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Naeglaeria infection of the central nervous system, CT scan findings: a case series

Rohana Naqi,¹ Muhammad Azeemuddin²

Abstract

The imaging findings in four cases of a rare infection of the central nervous system caused by amoebae, *Naeglaeria fowleri* are presented. *Naeglaeria fowleri* are pathogenic free-living amoebae. They cause primary amoebic meningoencephalitis (PAM), a rapidly fatal disease of the central nervous system. The computed tomography brain findings in 3 (75%) of our cases of pan amoebic meningoencephalitis showed non-specific brain oedema; 2 (66%) of these cases also had moderate hydrocephalus and among that 1 (50%) case showed an old lacunar infarction in peri-ventricular region. In the remaining 1 (25%) case the scan was normal with no evidence of oedema or abnormal lesion. Out of three cases with diffuse brain oedema, post-contrast images showed abnormal meningeal enhancement throughout the brain parenchyma in 1 (33%) case. However, no definite focal enhancing lesion was noted. In the rest of the cases, no abnormal parenchymal or meningeal enhancement was seen on post-contrast images.

The study was conducted in the Radiology Department of Aga Khan University Hospital, Karachi from July to December 2010.

Keywords: Central Nervous System, *Naeglaeria fowleri*, Amoebic meningoencephalitis.

Introduction

The incidence of protozoal and helminthic infestations of the central nervous system (CNS) is less than 1%, but these infestations tend to follow a fatal course.¹ The free-living amoebae *Naeglaeria fowleri* (NF) cause extremely rare and sporadic CNS infections termed as primary amoebic meningoencephalitis (PAM).² It is an acute, fulminant, rapidly fatal disease that occurs generally in previously healthy children and young adults with a history of swimming and diving and other recreational activities in fresh water and contaminated swimming pools.³ The portal of entry is via the olfactory mucosa and neuroepithelium. Incubation period is 3-8 days with acute and rapidly fatal course. Death occurs within 7 to 10 days. It causes a fulminating haemorrhagic necrosis of the

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brain.⁴ About 300 cases of PAM have been reported internationally, mostly from the US, Australia and Europe. These infections were nearly uniformly fatal, with only 7 survivors of PAM reported in Western literature.⁵ There is little published literature available locally. Previous reports on NF cases from Karachi, Pakistan, focussed on changing climatic conditions and deteriorating water distribution system, and did not address much the issue of imaging findings. This series primarily focusses on the computed tomography (CT) features of NF causing PAM.

In 2011, a study reported 13 NF cases of PAM from Karachi. Magnetic Resonance Imaging (MRI) showed basal meningeal enhancement in one patient.⁶

Another study described the imaging features of amoebic meningoencephalitis. In the case of PAM, there was obliteration of cisterns with enhancing basilar exudate. Besides, there was infarction of right basal ganglia.⁷

In the case of PAM reported by another study, both CT and MR imaging revealed a pattern of brain oedema and hydrocephalus with rapid progression of the disease.⁸

Case Series

There were four cases in the series during July to October 2010.

Case-1

A young 18-year-old female patient was referred for CT scan of the brain after complaints of headache, seizures and altered consciousness for one day. On examination, she was drowsy and disoriented. Her CNS examination showed signs of meningeal irritation e.g. neck rigidity and positive Kerning sign. Her laboratory investigations showed white blood count (WBC) 24.5, with neutrophils 93.1. cerebrospinal fluid (CSF) examination showed glucose: 80; proteins: 371; total Leucocyte count (TLC): 150. CSF culture showed motile Amoebic Trophozoites, NF. Her CT scan brain was done which showed mild diffuse oedema in bilateral cerebral convexities with effacement of cortical sulci and partial effacement of ventricles (Figure-1). Post-contrast images showed no abnormal parenchymal and meningeal enhancement. She was intubated due to low Glasgow Coma Scale (GCS) rating and was shifted to the Intensive Care Unit (ICU). She was instantly started on high doses of Amphotericin-B. However, she did not make it, and was declared brain-dead.

