

**STUDY OF INTRACRANIAL ABSCESS IN GANDHI MEDICAL COLLEGE
BHOPAL FROM JUNE 2010 TO MAY 2014**Sudhir Singh¹, Archana Shukla², Sanjiv Kumar³**HOW TO CITE THIS ARTICLE:**

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ABSTRACT: A prospective and retrospective study was conducted in Hamidia hospital and Gandhi medical college Bhopal from year June 2010 to May 2014. Study included patients having intracranial abscesses. The objective was to know the incidence and prevalence, clinical features and management as well as outcome. Radiological and microbiological investigations were done. Procedures performed were lumbar puncture and intracranial abscess drainage by aspiration or craniotomy in few patients. There were total 33 patients in five years out of which 17 patients were operated and 16 were treated conservatively. Most common complication was residual abscess, meningitis and persistent hemiplegic.

KEYWORD: Intracranial abscess, Conservative antibiotic therapy, Surgical craniotomy.

INTRODUCTION: Intracranial abscesses are uncommon, serious, life threatening infections. Similar to the abscess in other sites, the pyogenic brain abscess is a collection of pus presenting as a mass like lesion. The pyogenic brain abscess can present with fatal illness.⁽¹⁾ Brain abscess is still related to high rates of neurological impairments and death. Intracranial abscesses include brain abscess, subdural empyema and intracranial epidural abscess and are classified according to the anatomical location or the etiological agent. The term brain abscess is used in this article to represent all types of intracranial abscesses. A brain abscess is a focal, intra cerebral infection that begins as a localized area of cerebritis and develops into a collection of pus surrounded by a well – vascularized capsule. Subdural empyema is focal collection of pus located between the,^(2,3,4) duramater and the arachnoid mater, while an intracranial epidural abscess is accumulation of pus between the skull and dura mater. Intracranial abscesses can originate from infection of contiguous structures (e.g., otitis media, dental infection, mastoiditis, sinusitis) secondary to hematogenous spread from a remote site (Especially in patients with cyanotic congenital heart disease), after skull trauma or surgery, and rarely, following meningitis. In at least 15 % of cases, no source can be identified.^(5,6)

Most of the reports from India are from latter half of the 20th century. These reports mainly deal with the bacteriology of brain abscesses. Aim of this Study is to analyze the current epidemiological, bacteriological, pathological and radiological picture of brain abscess. We shall also analyze various treatment options. The data will then be compared with the available literature. In recent years, the complex array of etiologic agents that cause brain abscess has become better understood.

AIMS AND OBJECTIVES:

1. To Study Incidence and Prevalence of Brain Abscess.
2. To Study Causes and Portal of Entry of Brain Abscess.
3. To Study Clinical Feature and Management Of Brain Abscess.
4. To Study Outcome of Brain abscess.

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PATHOPHYSIOLOGY: Animal and human modeling of brain abscess has demonstrated a 4-stage process of disease progression.^[5,6] The process begins with direct inoculation of microorganisms into the brain parenchyma, resulting in focal inflammation in the 1-3 days following, which is referred to as early cerebritis.^[7]

Polymorphic neutrophils are then recruited, leading to edema. Glial cells are activated, and the area of inflammation continues to grow as the central zone develops coagulation necrosis; this is a hallmark of the second stage, called late cerebritis, which occurs at approximately 3-6 days.^[8,9]

A well-vascularized, ring-enhancing capsule forms after approximately 2 weeks and may be seen on CT. As the host defenses mount, the capsule is walled off, thus completing the development of the abscess.

The fourth stage is often marked by considerable gliosis on the cortical surface of the abscess. Tissue destruction is likely dependent on the virulence of the organism and the exuberance of the host response.^[10]

The pathogenesis of an invading organism that has inoculated the brain parenchyma is variable and dependent on the initial site of infection, host factors, and geographic location. Infection can be due to bacteria, fungi, or protozoa. Brain abscess has traditionally been classified by the primary source of the pathogen,^[2,11] with the most common etiologies being direct extension, metastatic spread, and intracranial penetrating trauma.^(12,13)

ETIOLOGY: DIRECT EXTENSION: Brain abscess may be caused by the contiguous spread of pathogens from a primary focus of infection outside of the CNS that extends into the brain. Pathogens may originate from adjacent bone, teeth, sinus mucosa, internal auditory canal, or cochlear structures and travel into the intracranial vault via venous drainage or valve less emissary veins, thus inoculating the brain parenchyma.^[11] Abscess caused by direct extension usually leads to a solitary lesion.

Although less common, brain abscess has been described as a complication of frontal, ethmoidal, or sphenoidal sinusitis.^[12] Dental infections can lead to brain abscess via either contiguous or hematogenous routes. Meningitis rarely results in brain abscess by direct extension, particularly in adults, and therefore in most cases the finding of brain abscess should not prompt a search for meningeal infection via lumbar puncture.^[13]

In the past, chronic otitis media and mastoiditis were the most common underlying etiologies; however, complications of these infections have decreased in incidence with improvement in diagnostic modalities and antibiotic therapy.

Overall, abscess caused by direct extension now comprises 12-25% of all brain abscesses;^[2] however, where adequate healthcare infrastructure is lacking, direct extension continues to comprise approximately 50% of brain abscess collectively.^[14,15]

TRAUMA AND PROCEDURES: Penetrating intracranial trauma may be complicated by brain staphylococci, *Pseudomonas aeruginosa*, *Enterobacter*-species, *Enterobacteriaceae* species, and *Clostridium* species.

Though less common, neurosurgical procedures may be complicated by similar infections. In particular, methicillin-resistant *Saureus* (MRSA) infection should be considered in neurosurgical patients, and treatment with antimicrobials must take into account CNS penetration.^(7,8)

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Rare cases of brain abscess after endoscopic procedure, tongue piercing, dental braces, near drowning, and foreign body aspiration have been documented.^(9,11)

METASTATIC SPREAD: Hematogenous seeding of the brain from an extracranial source is the second most common etiology of brain abscess, accounting for approximately 25% of cases. While most bacteremias do not cause brain abscess, when they do, abscesses are frequently multiple and are often found in the distribution of the middle cerebral artery or watershed zones.^[11] When hematogenous spread is the underlying cause, there is often an additional predisposing factor, patients with comorbid conditions such as congenital heart disease with right-to-left shunt, pulmonary venous malformations, or hereditary hemorrhagic telangiectasia are at relatively high risk for brain abscess.

