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Original Article

Community-acquired intracranial suppurative infections: A 15-year report

Taner Yıldırmak, Habip Gedik, Funda Şimşek, Arzu Kantürk

Department of Infectious Diseases and Clinical Microbiology, MoH Okmeydanı Training and Research Hospital, İstanbul, Turkey

E-mail: Taner Yıldırmak - mtanery@gmail.com; *Habip Gedik - habipgedik@yahoo.com; Funda ŞimŞek - fundasimsek67@gmail.com; Arzu Kantürk - drakanturk@gmail.com
*Corresponding author:

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Abstract

Background: The aim of this study was to retrospectively evaluate the characteristics, treatment, and prognosis of patients with intracranial suppurative infection (ISI) by review of clinical, radiological, and laboratory findings.

Methods: The data collected from all patients who had been diagnosed with ISI and followed up at the Infectious Diseases and Clinical Microbiology Department of the study site between 1998 and 2013 were reviewed.

Results: Of the 23 ISI patients identified, the mean age was 38.21 ± 12.61 years (range: 19–67 years, median: 34) and mean symptom duration was 22.25 ± 20.22 days. Headache was the most common symptom, the frontal lobe the most common localization of ISI, and mastoiditis due to chronic suppurative otitis media the most common source of infection causing ISI. *Proteus mirabilis*, *Pseudomonas* spp., *Peptostreptococcus* spp., *Enterococcus avium*, *Mycobacterium tuberculosis* complex, and *Toxoplasma gondii* were isolated from the specimens collected from 6 (37.5%) of the 16 patients who underwent invasive procedures. Of these 16 patients, 2 underwent craniotomy, 12 burr hole aspiration, and 2 stereotactic biopsy. The rate of recurrence was 0% and the rates of sequelae and fatality were both 8%.

Conclusions: ISI should be considered in male patients presenting with headache and neurological signs and symptoms, whether with or without fever, on admission for early diagnosis and provision of timely, adequate therapy and, if required, surgical intervention to reduce mortality and sequelae rates.

Key Words: Epidural abscess, HIV, intracranial abscess, intracranial suppurative infection, spinal cord abscess, subdural empyema, toxoplasmosis, tuberculosis

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INTRODUCTION

Intracranial suppurative infection (ISI) is an infection of the central nervous system (CNS) that includes intracerebral abscess, spinal cord abscess, subdural empyema, epidural abscess, and suppurative intracranial phlebitis. ISI is reported in 1 case per 1000 patients admitted to the hospital and is primarily diagnosed in male patients.[22] Among the types of ISI, intracerebral abscesses are suppurative focuses that develop from local

cerebritis followed by capsule formation within 2 weeks of cerebritis onset.[3]In ISI patients, the infectious process attacks the CNS, which is vulnerable and incapable of exerting a significant immune defense by itself,[15] generally leading to subdural empyema after sinusitis due to breach of the dura mater.[3]

Adjacent focus and hematogenous spreading have been found to lead to development of ISI. Pia mater has been identified as a strong barrier against invasion of bacteria that cause bacterial meningitis, and rupture of the abscess into ventricle or subarachnoid space resulting in rapid deterioration of the clinical condition and neck stiffness.[6]The magnitude of the signs and symptoms of ISI are related to the site of localization, size and number of lesions, and magnitude of injury.[8]Leukocytosis and raised erythrocyte sedimentation rate are observed in 80% of patients with ISI.[20]Cerebrospinal fluid (CSF) examination is contraindicated in patients with a suspected intracranial occupying lesion.[18]With the advancement of radiological modalities and development of a great number of broad-spectrum antibiotics, the number of ISI cases has decreased over the past years. However, the number of immunosuppressed and human immunodeficiency virus (HIV)-positive patients with ISI has been increasing.[10,30]CNS tuberculosis is a form of ISI in which caseous necrosis and liquefaction of tuberculoma are the origins of the intracranial abscess.[25]In patients with acquired immunodeficiency syndrome (AIDS), cerebral tuberculous abscesses are observed as other space-occupying lesions when using radiological modalities.[31]*Toxoplasma* abscess, which develops from *Toxoplasma* encephalitis in patients with AIDS and cannot be distinguished from pyogenic abscess, consists of necrotic tissue and purulent fluid that does not include viscous, proteinaceous, and inflammatory debris.[23]