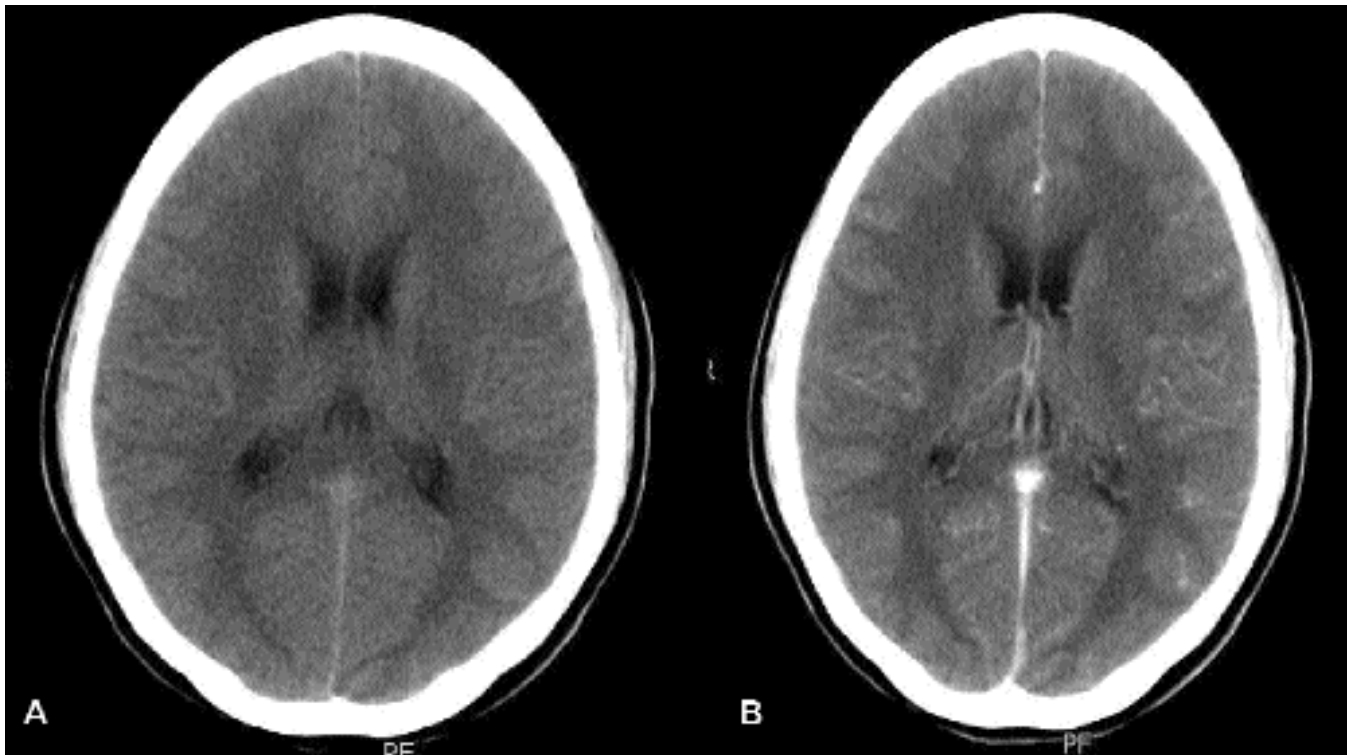


Figure-1: (A) Plain Computed Tomography (CT) Brain Axial Image: Oedema in bilateral cerebral convexities with effacement of cortical sulci and partial effacement of ventricles. (B) Post-Contrast Axial Image: No abnormal parenchymal or meningeal enhancement.

Case-2

The second case related to a 64-years-old male patient who presented with fever, nausea, vomiting and



Figure-2: Plain Computed Tomography (CT) Brain Axial Image: Normal ventricles and cortical sulci. No evidence of oedema or abnormal lesion.

drowsiness. On examination, he was comatose with fixed and dilated pupils, and absent reflexes. Laboratory investigations showed WBC 19.9 with neutrophils of 95.7. CSF examination showed glucose: <5; proteins: 1342; TLC: 5200. CSF culture showed motile Amoebic Trophozoites, NF. He underwent CT scan brain which was normal with no evidence of oedema or abnormal lesion (Figure-2). The patient was admitted in a critical state and intubated. He was instantly started on high doses of Amphotericin-B. However, he did not survive and was declared as brain death.

Case-3

In the third case, a 60-year-old male patient was referred for CT scan of the brain after complaints of fever, headache, seizures and drowsiness for two days. On examination, he was drowsy and disoriented. His CNS examination showed signs of meningeal irritation. The laboratory investigations showed WBC 25.9, with neutrophils 92.6. CSF examination showed glucose: 26; proteins: 504; TLC: 2500. CSF culture showed motile Amoebic Trophozoites, NF. CT scan brain was done which showed oedema in bilateral cerebral convexities and posterior fossa with effacement of basal cisterns and compression of the fourth ventricle. There was moderate hydrocephalus with old lacunar infarction in the right



Figure-3: (A) Plain Computed Tomography (CT) Brain Axial Image: Oedema in mid-brain and posterior fossa with effacement of basal cisterns and compression of the fourth ventricle. There is also moderate hydrocephalus with old lacunar infarction in the right peri-ventricular region. (B) Post-contrast Axial Image: No abnormal meningeal or parenchymal enhancement.

peri-ventricular region. Post contrast image showed no abnormal meningeal or parenchymal enhancement (Figure-3). He was intubated due to low GCS and was shifted to the ICU. He was instantly started on high doses of Amphotericin-B. The patient died after two days.

