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Case Report

Rapid Development of Brain Abscess Caused by Streptococcus Pyogenes Following Penetrating Skull Injury via the Ethmoidal Sinus and Lamina Cribrosa

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Objective : Streptococcus pyogenes is a beta-hemolytic bacterium that belongs to Lancefield serogroup A, also known as group A streptococci (GAS). There have been five reported case in terms of PubMed-based search but no reported case of brain abscess caused by Streptococcus pyogenes as a result of penetrating skull injury. We present a patient who suffered from penetrating skull injury that resulted in a brain abscess caused by Streptococcus pyogenes.

Methods : The patient was a 12-year-old boy who fell down from his bicycle while cycling and ran into a tree. A wooden stick penetrated his skin below the right lower eyelid and advanced to the cranium. He lost consciousness on the fifth day of the incident and his body temperature was measured as 40°C. While being admitted to our hospital, a cranial computed tomography revealed a frontal cystic mass with a perilesional hypodense zone of edema. There was no capsule formation around the lesion after intravenous contrast injection. Paranasal CT showed a bone defect located between the ethmoidal sinus and lamina cribrosa.

Results : Bifrontal craniotomy was performed. The abscess located at the left frontal lobe was drained and the bone defect was repaired.

Conclusion : Any penetrating lesion showing a connection between the lamina cribrosa and ethmoidal sinus may result in brain abscess caused by Streptococcus pyogenes. These patients should be treated urgently to repair the defect and drain the abscess with appropriate antibiotic therapy started due to the fulminant course of the brain abscess caused by this microorganism.

KEY WORDS : Brain abscess · Skull base repairing · Penetrating head injury · Streptococcus pyogenes · Surgical evacuation.

INTRODUCTION

Streptococcus pyogenes is a beta-hemolytic bacterium that belongs to Lancefield serogroup A, also known as group A streptococci (GAS)[8,23]. It has rarely been reported as a cause of brain abscess. There have been five reported cases in terms of PubMed-based search but no reported case of brain abscess caused by Streptococcus pyogenes as a result of penetrating skull injury[2,10,11,14,17]. Brain abscesses may occur due to different etiologies including a lung abscess in adults, congenital heart disease in children, immunocompromised patients, pulmonary arteriovenous fistulas, bacterial endocarditis, gastrointestinal infections, septic embolization, contiguous spreading such as purulent sinusitis, middle-ear and mastoid air sinus infection[2,6,7,9,12,15,19]. In addition, neurosurgical procedures and penetrating cranial trauma may lead to brain abscess, and patients with malignant disease may be predisposed to them[1,3,4]. We present this case to emphasize prompt evaluation and the treatment of the underlying factors with the support of antibiotic therapy and to outline the characteristic clinic features of Streptococcus pyogenes abscess.

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CASE REPORT

A 12-year-old previously healthy boy was transferred to our center in comatose condition. His physical examination while in the emergency department revealed a blood pressure of 100/80 mm/Hg and heart beat of 125 per minute. The Glasgow coma scale value was 6/15, the body temperature was 40°C and there was 4+ nuchal rigidity. Physical exami-

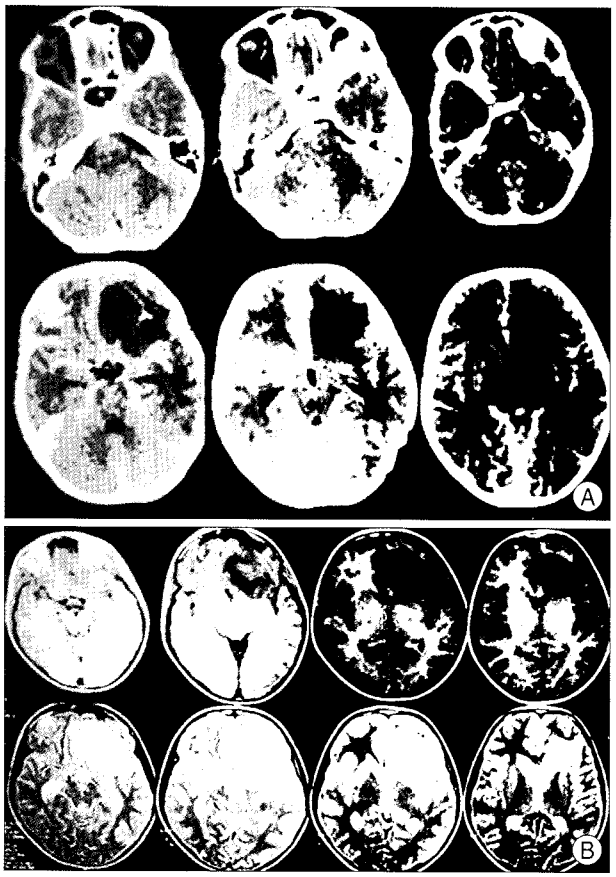

Fig. 1. Preoperative contrasted computed cranial tomography and noncontrasted cranial magnetic resonance image scan. A : Contrasted cranial computed tomography showing mild shift and hypodense lesion causing shift from the left to the right at left frontal lobe and no capsule formation. B : On the fourth day of the incident, preoperative noncontrast cranial magnetic resonance image showing hypointense abscess cavity in T1WI images and hyperintense abscess cavity in T2WI images.