Among extracranial sources, chronic pulmonary infections such as lung abscess, bronchiectasis, and empyema have been frequently associated with hematogenous brain abscess. Bacteremias associated with endocardial, abdominal, pelvic, or skin infections can lead to brain abscess. Approximately 15% of cases have no identifiable source.^[1]

INTRACRANIAL TRAUMA: The formation of brain abscess after intracranial trauma or neurosurgical intervention is well described. In the case of penetrating trauma, brain abscess may form as an immediate or delayed complication; direct inoculation of pathogens can quickly lead to abscess formation, whereas a retained foreign body or focus of necrotic tissue can serve as a nidus of infection months or years after the initial insult.^[11,12] Compared with earlier series, there has been an increase in the proportion of brain.

IMMUNOCOMPROMISE: In immunocompromised hosts, causes of brain abscess may differ and could be the result of either opportunistic or bacterial infections. In addition to transplantation patients and those living with HIV/AIDS, individuals with alcoholism, diabetes, or long-term steroid use may be at increased risk for the development of brain abscess.^[9]

N gal infection from *Aspergillus*, *Candida*, *Cryptococcus*, *Enterobacteriaceae*, and *Mucorales* species. However, these patients may also have opportunistic infection with *Toxoplasma gondii* or *Nocardia* species.^(2,3)

HIV/AIDS patients with impaired cell-mediated immune response are at a particularly high risk of developing abscesses infected with *T gondii*, *Mycobacterium* species, *Cryptococcus neoformans*, or *Listeria monocytogenes* bacilli, as well as infection with *Aspergillus*, *Mucorales*, and *Candida* species. Long-term corticosteroid use predisposes to increased risk of bacterial abscess. The incidence of brain abscess colonized with *Listeria* is elevated in patients on long-term steroid use and carries an elevated risk of mortality compared with brain abscess colonized with other bacterial pathogens.^[11,12]

DIRECT EXTENSION: When the mode of acquisition is via direct extension, polymicrobial infections are common, occurring in approximately 20-25% of cases; these may involve aerobes and anaerobes, reflecting closely the flora of the contiguous site.

Aerobic and anaerobic streptococci are the most common isolates, found in approximately 70% of cases of bacterial brain abscess, and are frequently a component of mixed infections. *Streptococcus milleri* is a particularly common isolate because its proteolytic enzymes can effectively

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necrose tissue and lead to the formation of abscess. In addition to Streptococcus species, site-specific pathogens include the following;

- Otitis media and mastoiditis: Bacteroides, Enterobacteriaceae, Pseudomonas, Fusobacterium, Prevotella, Peptococcus, Pseudomonas, and Propionibacterium.
- Paranasal sinusitis: Bacteroides, Enterobacteriaceae, Staphylococcus aureus, Fusobacterium, and Haemophilus Odontogenic infection: Fusobacterium, Prevotella, Actinomyces, and Bacteroides.
- Brain abscesses that result from hematogenous spread are often multiple, poorly encapsulated, and found in the territory of the middle cerebral artery. These abscesses often involve bacteria from the source infection, as in the following:
 - In pulmonary infections, common organisms are Streptococcus species, Fusobacterium species, anaerobic gram-negative bacilli, and aerobic and anaerobic streptococci. Infections with Act and Nocardia species have been documented.
 - In bacterial endocarditis, the most common organisms are Streptococcus viridans, Streptococcus anginosus, and Streptococcus intermedius.
 - Congenital heart disease, as well as patent foramen ovale and other shunts, including intrapulmonary shunts, predispose to intracerebral spread of bacteremia. Typically, cultures grow Streptococcus species. Evidence suggests that the severity of the grade of right-to-left intrapulmonary shunts is strongly associated with the prevalence of cerebral complications, such as brain abscess.
- Intra-abdominal infections and genitourinary tract infections are another source of hematogenous spread of bacteria. These infections can be caused by aerobic genital urinary or anaerobic flora streptococci, Bacteroides, (Including Bacteroides fragilis), Escherichia coli, Proteus, Salmonella, Enterobacter, Prevotella, Propionibacterium, Eubacterium, Skin infections can also lead to bacteremia, which can cause brain abscess. In addition to Streptococcus species, Staphylococcus aureus is frequently implicated.

PROTOZOAL AND HELMINTHIC INFECTIONS: Several mycotic, protozoal, and helminthic infections may be endemic in certain regions. For example, Latin American populations have higher rates of neurocysticercosis from Taenia solium infection, while countries with endemic HIV/AIDS and tuberculosis experience higher rates of tubercular brain abscess. Parasites are the most frequent cause of abscess in individuals who have lived in or emigrated from low-resource settings. Note the following;

- Mycoses: Histoplasma capsulatum, Blastomyces dermatitidis, and Coccidioides immitis.
- Protozoal: Trypanosoma cruzi, Entamoeba histolytica, Schistosoma species, and Paragonimus species.
- Helminthic: Neurocysticercosis caused by the larval form of T solium.

Mortality/Morbidity: In the preantibiotic era, mortality from brain abscess was nearly 100%. Despite the introduction of antibiotics and improvements in neurosurgical drainage techniques, the mortality rate remained around 30-50% through the 1970s. The introduction of enhanced neuroimaging techniques, such as CT and MRI, allowed for rapid, accurate diagnosis and localization of brain abscess. In most modern series, the mortality rate is typically less than 15%.^[2] Rupture of a

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brain abscess infrequently occurs and is associated with a high mortality rate (up to 80%).^[2]

Significant morbidity, including seizures, persistent weakness, aphasia, or cognitive impairment, affects an estimated at 20-30% of survivors. In pediatric populations, outcomes have been shown to vary according to how rapidly antibiotics are initiated. Favorable outcomes have been associated with a number of factors, including initial Glasgow Coma Scale score of higher than 12, absence of underlying disease, or sepsis.

CLINICAL PRESENTATION:

RACE: No compelling evidence exists for racial differences in the incidence of brain abscess.

SEX: Although brain abscess can affect both sexes, in multiple series of both pediatric and adult patients, the male-to-female ratio of brain abscess has been demonstrated to range from 2: 1 to as great as 4: 1.^[15]

AGE: Throughout the first half of the 20th century, the age distribution of brain abscess was bimodal, with the highest rates being among children and adults older than 60 years. However, advancement in vaccination trends and antibiotic strategies, as well as a growing population of chronically immunosuppressed patients, has led to a shift in the demographics towards the middle decades.^[11,12] Overall, about 25% of cases of brain abscesses still occur in children, typically among those aged 4-7 years.^{7,8)} In pediatric series, congenital heart disease remains the most common predisposing factor.^(12,13)

HISTORY: The natural history of brain abscess ranges from indolent to fulminant. The clinical presentation is dependent on multiple factors, including the location, size, and age of the lesion(s). The microbiology of the infection, the host's immune status, and the mode of acquisition all contribute to the presentation.

