher prevalence in immunocompromised patients.
Gender Ratio	Slight male predominance, likely due to higher exposure to environmental reservoirs.
Age Predilection	No specific age predilection; affects all age groups, with higher risk in older adults and severely immunocompromised individuals.
Risk Factors	Immunosuppression (e.g., hematologic malignancies, organ transplantation, neutropenia, corticosteroid use), chronic granulomatous disease.
Treatment	First-line: Voriconazole (IV/PO) ± surgical intervention. Alternatives: Amphotericin B and echinocandins in refractory cases.
Prognosis	Poor; mortality rates exceed 70% in untreated cases. Early diagnosis and intervention improve outcomes.
Findings on Imaging	Brain MRI: ring-enhancing lesions, infarcts, or abscesses. CT: hypodense lesions with surrounding edema. Contrast studies show irregular patterns.

Table 2: Differential diagnosis table for CNS Aspergillosis

Condition	Clinical Symptoms	Risk Factors	Lumbar Puncture (CSF)	Magnetic Resonance (MRI)
CNS Aspergillosis	Headaches, altered mental status, focal neurological signs (e.g., hemiparesis), seizures	Immunocompromised state (e.g., organ transplant, hematologic malignancy, corticoids)	*Hyperproteinorrachia*, pleocytosis, Galactomannan positive.	Multiple ring-enhancing lesions, abscesses, or areas of infarction.
CNS Tuberculosis	Chronic headaches, fever, weight loss, meningitis, focal deficits	Recent TB exposure, HIV/AIDS, immunocompromised state	*Hyperproteinorrachia*, Hypoglycorrhachia, lymphocytic pleocytosis	Meningeal enhancement, tuberculomas, hydrocephalus, basal cistern enhancement.
Cryptococcal Infection	Headaches, fever, altered mental status, nausea, blurred vision	HIV/AIDS, immunosuppression, exposure to pigeon droppings	Elevated opening pressure, positive India ink or Cryptococcal antigen	Gelatinous pseudocysts, dilated perivascular spaces, meningeal enhancement
Cerebral Infarction	Sudden onset of focal neurological deficits (e.g., hemiparesis, aphasia), headache	Atherosclerosis, cardiac disease, vasculitis, embolic sources	Typically normal, unless secondary infection	Restricted diffusion in affected area, may show infarcts related to vascular territories
Mycotic Aneurysms	Sudden severe headache, focal neurological deficits, signs of subarachnoid hemorrhage	Endocarditis, intravenous drug use, immunocompromised	May show *Hyperproteinorrachia*, red blood cells	Vascular irregularities, aneurysmal dilatations, signs of hemorrhage
Primary Brain Tumors	Progressive headaches, seizures, focal neurological deficits (depending on tumor location)	Family history, genetic syndromes	Typically normal, may show *Hyperproteinorrachia* in rare cases	Space-occupying lesion, possible surrounding edema, irregular contrast enhancement
Granulomatous Diseases	Headaches, neurological deficits, cranial neuropathies	Sarcoidosis, autoimmune disorders, chronic inflammatory conditions	*Hyperproteinorrachia*, lymphocytic pleocytosis, elevated ACE levels	Nodular or mass-like lesions, possible meningeal or periventricular enhancement
Multiple Sclerosis	Episodes of neurological deficits (e.g., visual disturbances, motor weakness), headache	Young adults, family history, autoimmune predisposition	Oligoclonal bands, elevated IgG index	Multiple white matter lesions, periventricular plaques, typically non-enhancing during remission
Neurosarcoidosis	Chronic headaches, cranial nerve palsies, seizures, behavioral changes	History of sarcoidosis, autoimmune conditions	*Hyperproteinorrachia*, lymphocytic pleocytosis, ACE may be elevated	Basal leptomeningeal enhancement, hypothalamic or pituitary involvement, possible mass effect

Journal of Radiology Case Reports

www.RadiologyCases.com

ABBREVIATIONS

CNS = Central Nervous System
CSF = Cerebrospinal Fluid
MRI = Magnetic Resonance Imaging
CT = Computed Tomography
PCR = Polymerase Chain Reaction

Online access
This publication is online available at:
www.radiologycases.com/index.php/radiologycases/article/view/5274

Peer discussion
Discuss this manuscript in our protected discussion forum at:
www.radiolopolis.com/forums/JRCR

Interactivity
This publication is available as an interactive article with scroll, window/level, magnify and more features.
Available online at www.RadiologyCases.com

Published by EduRad

www.EduRad.org

Journal of Radiology Case Reports

www.RadiologyCases.com