nation showed a skin perforation that was 0.5 cm long and located next to the nose at the right infraorbital region. His medical history revealed no abnormality except minor trauma. He had fallen off his bicycle 5 days ago while cycling around his neighborhood and run into a tree and a wooden stick about 12 cm long and 4 mm wide. The stick had penetrated his skin 2 cm below the right lower eyelid and advanced to the cranium. After the incident, he had taken out the inserted wooden stick from his face himself and his family noticed no abnormality. He felt very tired and sleepy afterwards and his family noticed a fever. He was taken to the local hospital on the third day of the incident where he lost consciousness completely and his body temperature was measured at 40°C on the fifth day of the incident. He was then transferred to our hospital within two hours and the patient immediately underwent surgery after undergoing basic tests and cranial computed tomography (CT). A complete blood count showed a white blood cell count of 25,000/mm^3 with 82.8% neutrophils, C-reactive protein level of 145.7 mg/L and sedimentation rate of 74 mm/hour. Biochemistry and blood coagulation parameters were within normal limits. A cranial CT scan at the time of admission revealed a 4×3×3 cm right frontal cystic mass with a perilesional hypodense edema zone, and no capsule formation around the lesion after intravenous contrast injection (Fig. 1A). Paranasal CT showed a defect between the lamina cribrosa and the ethmoid sinus (Fig. 2A). Empirical antibiotic therapy was promptly started before the surgery [ceftriaxone (100 mg/kg), vancomycin (60 mg/kg), metronidazole (30 mg/kg)]. The patient underwent surgery an hour after his admission to our hospital. The head was fixed in the extension position with a three-pin holder, while the patient was in the supine position. A bicoronal skin incision and bifrontal craniotomy were performed. The anterior third of the superior sagittal sinus was ligated and the dura mater opened. The right frontal lobe was minimally retracted and a bone defect at the lamina cribrosa which was connected to the ethmoidal sinus, and torn dura mater were found. The bone defect was 0.8 cm^2 in size and the dural tear was observed at the right and left anterior base of the frontal lobes (Fig. 3). The bone defects were repaired with two pieces of bone harvested from the internal tabula placed on the defective area and Glubran®2 (GEM s. r. I., Viareggio, Italy) was

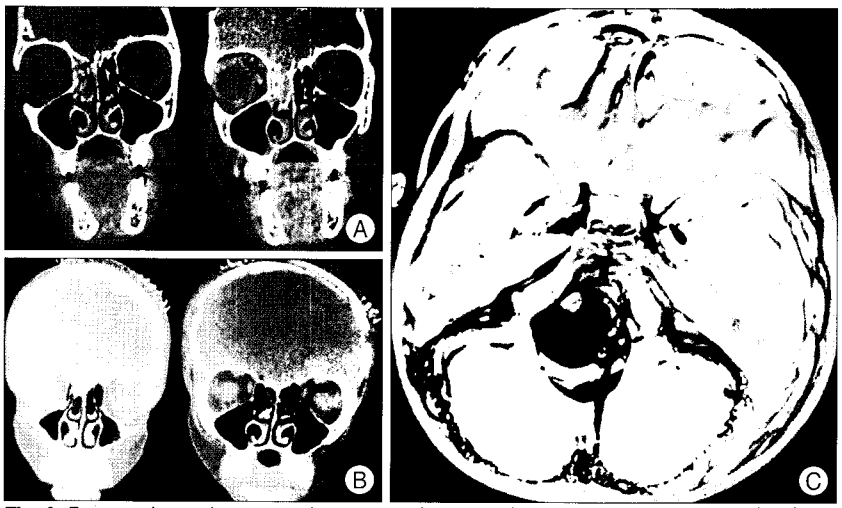

Fig. 2. Preoperative and postoperative paranasal computed tomography and postoperative three-dimensional cranial computed tomography. A : Preoperative paranasal computed tomography showing the defective area of the bone located between ethmoidal sinus and lamina cribrosa. B : Postoperative paranasal computed tomography scan showing the repaired part of the bone defect and resolution of the edema and hemorrhage around the ethmoidal sinus. C : Postoperative three dimensional cranial computed tomography showing the repaired part of the bone defect.