The classic clinical triad of fever, headache, and focal neurologic deficit is present in less than 20% of cases.^[15] Typically, clinical manifestations of brain abscess are due to local effect or mass effect and often are not heralded by signs and symptoms of systemic infection.^[12] When brain abscess is caused by direct spread of infection, symptoms often localize in predictable patterns; for example, otogenic infections are associated with cerebellar and temporal abscess, while sinus infection may lead to abscess in the frontal lobes.^[1]

Headache is the most common presenting symptom and is observed in an average of 50-75% of patients.^[2] The headache is often nonspecific and may range from mild to severe and focal or generalized. Rupture of the abscess into the ventricular space may present as a sudden worsening of the headache and is often associated with high mortality.

Fever is present in less than 50% of cases and may be particularly unreliable in patients with HIV infection or compromised immunological function.

Focal neurological deficit such as hemiparesis or aphasia is present in 20-57% of patients and may correlate with the area of infection.^[12] Third or sixth cranial nerve palsies, anisocoria, or papilledema should all prompt concern for increased intracranial pressure and impending herniation.

Neck stiffness and meningeal signs have been reported in 25% of presentations; when present, especially if associated with sudden worsening of headache, these signs may herald abscess rupture and subsequent poor outcome.

Nausea and vomiting have been reported anywhere from 29-85% of cases, and the sign lacks sensitivity.^[8,10]

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Seizure has been reported from 7–25% of patients in case series.^[2,9]

Mental status changes are a common, but insensitive, marker. Glasgow Coma Scale score was less than 15 upon admission in 54% of patients in one large series.

PHYSICAL: Initial manifestations of brain abscess may be nonspecific, and, therefore, delay in diagnosis is common. Mean time from symptom onset to diagnosis is 2 weeks.] Symptoms often correspond to increased intracranial pressure or local disruption of brain parenchyma. A thorough physical examination must include inspection of the external and middle ear, as well as inspection of the mastoid bone, sinuses, and dentition.

Fever may be present in approximately half of all presentations and does not reliably aid diagnosis or exclusion.

Focal neurological deficits may be present in 40-60% of patients and may correlate with the area of infection.^[2] If the affected brain region(s) has redundant functions with an unaffected area, then focal deficits may more difficult to recognize.

Brainstem abscess may lead to facial weakness or hypothalamic dysfunction. Cerebellar abscess may cause nystagmus, ataxia, or dysmetria. Occipital abscess may cause neck rigidity. Temporal lobe abscess may be associated with headache, visual defects, or dysarthria. Frontal abscess may be associated with motor, sensory disorders, or headache. Lethargy may be a sign of global cerebral edema and should prompt concern for increased intracranial pressure and impending herniation. Neck stiffness and meningeal signs have been reported in 25% of presentations. For patients who display Kernig and Brudzinski signs, with concomitant neurological deficits, a careful evaluation should be conducted for brain abscess.^(8,9)

Seizures have been reported in 7-25% of patients and may be generalized or focal, but they are more likely generalized.^(2,12)

Mental status changes are a common but insensitive finding.^(7,9)

Third or sixth cranial nerve palsies, anisocoria, and papilledema may also be indications of increasing pressure and may indicate that the disease process has caused substantial edema either during the cerebritis stage or as a periaabscess inflammatory response; therefore, a cranial nerve and funduscopic examination should be performed. A bedside ocular ultrasound is a reasonable adjunct to funduscopic examination to assess for increased intracranial pressure.

Infants may present with irritability or seizure. Examination must evaluate for bulging fontanels and should include measurement of head circumference.⁽¹⁰⁾

LABORATORY STUDIES: Although nondiagnostic, patients with suspected brain abscess should have routine laboratory tests drawn to aid in narrowing the differential diagnosis. If surgical intervention appears likely, preoperative laboratory testing should be considered. CBC count should be obtained. Elevation of white blood cell count is present in 30-60% of patients.⁽¹¹⁾

Sedimentation rate and C-reactive protein may be obtained. Elevation of acute phase may be present, but results are nonsensitive, nonspecific findings. In one series, only 47% of patients had elevated erythrocyte sedimentation rate.

Two sets of blood cultures should be obtained, optimally prior to antibiotic administration; positive blood cultures are found in approximately 10% of cases. Brain abscess secondary to *L. monocytogenes* may be more likely to yield positive blood cultures.⁽⁸⁾

When hematogenous spread is suspected, consider obtaining cultures from the suspected primary infection.

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In immunocompromised patients, laboratory testing may include tuberculin skin testing and serotesting for toxoplasmal and anticysticercal antibodies in the cerebrospinal fluid (CSF). Background seroprevalence of *Toxoplasma* immunoglobulin G is high, and more than 97% of HIV/AIDS patients with toxoplasmosis brain abscesses have positive titers. Therefore, a negative test in an HIV/AIDS patient may be helpful in excluding toxoplasmosis from the differential.^(11,12)

IMAGING STUDIES: Advanced neuroimaging modalities, such as CT and MRI are credited with a substantial reduction in morbidity and mortality of brain abscess;⁽⁶⁾ this is due, in part, to the reduction of diagnostic delay. CT imaging is readily available in most emergency departments and may aid in rapid diagnosis of intracranial lesions; however, findings may vary with the stage of disease.

CT scans should be conducted with and without intravenous contrast. During the early stage of cerebritis, the lesion may appear irregular or hypodense and may be enhancing or non-enhancing with contrast. As the abscess begins to wall-off, CT imaging may reveal a classic ring-enhancing lesion. CT may be insufficient to detect infection during the cerebritis state, as well as for detecting posterior fossa lesions.^(7,8) CT is not 100% sensitive and may miss early lesions or those abscesses that fail to wall-off. Additionally, it cannot reliably distinguish between abscess and other causes of ring-enhancing lesions, such as tumor. Despite these challenges, CT imaging is generally sufficient to mandate admission and further inpatient workup.