Case-4

The last case in the series was that of a 30-year-old male patient who was referred for CT scan of the brain after complaints of fever, headache, and drowsiness for two days. On examination, he was drowsy and disoriented. His CNS examination showed signs of meningeal irritation with fixed and dilated pupils. Laboratory investigations showed WBC 12.8, with neutrophils 89.3. CSF examination showed glucose: <5; proteins: 1147; TLC: 7500. CSF culture showed motile Amoebic Trophozoites, NF. CT scan brain was done which showed diffuse oedema in bilateral cerebral hemispheres with partial effacement of cortical sulci. There was also moderate hydrocephalus. Post-contrast images showed abnormal meningeal enhancement throughout brain parenchyma. There was no definite focal enhancing lesion (Figure-4). He was intubated in emergency due to low GCS and was shifted to the ICU. He was instantly started on high doses of Amphotericin-B. The patient died after 5 days.

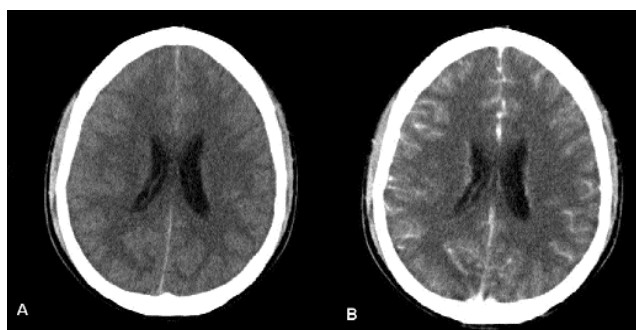


Figure-4: (A) Plain Computed Tomography (CT) Brain Axial Image: Diffuse oedema in bilateral cerebral hemispheres with partial effacement of cortical sulci. Moderate hydrocephalus is also seen. (B) Post-contrast Axial Image: Abnormal meningeal enhancement throughout brain parenchyma. There was no definite focal enhancing lesion.

Discussion

PAM is characterised by the sudden onset of severe frontal headache, fever, nausea, vomiting and rhinitis. These are followed by stiff neck, diplopia, loss of sense of smell, confusion and seizures, progressing rapidly to coma and death. An elevated white cell count is usual with a marked increase in neutrophils.⁴ Diagnostic test of choice involves CSF examination as a wet mount⁴ with direct visualisation of Naegleria under light microscope. These are actively

motile and can be stained with Heidenhain's iron Hematoxylin and Wheatley's stains. There is pleocytosis with neutrophilic dominance and a high protein with low sugar.

Pathologic changes in cases of PAM are extensive damage to brain parenchyma, ependyma and meninges. Congestion of meningeal vessels, oedematous cortex with herniation of uncus and cerebellum are other features.⁷

It has been more than a decade since the first report of the CT findings in panamoebic meningoencephalitis. The report described contrast enhancement and obliteration of the basilar cisterns was described as a non-specific, but central feature of the disease.⁹ Review of the literature suggests that the most common findings on CT (seen in 50%) are interpreted as cerebral oedema. Pan-amoebic meningoencephalitis primarily affects the base of brain and basilar subarachnoid cisterns, and because one of the hallmarks of diffuse cerebral oedema is effacement of the basilar cisterns.¹⁰

The imaging modalities in PAM, i.e. CT and MRI may show non-specific positive findings.¹⁰ CT scans may show obliteration of the cisterns surrounding the mid-brain and the subarachnoid space. MRI is usually suggestive of cerebral oedema with meningeal enhancement.⁸

In 1995, a study reviewed 10 CTs that had been reported in the literature. The CT was interpreted as normal in four cases. Cerebral oedema was the only finding in four cases. One case showed both cerebral oedema and contrast enhancement of the basilar cisterns and sulci. One case showed generalised meningeal enhancement in the basilar cisterns with mild hydrocephalus but no oedema.¹⁰

The drug of choice is Amphotericin-B (intrathecal and intravenous). Rifampicin, tetracycline may be added for better results.¹⁰

In our series, four cases were reviewed. All patients were residents of Karachi and came from different localities. Cerebral oedema was the only finding in our first case, with no abnormal parenchymal and meningeal enhancement. The CT was interpreted as normal with no

evidence of oedema or abnormal lesion in the second case while the third case showed oedema in bilateral cerebral convexities and posterior fossa with effacement of basal cisterns and compression of the fourth ventricle with moderate hydrocephalus and lacunar infarction in the right peri-ventricular region, and no abnormal meningeal or parenchymal enhancement. The fourth case showed cerebral oedema with moderate hydrocephalus and generalised meningeal enhancement.

Conclusion

CT findings in the case series were non-specific and similar to those reported in earlier studies despite tremendous improvement in CT resolution over time. The affected patients can show cerebral oedema, hydrocephalus or normal brain on CT. Therefore, PAM should be considered in any patient whenever these non-specific findings are accompanied by relevant clinical history of meningitis and exposure to fresh-water pools. CSF cytology of wet mount becomes mandatory in such cases as early treatment with Amphotericin may improve survival chances.

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