To further examine these findings, this study retrospectively evaluated the characteristics, treatment, and prognosis of ISI patients who presented at the study site between 1998 and 2013 in review of their clinical, radiological, and laboratory data.

MATERIALS AND METHODS

Sample preparation

All patients with ISI who had been treated and followed up at the Infectious Diseases and Clinical Microbiology Department at the study site between 1998 and 2013 were retrospectively evaluated by review of the data, including physical examination, laboratory (biochemistry and microbiology) testing, and radiological examination collected. All ISI patients had been treated and followed up at a tertiary hospital with an 800-bed capacity. ISI had been diagnosed on the basis of the review of radiological findings obtained by computed tomography (CT) initially performed to detect intracranial masses or hemorrhage and of magnetic resonance imaging (MRI) generally used for differentiation of masses or other lesions. Review of the findings and specimen testing of the patients who had undergone surgical intervention were performed to confirm diagnosis. All cases with postoperative brain abscess or epidural abscess were excluded from this study.

Each sample of the abscess was examined by microscopy with Gram, Ehrlich–Ziehl–Neelsen (EZN), and May–Grunwald staining for the trophozoite form of *Toxoplasma gondii*. Blood samples drawn from the veins or by catheter for culture were inoculated in bottles and processed manually or automatically using the BactAlert 3D (bioMérieux, Marcy-L'Etoile, France) or BACTEC system (Becton Dickinson, Sparks, MD), depending on availability. Fluid samples, including abscess, urine, and sputum samples, were inoculated onto 5% sheep-blood agar (Salubris Inc., Istanbul, Turkey), chocolate agar (Salubris Inc., Istanbul, Turkey), and MacConkey agar (Salubris Inc., Istanbul, Turkey). Susceptibility testing was performed using disc diffusion and an automated broth microdilution method (Vitek 2; bioMérieux, Marcy-L'Etoile, France) for bacteria. If required, the results were confirmed using the E test method (AB BIODISK, Solna, Sweden). The breakpoints defined by the Clinical and Laboratory Standards Institute were used to evaluate the results. Pus samples were incubated in an anaerobic jar with the GasPak System (BBL Microbiology Systems, Becton, Dickinson and Co., Cockeysville, MD, USA) equipped with a GasPak hydrogen and carbon dioxide-generating envelope for a period of at least 7 days.

Lumbar puncture had been performed in cases with suspected acute purulent meningitis on admission if there had been no contraindication. Each CSF sample collected from lumbar puncture, which contained at least 5 ml of fluid was centrifuged, and a portion of the deposit was examined by microscopy with Gram, EZN, and India ink staining. The remaining deposit was cultured on blood and chocolate agar and *Löwenstein*-Jensen medium (Salubris Inc., İstanbul, Turkey), as well as inoculated in a mycobacteria growth indicator tube (BBL MGIT medium, BD, Sparks, MD, USA) for later examination when the BACTEC system became available. Patients at high risk for HIV infection were tested for HIV and *Toxoplasma gondii* antibodies. Leukocytosis was defined as any value above 10,000 cells/mm^3.

Statistical analysis

Standard descriptive statistical methods were used. Patient age was described in terms of range, median, and mean ± standard deviation.