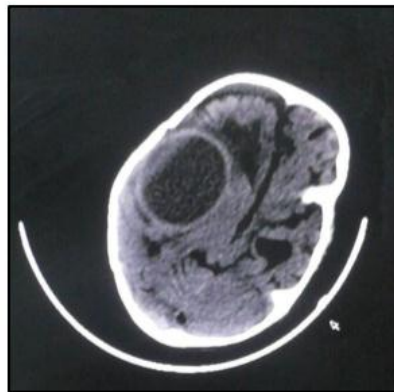


Figure showing brain abscess in right parieto-temporal region

MRI is more sensitive than CT imaging and may be used to aid in the diagnosis of brain abscess. MRI should be performed using gadolinium, which can increase the signal intensity of the lesion on T1.^(13,14)

MRI can be particularly useful during the early cerebritis stage or when there is suspicion of a posterior fossa lesion or satellite lesions.^[2] Moreover, unlike CT, MRI is capable of distinguishing between pyogenic and nonsuppurative lesions on diffusion-weighted imaging (DWI). On MRI, cerebritis appears as an area of low-signal intensity on T1-weighted imaging and has increased signal intensity on T2-weighted imaging.

DWI is used for differentiating ring-enhancing lesions that are neoplastic in origin, versus infectious causes. Suppurative abscess fluid restricts diffusion, leading to a hyperintense appearance of the lesion on DWI. In contrast, neoplastic lesions are typically hypointense or varied in intensity on DWI.

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Neuroimaging aids in the detection of abscess and it plays a critical role in microbiological sampling, disease management, and follow-up. The advent of stereotactic CT-guided aspiration of abscess fluid has allowed for greater yield of abscess fluid and can aid in directing antimicrobial therapy.

OTHER TESTS: As ultrasonography is becoming widely used in the emergency department, bedside ocular ultrasonography may be performed to assess for increased intracranial pressure.

Abscess aspiration and culture can be performed. Brain abscesses are aspirated by a neurosurgeon. Microbiological findings of the aspirate often help steer antimicrobial therapy. Therefore, patients with suspected brain abscess warrant urgent neurosurgical consultation. Neurosurgical intervention may include brain biopsy for culture and histopathology. The abscess may be accessed by stereotactic CT-guided aspiration, open evacuation of the abscess, or with ultrasound guidance. CT guidance is the most common modality. Abscess fluid is typically evaluated by Gram stain and culture, including aerobes, anaerobes, acid-fast staining for mycobacteria, and modified acid-fast staining for *Nocardia*. Specific fungal staining, such as methenamine silver or mucicarmine, can be performed on the aspirate. In the case of suspected neurocysticercosis, serology may be obtained for anticysticercal antibodies

PROCEDURES: Lumbar Puncture: In instances in which meningitis is suspected, a lumbar puncture (LP) may have initially been obtained; however, lumbar puncture without prior CT imaging is contraindicated in the setting of focal neurological deficit or papilledema. Furthermore, if neuroimaging reveals a mass lesion, LP is contraindicated. Occasionally, as is the case with early cerebritis, neuroimaging may not reveal a mass lesion. Although the risk of herniation may be low, CSF sampling to confirm diagnosis of brain abscess is rarely indicated and of very low yield.

CSF findings may include increased or normal cell count, increased polymorphic neutrophils, elevated protein, and normal glucose-findings that are neither sensitive nor specific for brain abscess. Gram stain and cultures of CSF are rarely helpful.^[2]

In the case of intraventricular rupture, CSF may show an abundance of red blood cells and leukocytosis. Elevated CSF lactic acid levels have been reported and may aid in diagnosis.

PREHOSPITAL CARE: Although brain abscess is rare, the emphasis of prehospital care should be on expedient transport of the patient to the hospital. If rupture is suspected, urgency in transport is even greater. Emergency Department Care: Initial management is a function of the severity of the patient's presentation.

Goals of initial therapy include stabilizing the patient and minimizing neurological impairment. Evaluation begins with the primary survey and confirmation of the patient's airway, breathing, and circulatory function, followed by rapid initial assessment of the patient's neurological function.

If the patient is unable to protect his or her airway, has unstable respiratory function, or is obtunded, emergent intubation is required, and, therefore, rapid-sequence intubation should use cerebroprotective medications.

After initial stabilization, patients in whom brain abscess is suspected should undergo neuroimaging. Contrast CT of the brain is of greater utility in this population than noncontrast scanning. Frequent neurological evaluation is recommended, particularly when intraventricular rupture is suspected.

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Antibiotics are the first-line treatment for brain abscess. High-dose, broad-spectrum, intravenous antibiotics should be administered as early as possible in the patient's course. Emergent consultation with neurosurgery is recommended; however, delay in consultation should not delay antibiotic administration. Attempt to obtain blood and other cultures prior to antibiotic administration; however, it is not advisable to delay administration.

Seizure prophylaxis with anticonvulsants is typically indicated in patients with suspected brain abscess, given the high risk of seizure, which often exacerbates intracranial pressure.^[14] Patients presenting with active seizure should be treated aggressively in order to avoid worsening intracranial pressure.

The use of glucocorticoids is controversial and they should not be routinely administered. Patients who are rapidly decompensating may warrant corticosteroid therapy in order to reduce the life-threatening effects of vasogenic edema, this approach is controversial given the risk that steroids can worsen inflammation, prevent abscess formation during cerebritis stage, exacerbate necrosis, and increase the risk of ventricular rupture.^[1] used, steroids are typically given for a short course.

CONSULTATIONS: Special patient populations include individuals with HIV/AIDS and those with immuno compromised status; these patients may require early consultation with infectious disease specialists.

Although the prevalence is decreasing, patients with suspected brain abscess due to maxillofacial, otogenic, or odontogenic infections require additional consultation with an oromaxillofacial surgeon or otolaryngologist, as appropriate.

MEDICATION SUMMARY: Medical therapy alone may be adequate in a select group of patients; however, for most, surgical aspiration and drainage is required for definitive treatment. Initial empiric parenteral antimicrobial selection must take into account host status, mode of transmission, and antimicrobial CNS penetration. Immunocompromised hosts may warrant empiric coverage for fungal and parasitic infections. In general, antimicrobial selection should attempt to maximize bactericidal activity and be tolerable for weeks to months of therapy.

When hematogenous spread is suspected, antibiotic selection should also take into account site penetration at the source of infection. Although empiric treatment with antibiotics for several days prior to biopsy may reduce the yield, the true effect is not known. Seizures may occur at the time of presentation or may complicate the patient's course during disease progression and resolution; therefore, prophylaxis with anticonvulsants is a mainstay of treatment. Anticonvulsant therapy may be initiated in the emergency department and may continue until complete disease resolution, or longer. First-line agents include phenytoin, carbamazepine, valproate, and levetiracetam. Glucocorticoids remain controversial, and many authors recommend they only be considered as a life-saving measure in an unstable patient for whom there is concern for imminent brain herniation.