poured on it to stick the harvested bone on the defective area and prevent any passage between the ethmoidal sinus and the cerebrum (Fig. 2). The dural tear was sutured and muscle tissue harvested from the temporal muscle placed on the repaired area. The middle frontal gyrus was then dissected and the abscess was reached at a depth of 2 cm. A total of 60 mm^3 abscess material was aspirated and the aspirated material was transported appropriately for aerobic and anaerobic culture. Cultures from the abscess grew beta-hemolytic Streptococcus pyogenes [group A streptococcus (GAS)] as the sole isolate; no anaerobes were identified. Postoperatively the patient's clinical condition was better, and he was fully conscious with no motor deficit. The body temperature, white blood cell count and C-reactive protein levels decreased dramatically day by day (Fig. 4, 5, 6). Periodic cranial CT was performed on the postoperative 3rd, 10th, 22nd, 32nd and 43rd day. A cranial CT on the 3rd postoperative day showed no shift and no contrast enhancement around the lesion, but there was edema formation around the hypodense abscess cavity (Fig. 7). Follow-up CT on the 10th, 22nd, and 32nd days revealed no shift, but there was contrast enhancement around the lesion and edema formation in the frontal lobe (Fig. 8, 9, 10). The contrast enhancement and the size of the edema gradually decreased. The last follow-up cranial CT performed on the postoperative 43rd day revealed no contrast enhancement and little edema (Fig. 11). The patient was discharged with no neurological deficit and the observation of remarkably decreased edema and loss of contrast enhancement.

DISCUSSION

Brain abscess treatment consists of a combination of antibiotics, surgical intervention, and eradication of the primary origin of infection. Medical treatment was preferred in some large brain abscess series for patients for abscesses < 2 cm in diameter, high density lesions, multiple abscesses, clinically stable patients, those who are poor candidates for surgery, and for patients with surgically inaccessible lesions[6,7,18,19,24]. Kao et al.[16] surgically treated patients with abscesses > 2 cm in size (average abscesses diameter was 3.75 cm and range 2-6 cm). The depth of the lesion within the brain was the main determining factor for the approach to these lesions rather than the lesion's location. Superficial and encapsulated abscesses were excised. Abscesses located in deeper critical regions (e.g., brain stem, cerebellum, or diencephalic structures adjacent to the ventricle) should be stereotactically drained or a biopsy taken for culture and tissue diagnosis[13,22,25]. Staphylococcus aureus and enterobacteriaceae are commonly isolated from brain abscesses following head injury and postoperative infections[7,19,24]. Otic infection and sinusitis may lead to an infection caused by abscess formation adjacent to the brain tissue[6,7,19,24]. In our case, the wooden stick penetrating the ethmoidal sinus and lamina cribrosa would be contaminated with the flora of the ehmoidal sinus and transport the

Fig. 3. Intraoperative appearence of the bone defect located between ethmoidal sinus and lamina cribrosa.

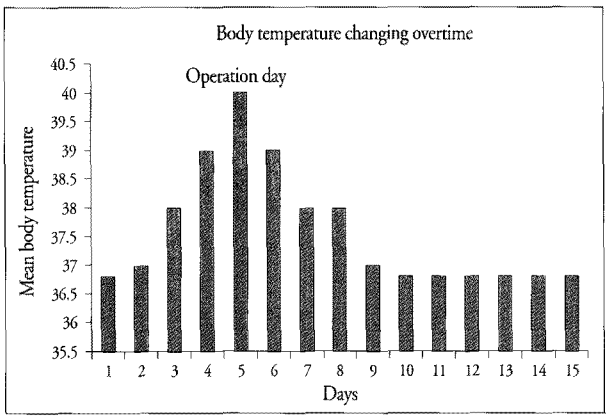

Fig. 4. Changing of the body temperature overtime.

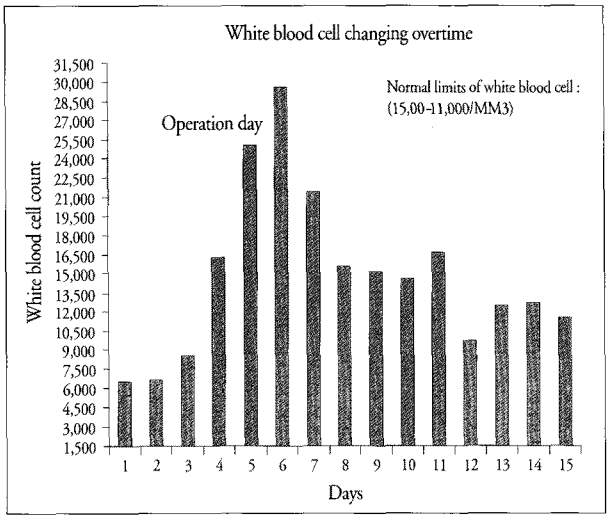

Fig. 5. Changing of the white blood cell count overtime.