RESULTS

A total of 23 patients, of whom 17 (73%) were male, of a mean age of 38.21 ± 12.61 years (range: 19-67 years,

median: 34) with a mean symptom duration of 22.25 ± 20.22 days were identified and evaluated retrospectively. The distribution of cases by year was 11 cases between 1998 and 2000, 3 cases between 2001 and 2003, 4 cases between 2004 and 2008, and 5 cases between 2009 and 2013. Headache was the most common symptom [Table 1]. Frontal lobe was the most common localization of ISI and mastoiditis due to the fact that chronic suppurative otitis media was the most common source of infection causing ISI [Tables 2 and 3]. Sixteen (69%) patients underwent an invasive procedure, of whom 2 underwent open craniotomy, 12 burr hole aspiration, and 2 stereotactic biopsies due to the severity of signs and symptoms at admission, deteriorating clinical findings, and convulsion, respectively. *Proteus mirabilis*, *Pseudomonas spp.*, *Peptostreptococcus spp.*, *Enterococcus avium*, *Mycobacterium tuberculosis* complex, and *Toxoplasma gondii* were isolated from the specimens collected from 6 (37%) of the 16 patients who underwent invasive procedures. Lumbar puncture was performed in two cases with suspected acute purulent meningitis on admission and during treatment to assess treatment response. CSF culture yielded *Neisseria meningitidis* and *Streptococcus pneumoniae* in two patients who had developed ISI secondary to acute bacterial meningitis. *Proteus vulgaris* ($n = 2$), extended spectrum beta lactamase-producing *Escherichia coli* ($n = 1$), *Enterococcus avium* ($n = 1$), and *Pseudomonas spp.* ($n = 1$) were isolated from the culture of ear discharge samples. Leukocytosis was observed in 13 patients (11,527 ± 4552). The sizes of intracranial lesions were from1.2 × 0.8 to 4.3 × 4.1 cm.

Ceftriaxone(2 g intravenously every 12 h) combined with metronidazole (500mg intravenously every 8 h) was administered to patients between 21 and 120 days until clinical signs and resolution of radiological findings. Initial standard treatment was changed to a combination therapy including, vancomycin and meropenem in five patients and to meropenem therapy in two patients for 2 weeks owing to the severity of symptoms at admission and the deterioration of signs and symptoms under standard combination therapy. Toxoplasma encephalitis was diagnosed and treated with pyrimethamin (a loading dose of 200 mg followed by 7.5 mg/day), clindamycin (600 mg 4 times/day intravenously), and folinic acid (20 mg/day) in a patient with a multifocal abscess who was confirmed to have HIV infection after surgical intervention. A patient with multifocal intracranial abscess secondary to tuberculous meningitis was administered 300 mg/day of isoniazid, 600 mg/day of rifampicin, 2 g/day of pyrazinamide, 1.5 g/day of ethambutol, and 1 g/day of streptomycin for 5 months owing to a persistent abscess. Dexamethasone was administered intravenously for 4 weeks, followed by 2 weeks of oral prednisolone. Isoniazid and rifampicin were continued for 12 months in patient with tuberculous meningitis.

Table 1: Signs and symptoms of 23 patients with intracranial abscess at admission

Symptom or condition	*n*
Headache	22
Fever	14
Vomiting	9
Convulsion	7
Stiff neck	6
Unconsciousness	6
Focal neurological deficit	4
Ataxia	3
Ptosis	2
Abducens paralysis	2
Dysarthria	2
Facial paralysis	1
Hemiplegia	1

Table 2: Characteristics of 23 patients with intracranial abscess

	n
Anatomical location of intracranial abscess	
Solitary intracerebral abscess	17
Frontal lobe	6
Cerebellum	5
Temporal lobe	3
Temporoparietal lobe	2
Pontobulbar region	1
Multifocal intracerebral abscess	2
Subdural empyema	2
Frontal abscess with subdural empyema	1
Hypophyseal abscess	1
Source of infection causing intracranial abscess	
Mastoiditis with chronic suppurative otitis media	9
Sinusitis	4
Acute purulent meningitis	3
Trauma	2
Bronchiectasia and pulmonary abscess	1
Tooth abscess with patent foramen ovale	1
Miliary tuberculosis	1
HIV/AIDS	1
Pneumonia	1
Unknown	1
Type of intracranial abscess treated with surgical drainage	
Temporal lobe abscess with ventricular compression	2
Subdural empyema	2
Subdural empyema with cerebellum abscess	1
Subdural empyema with frontal abscess	1
Subdural and epidural empyema	1
Frontal lobe abscess	1
Hypophyseal abscess	1
Temporoparietal mass	1