ANTIBIOTICS: Class Summary: In an immunocompetent host with direct extension of infection from a contiguous site, infection is frequently polymicrobial and empiric therapy should be directed at covering anaerobes and aerobes, including Streptococcus species. In addition to Streptococcus, site-specific pathogens include the following;

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- Otitis media and mastoiditis: Bacteroides, Enterobacteriaceae, Pseudomonas, Fusobacterium, Prevotella, Peptococcus, and Propionibacterium.
- Sinusitis: Bacteroides, Enterobacteriaceae, S aureus, Haemophilus.
- Odontogenic infection: Fusobacterium, Prevotella, Actinomyces, and Bacteroides.
- Penetrating trauma or surgical: S aureus, Streptococcus, Enterobacteriaceae and Clostridium.

Treatment for these infections typically includes a third- or fourth-generation cephalosporin in combination with metronidazole. Penicillin G offers coverage for anaerobic and aerobic Streptococcus species and has excellent CNS penetration but must be used in combination with an agent that targets anaerobic gram-negative bacilli such as metronidazole. For penetrating head trauma or a postoperative patient, MRSA coverage is strongly advised.

When hematogenous infection is suspected, antimicrobial coverage must be directed towards site-specific pathogens, as follows;

- Endocarditis: S viridans, S aureus.
- Pulmonary infections: Streptococcus, Fusobacterium, Corynebacterium, Peptococcus, Fusobacterium, Actinomyces, Bacteroides, Prevotella, and Nocardia.
- Cardiac defects: Streptococcus and Haemophilus 21.
- Intra-abdominal infections: Klebsiella, E coli, Enterobacteriaceae, and streptococcus.
- Urinary tract infections: Enterobacteriaceae and Pseudomonas.
- Wound infection: S aureus.

In many instances, antibiotics with beta-lactam-beta/lactamase inhibitor (eg, ampicillin-sulbactam, ticarcillin-clavulanate, or piperacillin-tazobactam) cross the blood-brain barrier and can be used effectively. However, overall evidence for appropriate therapy is lacking. A recent Cochrane review found that there are no randomized controlled trials addressing the effectiveness of antibiotic regimens for brain abscess in the setting of cyanotic congenital heart disease.

Where MRSA is suspected, vancomycin is a frequent choice, however, some authors have argued that it has poor CNS penetration and favor linezolid.] MRSA infection should be suspected if the patient is postoperative following a neurosurgical procedure, in the case of penetrating head wounds, or from hematogenous spread of a known MRSA infection. MRSA infection can also be community acquired.

For immuno compromised hosts, pathogens and treatment vary with host factors, as follows:

- HIV/AIDS infection: T gondii, Nocardia, Mycobacterium, L monocytogenes, or C neoformans
- Transplantation: Aspergillus, Candida, Mucorales, Enterobacteriaceae, Nocardia, or T gondii
- Neutropenia: Aerobic gram-negative bacilli, Aspergillus, Mucorales, or Candida Aspergillus infection has been shown to respond to voriconazole, while amphotericin B remains a mainstay of treatment for most other fungal infections.[First-line therapy for the protozoal infection T gondii is with pyrimethamine plus sulfadiazine. Effective first-line agents for Nocardia include sulfadiazine or trimethoprim-sulfamethoxazole. Finally, several mycotic, protozoal, and helminthic infections should be considered, in certain cases, where they are endemic to the region, as follows:
- Mycoses: H capsulatum, B dermatitidis, and C immitis
- Protozoal: T cruzi, E histolytica, Schistosoma, and Paragonimus
- Helminthic: Neurocysticercosis, caused by the larval form of T solium

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In these instances, targeted therapy is required, and selection depends on the suspected organism.

When initial investigation does not reveal a likely cause of brain abscess, the recommended empiric coverage is a third- or fourth-generation cephalosporin, plus vancomycin, and metronidazole.

Corticosteroids: Class Summary: The use of corticosteroids in brain abscess is controversial. Intravenous steroids are usually reserved for patients who have severely increased intracranial pressure, either from mass effect or substantial edema around the periphery of the abscess. Steroids are thought to reduce intracranial pressure by decreasing edema via their anti-inflammatory properties. However, steroid use may also decrease antibiotic penetration, as well as slow the encapsulation of the suppurative fluid, thus potentially offsetting the beneficial effects. Significant and potentially serious metabolic adverse effects can occur with high dosages.

DEXAMETHASONE (DECADRON, DEXASONE): Dexamethasone is the corticosteroid of choice for reducing intracranial pressure. It is used in the treatment of inflammatory diseases. It may decrease inflammation by suppressing migration of polymorphonuclear leukocytes and reversing increased capillary permeability.

FURTHER INPATIENT CARE: Despite considerable reductions in serious infection with potentially medical care of these patients approach.

Morbidity and mortality, brain abscess is a profound prognostic implications. Modern often requires a swift, multidisciplinary Initial management steps include confirmation of the size, location, and number of intracranial abscesses using CT with intravenous contrast, MRI, or both.

The decision to surgically manage the disease may vary with the characteristics of the abscess, and numerous practices are used. Typically, abscesses larger than 2.5cm are excised or aspirated, often during emergent surgery. Patients in the early cerebritis stage or those with abscesses that are smaller than 2.5cm may undergo aspiration for diagnosis only. Some neurosurgeons may prefer complete evacuation of the abscess capsule, while others may plan to reaspirate the javascript: show ref content ('Reference layer'); lesions.^[2]

Stereotactic CT or MRI-guided needle aspiration is a key procedure in facilitating expedient identification of the pathogen and may be a preferable approach compared with open craniotomy, with benefits of reduced morbidity and mortality. Culture data collected during aspiration are beneficial for targeting and narrowing antimicrobial regimens.

Empiric intravenous antibiotics given for several days have an unclear effect on the success of brain abscess aspiration culture data. A major trial looking at the use of antibiotic therapy for up to 10 days prior to aspiration showed that this practice does not alter culture growth. However, a second large study showed that approximately 40% of abscess cultures had no growth after empiric antibiotic therapy.^[15]

The duration of intravenous antibiotic treatment is frequently 4-8 weeks or longer, with subsequent transition to oral antibiotics for another 4-8 weeks in order to ensure complete resolution and prevent relapse. A small body of literature on the effects of hyperbaric oxygen therapy as adjunctive therapy for brain abscess exists. Primary outcomes include reduction in length of stay and decreased duration of antibiotics therapy; however, at this time, evidence is insufficient.

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SURGERY: In our experience, pyogenic abscess required surgical intervention while most of the tuberculous abscesses were managed conservatively. The initial approach is to drain the abscess through a twist drill craniotomy. If the pus is thick or there is inadequate drainage of abscess suspected, the next procedure would be therapeutic burr-hole drainage. Deep seated abscess like a thalamic abscess should be drained by a CT guided stereotactic procedure.

Adequate drainage of the pus produces an immediate clinical improvement and helps the patient to stabilize hemodynamically. It is our aim to drain the entire pus with a single attempt but to a large extent, the burrhole drainage is seldom complete. This could be achieved with intraoperative radiography or using neuronavigation. However, the patient is kept under close neurological and radiological monitoring. The residual pus can be evacuated if the patient does not exhibit significant improvement or serial radiography or CT imaging reveals moderate to large residue. About 90% of the supratentorial hemispheric abscesses resolve with burrhole drainage. We have seldom felt the need to perform a craniotomy. The indications for craniotomy are multiloculated abscess and thick pus. In case of otogenic abscesses, urgent otolaryngological consultation is mandatory and mastoidectomy should be performed at the earliest. The treatment of brain abscess has been a challenge. Small brain abscesses have been treated empirically with antibiotics. Patients presenting with rapidly progressive neurological deficits due to the mass effect of the neuroradiologically verified brain abscess are strict candidates for urgent decompression both for the neurosurgeons and internists. The choice of procedure is a matter of debate. Craniotomy was advocated in the pre CT era but is now rarely practiced as the first line of treatment. Aspiration repeated as necessary or with drainage, has widely replaced attempts at complete excision.

However, open surgical procedure is still preferred by the treatment of the brain abscess with the combination of medical treatment, if there is an evidence of increased intra-cranial pressure due to significantly mass effect of the brain abscess, if there are difficulties in diagnosis, if the abscess is traumatic and, if the lesion is located in the posterior fossa and if there is any presumption of fungal infection. Excision is recommended for the multiloculated abscesses, posttraumatic abscesses containing foreign bodies or contaminated retained bone fragments, and abscesses due to fistulous communication. Several reports have advocated excision as the procedure of choice because it is often followed by a lower incidence of recurrence and shorter hospitalization.

Even the decompression with the craniotomy or craniectomy will be helpful for the patients with poor neurological condition. As diagnosis based only on clinical and neuroradiological findings can be erroneous, nonsurgical therapeutic decisions should not be taken without positive pathological diagnosis. Favorable outcome was not significantly different between the patients treated by excision or aspiration however, the mortality rate was significantly lower in the patients treated with excision than the patients treated with aspiration. This is probably due to the better general condition and/or more favorable location of abscess that could be excised surgically in such patients.

Stereotactic management of brain abscess, allowing both confirmation of the diagnosis and institution of therapy by aspiration of its contents and identification of the offending organism, has become widespread with the introduction of CT-guided stereotaxy. A review of the recent literature shows several series of brain abscesses primarily treated with stereotactic techniques. Stereotactic aspiration should be considered the treatment of choice in all but the most superficial and the largest cerebral abscesses. Kondziolka et al. related the failure of stereotactic treatment of brain abscesses in a series of 29 cases, because of either inadequate aspiration, lack of catheter drainage, chronic

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immunosuppression, or insufficient antibiotic therapy. Neuroendoscopic technique with free hand stereotaxy has also been practiced. Both Hellwig and Kamikawa reported their experiences with a flexible scope (free-hand or stereotactic-guided). Longatti et al. reported the usefulness of flexible endoscope in certain crucial surgical actions, such as aspirating and inspecting the abscess in all space directions or in firm and elastic membrane requires scissors or other instruments for its perforation. The use of drainage catheters inside the abscess cavities is controversial. Longatti et al. reported that no significant difference could be obtained in the length of hospital stay, number of postoperative CT scans, and duration of the antibiotic therapy between traditional and endoscopic stereotactic aspiration. Intraoperative imprint-smear diagnosis of brain abscess is fraught with pitfalls viz. abscess related necrosis must be differentiated from tumor necrosis. In deep seated, multiloculated and periventricular abscesses, a reduction of 1 mm in the distance between the ventricle and brain abscesses will increase the rupture rate by 10%. Surgical therapy can be preferred for the patients with neurological deterioration and/or radiological unresolved lesions. The surgical technique of choice for intra-cranial abscess should be specific to each patient. A combination of the surgical aspiration or removal of all abscesses larger than 2.5cm in diameter, a six weeks or longer course of intravenous antibiotics, and weekly CT or MRI imaging should result in a cure rate of more than 90%. It is important to follow the patient carefully by CT or MR imaging until the abscess has completely resolved. If any abscess enlarges after two weeks of antibiotics or fails to resolve after three to four weeks of antibiotics, further surgical aspiration or excision should be performed.

FURTHER OUTPATIENT CARE: Serial neuroimaging, either with CT or MRI, is typically recommended in order to follow treatment successfully to resolution of the abscess. Management may vary from weekly to monthly reimaging, depending on local practice patterns, as well as the patient's course and symptomatic resolution. Enhancement of the lesion on neuroimaging may persist for months. Data on the precise interval for when to obtain reimaging studies are currently insufficient. In some patients, brain abscess fails to respond to antimicrobial therapy, while in others the response is not durable and the abscess reaccumulates; in these instances, reaspiration may be necessary.^(11,12)

TRANSFER: These patients frequently require neurosurgical management; thus, prompt transfer to a neurosurgical-capable hospital is appropriate if unavailable at the initial treating facility. Patients may also require ICU level of care, which is another indication for transfer.

COMPLICATIONS: Complications of brain abscess may be localized or global and include persistent weakness, aphasia, or cognitive impairment, as well as life-threatening complications such as herniation or intraventricular abscess rupture. Brain herniation is frequently secondary to increased intracranial pressure from profound periabscess edema. Rupture of abscess into ventricles or subarachnoid space is a complication that is often lethal. High-risk features for this complication include an abscess that is deep seated, multiloculated, and/or close to the ventricular wall.