HIV: Human immunodeficiency virus, AIDS: Acquired immunodeficiency virus

Table 3: Isolated microorganisms and presence of antibiotic treatment as specimens were taken

	Microorganism isolated from			
Patient	**Intracranial aspiration specimen**	**Ear discharge specimen**	**Cerebrospinal fluid specimen**	**Presence of antibiotic**
1	*Proteus mirabilis*	*Proteus vulgaris*		+
2	*Pseudomonas spp*	*Pseudomonas spp*		+
3	*Toxoplasma gondii*			+
4	*Peptostreptococcus spp.*			-
5	*Mycobacterium tuberculosis*			+
6	*Enterococcus avium*			+
7			*Neisseria meningitidis*	-
8			*Streptococcus pneumoniae*	-
9		*Proteus vulgaris*		
10		Extended spectrum beta lactamase-producing *Escherichia coli*		-
11		*Enterococcus avium*		+

Two patients underwent surgery after admission and 16 patients required surgical intervention between 3 and 18 days (range: 8 days) of medical treatment. Patients whose lesions are small and/or difficult to access were followed up with medical treatment and radiologically. One patient underwent surgical intervention twice and one underwent surgical intervention thrice, but only one patient underwent burr hole aspiration twice due to deterioration of signs and symptoms or lack of regression of the abscess under medical therapy. Persistent fever, lack of regression in the size of the abscess, deterioration of signs and symptoms, appearance of cranial nerve deficits or seizures may indicate failure of therapy. Mastoidectomy was performed in seven patients and functional endoscopic sinus surgery in three patients in addition to medical treatment. Anticonvulsive therapy was initiated in nine patients, of whom seven had undergone surgical drainage and two had multifocal intracerebral abscesses. Four of these patients continued to undergo anticonvulsive therapy regularly, while the other five underwent it for between 2 and 4 years until recovery of electroencephalography and radiological findings. Sequelae developed in two (8%) patients as generalized convulsive seizure in one patient and as both abducens paralysis and hemiplegia in the other patient who were admitted with pontobulbar abscess and hemiparalysis, respectively. Relapse did not occur in any patient. Two (8%) patients died, one of hydrocephalus developing in spite of ventricular shunting and surgical drainage and one of toxoplasmosis encephalitis and abscesses related to AIDS.

DISCUSSION

ISI is the most common suppurative infection of CNS requiring neurosurgical intervention.[27]The decrease in mortality from ISI since the 1950s may be associated with early diagnosis with developed radiological modalities, use of broad-spectrum antibiotics, and aggressive management.[27]While almost half of the cases described here were observed between 1998 and 2000, the incidence was observed as 1 case/year. Most reported cases of ISI have occurred in between 30 and 50 years of age, as observed in our study, in which headache, neurological signs and symptoms, and age between 30 and 50 years were distinguishing characteristics.[1,27,33] Accordingly, sources of infections leading to development of ISI are frequently observed in this age group and gender.[13,22]Predominance of male gender were considered to be related to the presence of mastoiditis and chronic suppurative otitis media in which male gender was reported to predominate.[33]

Fever and headache, which are also the main signs of acute bacterial meningitis, should be considered in the differential diagnosis of ISI. The concurrence of acute purulent meningitis with ISI is rarely observed as a complication of meningitis, as observed in the present study. Lumbar puncture performed to diagnose acute purulent meningitis should be avoided in patients who have neurological findings and headache accompanied by convulsions, focal neurological signs, and papilloedema owing to its hazards, including uncal herniation, rupture of an abscess cavity into the ventricle, etc., as evidenced by the death of 25 of the 140 ISI patients who underwent lumbar puncture.[26]ISI duration was reported as less than 1 week in 25% of our cases, among whom the mean was 22 days, suggesting that prolonged duration may be an indicator in the differentiation of ISI from acute purulent meningitis.[14]

A combination of isotope scanning and computerized axial tomography has been reported to allow for identification of an appropriate approach to treatment that can result in an immediate reduction in mortality in approximately 10% of ISI patients.[16] CT has also been found also useful for follow-up of patients.[17] MRI is more sensitive for early cerebritis, detecting satellite lesions, estimating the extent of central necrosis, ring enhancement, and cerebral edema and visualizing the brainstem compared with CT. Especially diffusion-weighted MRI

can differentiate ring-enhancing lesions due to brain abscess from neoplastic lesions.[12] CT which is crucial for early and differential diagnosis could be perfomed at admission in the emergency room. During follow-up at ward, they had undergone MRI as well. Although leukocytosis and raised erythrocyte sedimentation rate are observed in 80% of ISI patients, the leukocyte count was not excessively high in our cases.[17,20]Otogenic infection has been found to be the most common source of ISI, followed by sinusitis infection, odontogenic infection, postneurosurgery infection, bronchogenic infection, head trauma, congenital heart disease; the likely distant sources of ISI are pneumonia, osteomyelitis, complicated soft-tissue infection, etc., as reported.[27]Even though the number of otogenic cases has declined with improvement in medical treatment and surgical procedures, the number of cases with a history of neurosurgical procedures and distant focus of infection has been increasing.[27]The primary focus of infection has been associated with the anatomical location of ISI. Otogenic and odontogenic infections are associated with temporal and frontal abscess, while sinusitis is generally associated with frontal abscess.[13]Thus, sinusitis, otogenic, and odontogenic infection should be considered in the diagnosis and treatment of ISI. Hematogenous spread due to distinct sources of infection, such as pneumonia, and pulmonary abscess, as observed in our study, may cause ISI in the context of bacterial virulence, impairment of host immunity due to comorbid factors including liver cirrhosis and diabetes mellitus, and bacterial load.[17]

Blood culture has been reported to yield microorganisms in 35% of ISI cases.[12]Among the six (37%) patients whose specimens yielded microorganisms, no isolate predominated. Although five specimens obtained from patients with mastoiditis due to chronic suppurative otitis media yielded bacteria, the abscess and ear discharge cultures of only one patient yielded the same bacteria. *Peptostreptococcus spp*. was isolated from only one patient. *Fusobacterium* has been reported to be the predominant anaerobe among corresponding sinuses and abscesses, followed by *Prevotella* and *Peptostreptococcus* when samples are transported and cultured appropriately.[4]Alpha-hemolytic *Streptococcus* spp. has been reported to be a common pathogen for ISI, followed by *Proteus mirabilis*, *Peptostreptococcus spp.*, *Staphylococcus aureus*, *Haemophilus influenzae* type b, Microaerophilic streptococci, and *Bacteroides ureolyticus*.[16]Sampling under antibiotic treatment is more likely to be related to low positive culture rates. Previous studies have not found a correlation between sinus cultures and ISI.[13]

Tuberculous abscess is rarely seen and usually presents acutely in the third and fourth decades in the supratentorial location with focal neurologic signs.[32]As such, it can be missed unless EZN staining, Löwenstein–Jensen culture, and other microbiological techniques are performed using an abscess sample.[13]Tuberculous abscess may develop after initiation of antituberculous treatment to treat pulmonary or other forms of tuberculosis.[7]Tuberculous brain abscess may be observed with *Toxoplasma* encephalitis or brain abscess concurrently after initiation of highly active antiretroviral therapy as well.[11]While Toxoplasma encephalitis cannot be identified in AIDS patients unless neurosurgical sampling from the cranial mass yields the bradyzoite form of *Toxoplasma gondii*, it can be diagnosed and treated, depending on the clinical and radiographic response to specific antitoxoplasma therapy.[29]Although clindamycin and sulfadiazine have been reported as effective treatments for *Toxoplasma* brain abscess, the patient in our study who was administered these agents died.[9]