Morbidity, including persistent neurologic sequelae such as focal deficits, seizures, and headache, occurs in approximately 50% of patients.^[11]

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PROGNOSIS: The mortality rate of brain abscess has decreased to 15% in most developed countries, while mortality in resource-poor settings remains higher. One of the most important factors in prognosis is the availability of healthcare resources. Mortality rates among immuno compromised patients are higher, despite appropriate surgical and medical therapy. If immunosuppressive agents can be reduced, the chance of a positive outcome is improved.^[1]

Key prognostic factors include associated with improved prognosis include the following;

- Young age.
- Absence of severe neurologic defect on initial presentation.^[12,]
- Absence of neurologic deterioration during initial presentation.^[4]
- Absence of comorbid disease.^[2,12]

Worse prognosis of brain abscess is associated with the following:

- Advanced age
- Hematogenous spread.⁽²⁾
- Immunosuppression.^(3,4)
- Evidence of intraventricular rupture.^(8,9)
- Evidence of herniation on initial presentation.^(9,11)
- Evidence of altered sensorium on initial presentation.^[15]
- Severity of abscess and abscess location on initial neuroimaging.^(4,8)
- Delay in diagnosis or definitive surgical intervention.^(5,8)
- Certain pathogens, including gram-negative bacteria, [nocardial abscess,^(8,9) or aspergillosis] portend a worse prognosis and higher mortality rates, particularly in immune-compromised patients.^(8,10)

METHODS AND MATERIAL: This study comprised all patients of intracranial abscess admitted in various departments of Gandhi Medical College, Bhopal during June 2010 to May 2012 (Retrospectively) and June 2012 to 2014 (Prospectively). This study had been carried out after obtaining necessary ethical clearance from the institutional ethical committee. A complete neurological examination had been conducted on admission and regularly followed.

Relevant investigation had been conducted as per unit protocols. At the time of diagnosis, initial intravenous broad spectrum antibiotics had been given, usually for 2 weeks or more, to cover aerobic (gram positive and gram negative) and anaerobic organisms. Therapy had been was then tailored according to the sensitivities of organisms isolated from pus specimen.

Intravenous therapy had been was continued for a minimum of 2 weeks after surgical drainage, usually followed by a minimum of 4 weeks of oral therapy.

Pus from brain abscesses was collected during neurosurgery either by burr hole or by craniotomy. The pus had been inoculated on to suitable solid media for aerobic and anaerobes. A direct smear had been made, fixed in methanol for 30 s and stained by gram stain for studying the morphology of infective agents. Ziehl – neelsen (ZN) staining had been as also performed on all the pus specimens. A KOH (10%) preparation of pus specimens had been made when fungal filaments are suspected on a Gram stain.

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OBSERVATION AND RESULTS: In our study, "Study of Intracranial Abscess Patients, Admitted in GMC Bhopal from June 2010 To May 2014" the following observations were recorded: -TABLE 1

AGE	Total no. Patient	Male	Female
0-10yr	9(27.27 %)	5(55.55%)	4(37.5%)
11-20yr	8(24.24%)	5(62.50%)	3(42.36%)
21-30yr	7(21.21%)	4(57.14%)	3(33.33%)
31-40yr	3(9.09%)	2(66.66%)	1(33.33%)
41-50yr	3(9.09%)	1(33.33%)	2(66.67%)
51-60yr	1(3.03%)	0(0 %)	1(100.00%)
61yr-more	2(6.06%)	1(50.00%)	1(50.00%)

Age & Sex Distribution of Cases

Maximum no of patients 9 (27.27%) is in 0-10yrs age group followed by 8 (24.24%) is in 11-20yrs ages group. out of total 33 patients 18(54.54%) are male and 15(45.45%) are female.

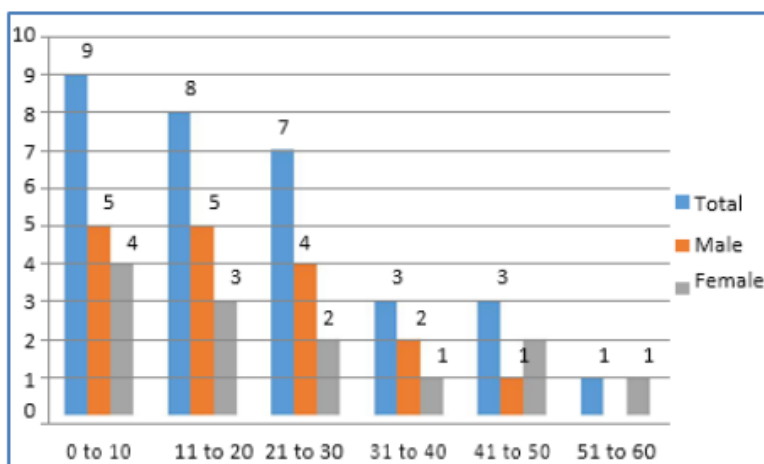


Fig. 1: age & sex wise distribution of patients

DISCUSSION: The current study highlights that brain abscess continues to pose a significant problem as we dealt with 33 cases in the 5 year study period, giving a mean of 07 cases per year. From this study we noticed that brain abscess could occur at any age. The most affected age group was 0-10 years. However, the majority (50%) of cases occurred below the second decades of life. found that this disease occurs most often in the middle decades of life reported 74.89% of their patients were below 20 years of age. A brain abscess in neonates and infants is a rare condition, but there are occasional reports of brain abscess in,^(10,11) infants documented in the literature. Males were found to be more affected than females in the present study, irrespective of the age group. Similar observations have been reported from different parts of world. It is clear from all these studies that males are more vulnerable to brain abscess irrespective of the geographical region. The reasons for such a distribution are not very clear.^(5,6,7)

Brain abscesses are usually solitary, although multiple abscesses occurring simultaneously in

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the brain have been reported. Multiple brain abscesses are usually very rare. Although encountered 4 such cases. Multiple brain abscesses is one of the major features of metastatic abscesses, which occur through haematogenous dissemination from a remote site of infection. In the present study, all the patients with multiple brain abscess had cyanotic congenital heart disease. Headache, fever, and vomiting made an important triad of symptoms in the present study, which were present in 86.86 % of patients. Fever has been reported as the important presenting symptom in brain abscess patients by many workers; however, the absence of fever does not exclude the diagnosis.^(4,5,6)