Ceftriaxone combined with metronidazole achieved cure among our patients, without the need for a broader spectrum antibiotic. As this finding indicates, treatment of ISI should be based on antimicrobial therapy active against possible causative pathogens and able to penetrate the blood–brain barrier. Infected brain tissue and abscess are treated with only antimicrobial therapy for 4–6 weeks for patients with abscesses <2 cm in diameter, high-density lesions, multiple abscesses, unsuitable lesions, or poor conditions for surgical intervention.[5,21]In total, seven of our patients required escalated therapy including vancomycin and meropenem. Cure of ISI due to resistant bacteria, such as extended-spectrum beta-lactamase producing gram-negative bacilli or penicillin-resistant *Viridans streptococci*, or with tooth abscess drainage associated with methicillin-resistant *S. aureus* necessitates the usage of broader spectrum antimicrobial therapy.[24] A patient's history of neurosurgical operation; infection with resistant bacteria, such as methicillin-resistant *S. aureus*, carbapenem-resistant *Pseudomonas aeruginosa*, or *Acinetobacter baumannii*; and admission from settings with high antimicrobial resistance rates should be taken into consideration when selecting an antimicrobial therapy.

ISI accompanied by subdural empyema was the most common cause for surgical intervention in our cases. Our surgical intervention rate (69.5%) was both higher and lower than previously reported rates (62% and 76%).[1,14,16,17,26,27,33]The timing of surgical intervention among our cases was reported as within 72 h and 12 days of treatment, as most had undergone intervention after 1 week of treatment. Surgical intervention should be evaluated in patients whose condition is deteriorating, as manifested by the emergence of neurological signs and symptoms and/or unresponsiveness to medical therapy at admission or during follow-up. The number of our patients who had undergone surgical intervention to treat the source of ISI was nearly equal to the number of who had undergone surgical intervention to treat ISI. Our findings indicate that ISI patients should be evaluated to identify the source of the infection and a means of surgical intervention that can achieve a cure without relapse or sequelae.

Although relapse did not occur among our cases, it has been reported in patients who have undergone surgical intervention and inadequate therapy. Corticosteroids are recommended perioperatively to reduce intracranial pressure, although no well-controlled, randomized clinical study of their effectiveness has been performed.[19]

CONCLUSION

Delayed diagnosis of ISI, rapid progression of disease, coma, multiple lesions, intraventricular rupture, lower Glasgow coma score at admission, and fungal etiology have been reported as poor prognostic factors.[24]In our patients, timely and appropriate management of ISI was likely associated with the lower sequelae rate (8.6%) compared with previous studies, two of which reported rates of 37% and 44%.[1,14,16,17,26,27,33] Likewise, the mortality rate (8.6%) among our patients was much lower than that reported in previous studies, which reported rates ranging from 8% to 40%.[14,17,19,27,28]Although our patients experienced longer duration of signs and symptoms, the absence of poor prognostic factors among them was likely related to their low mortality rate. However, we are aware that the increasing number of HIV cases worldwide may lead to reversal of the decreasing rate of AIDS-related ISI, especially with *Toxoplasma gondii*, as observed in our cases. We therefore recommend that all patients with the ISI or mass images on CT or MRI should be tested for HIV. We also recommend that male patients with headache and neurological signs and symptoms, whether with or without fever, be evaluated for ISI on admission for early diagnosis and provision of timely, adequate therapy and, if necessary, surgical intervention to reduce mortality and sequelae rates.

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