Vomiting is another common symptom suggestive of raised intracranial pressure. An alteration in the level of consciousness is usually present in up to two-thirds of the patients. In the present study 42.42% of the patients were brought to the hospital in unconscious state. The state of consciousness of the patient at the time of admission is usually taken as a reliable prognostic marker. Out of the 33 patients, 11(33.33%) had a demonstrable infectious source leading to the intracranial suppuration. CSOM was found to be the major source of infection. In most large series of brain abscesses from developing countries, middle ear infection has been reported to be the commonest source of intracranial suppuration. This has been reflected in various studies from India. This clearly indicates that an ear infection is often neglected and not treated aggressively in developing countries, while in developed countries the incidence of complication of CSOM has come down to 0.04 % of all cases of suppurative ear disease. Our study we also noticed that otogenic abscesses are usually solitary and located in either the temporal or the cerebellar lobe. Similar reports have appeared in the literature. Intracranial lesions frequently occur in association with congenital malformation of the heart. Intracranial abscess and cerebral thrombosis are the two most serious complications of the brain due to congenital heart disease. When such a CHD remains uncorrected, it becomes a nidus for bacteria to settle and become a source of bacteraemia. Tetralogy of Fallot and transposition of the great vessels are the most commonly cited predisposing factors. We encountered six (18%) brain abscess cases with associated congenital heart disease. The brain lobe affected usually depends on the predisposing factor that led to the development of the brain abscess. In our studies, the most common lobe involved was the posterior fossa followed by parietal lobe because CSOM was found to be the major predisposing factor.^(8,9)

Cultures were positive in 11(64.7%) cases. A culture positivity for brain abscess pus specimens of 44–100% has been reported in the literature. Monomicrobial aetiology was also found to be a feature of brain abscesses. Similar findings have been reported in the literature, but there are also reports of anaerobes out numbering For 6(35.29%) specimens in this study no organisms were isolated upon culture. The high incidence of such cultures may be because the samples were collected from a tertiary care centre and patients had undergone a treatment of long duration with broad spectrum of antibiotics before they reported to the hospital. It has been reported that for 9–63% of primary pus specimens from abscesses of the central nervous system no organisms were isolated upon culture. These cases also may have had a non-bacterial etiology, which was not specifically looked for in the present study.^(4,7,8)

The empirical antimicrobial regimens commonly recommended for therapy of brain abscess vary from unit to unit. The basis for selecting the antibiotics is usually the site of the lesion and the suspected causative organism had been presumed based on previous scientific data. In vitro antibiotic sensitivity results also showed that a cefotaxime and metronidazole regime, which is usually used in most of the neurosurgery units, is a satisfactory presumptive choice of therapy. Chloramphenicol was found to be a potentially useful alternative. Mortality ranging from 8 to 53%

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has been reported in other studies. Various factors contribute to mortality, the important factors being the age of the patient, the level of consciousness at the time of admission, multiplicity of abscesses and the nature of the infection. In the present study 24 % mortality was observed, and a statistically significant association was shown between the age of the patient and mortality.^(1,2,8)

The most successful predictor of clinical outcome for patients with brain abscess was the extent of neurological compromise at the time of presentation and diagnosis. All the patients that died in this study had an altered sensorium at the time of admission. The level of consciousness at presentation has been shown by other authors to be of great prognostic value. We could not find any significant association. According to, staphylococci cause the largest number of deaths, whilst there seemed to be a low death rate for patients with abscesses produced by streptococci in monobacterial growth. Few reported 40 % mortality with Gram-positive cocci and 50% mortality with Gram-negative bacilli. They also noted that *Proteus* infections were associated with a high mortality. This may have been due, in some part, to the pathogenicity of the organism. The virulence of these organisms is difficult to assess due to the small number of such cases.^(11,12)

The question as to whether polybacterial growth carries a higher mortality is difficult to answer. It seems that the prognosis in such abscesses depends on the most virulent. According to few, staphylococci causes the largest number of deaths, whilst there seemed to be a low death rate for,⁽¹⁵⁾ patients with abscesses produced by streptococci in monobacterial growth. Few reported 40 % mortality with Gram-positive cocci and 50% mortality with Gram-negative bacilli. They also noted that *Proteus* infections were associated with a high mortality. This may have been due, in some part, to the pathogenicity of the organism. The virulence of these organisms is difficult to assess due to the small number of such cases. The question as to whether polybacterial growth carries a higher mortality is difficult to answer. It seems that the prognosis in such abscesses depends on the most virulent of the various organisms.^(11,12)

CONCLUSION: The following were conclusions in our study, "Study of Intracranial Abscess Patients, Admitted in Gmc Bhopal from June 2010 To May 2014" conducted on 33 cases (Both retrospective and prospective);

- In the present study, most of the patients were in the age group of 0-10 years (27.27 %) followed by age group 11-20 years (24.24 %).
- In the present study, male were 54.54% while 45.45% were females.
- Headache is present in 31 (93.93%) patients followed by fever in 29(87.88%) patients than vomiting in 26 (78.78%) patient.
- In the present study, CSOM is present in 22 (66.67%) patients, cyanotic heart disease in 06 (18.18%) patients and diabetes mellitus in only 04 (12.12%) patients.
- In the present study, 29 (87.88%) patients have GCS score 12-15, And 02 (6.06%) patients have GCS 8-11, and 02 (6.06%) patients have GCS score <8.
- Out of total 33 patients, posterior fossa abscess is presents in 12 (36.36 %) patients and supra tentorial abscess in 21 (63.63%).out of supra tentorial abscess, parietal abscess in 8 (24.24%) patients is most common.
- In the present study, 18 (54.54%) patients have abscess of size 4 cm or more in size.
- Out of total 33 patients, 19 (57.58%) patients is associated with hydrocephalous
- Out of total 33 patients, 27 patient(81.82%) have midline shift,

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- In the present study, 16(48.48%) patients are managed conservatively and 17 patients are managed by surgery, out of which 10(30.30%) patients undergo aspiration by burr hole and 7(21.21%) patients by aspiration by craniotomy. Redo-surgery required in 2(6.67%) patients.

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AUTHORS:

1. Sudhir Singh
2. Archana Shukla
3. Sanjiv Kumar

PARTICULARS OF CONTRIBUTORS:

1. Associate professor Department of Surgery, GMC Bhopal.
2. Assistant Professor, Department of Surgery, GMC Bhopal.
3. Resident, Department of General Surgery, GMC Bhopal.

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NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Archana Shukla,
148/2, Doctors Quarters,
Doctors Campus,
Hamidia Hospital,
Bhopal, Madhya Pradesh, India.
E-mail: archie2210@gmail.com

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