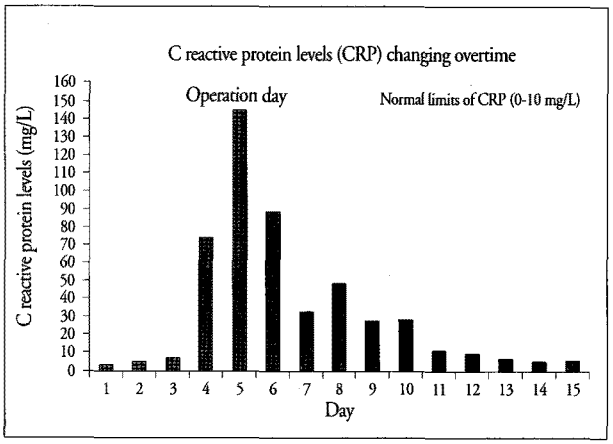

Fig. 6. Changing of the C-reactive protein levels overtime.

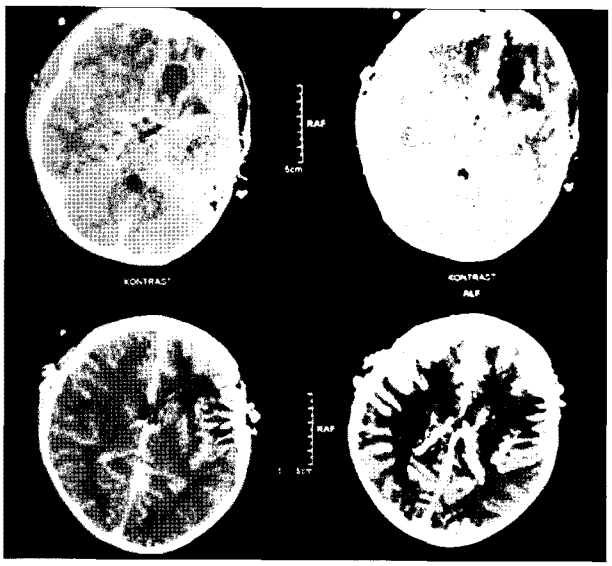

Fig. 7. Contrasted computed cranial tomography scan on the third day of the operation, showing no shift and no contrast enhancement around the lesion, but there is edema formation around the hypodense abscess cavity.

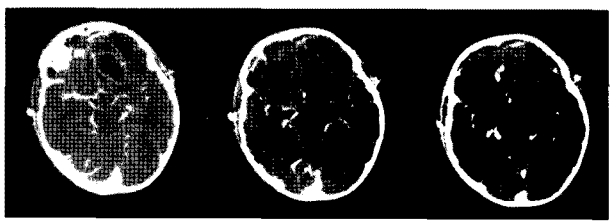

Fig. 8. Contrasted computed cranial tomography scan on the 10th day of the operation showing contrast enhancement around the abscess cavity and edema formation in the frontal lobe and no shift.

Table 1. The programme of the antibiotic treatment

Antibiotic	Duration of the Treatment	Dosage*
Vancomycin HCl	From the 1st to 43rd day	4 × 450 mg§
Tienam†	From the 21st to 43rd day	3 × 1 gr§
Meropenem	From the 1st to 12th day	3 × 1 gr§
Ceftriaxone‡	From the 12th to 21st day	2 × 200 mg§
Metronidazole	From the 1st to 12th day	3 × 400 mg§

*Application route of the antibiotics, †Thienamycin 500 mg + Cilastatin 500 mg, ‡Ceftriaxone disodium, §Intravenous

microorganisms from the ethmoidal sinus to the cerebrum through the lamina cribrosa. We repaired the defect located at the lamina cribrosa and connected with the ethmoidal sinus as it would be an ascending infection source otherwise. Repairing this defect prevents recurrent infection through this route. We immediately started empirical antibiotic therapy (vancomycin, metronidazole, meropenem) and we performed the operation under antibiotic therapy. Following bifrontal craniotomy, we repaired the defect, located at the base of the anterior fossa, with pieces of the tabula interna harvested from the calvarium and then sutured and sealed the duramater with Glubran® (GEM s. r. I., Viareggio, Italy). The last step was to drain the abscess. Brain abscess culture yielded Streptococcus pyogenes [group A streptococcus (GAS)] on the fourth postoperative day, and we decided to continue this treatment because of the good clinical response to these antibiotics. We modified the antibiotic therapy during the postoperative 12th and 21st days due to some allergic reactions (the antibiotic programme is presented in Table 1). Persistent neurological sequelae were reported in 44 to 65% of children with brain abscess secondary to other pathogens in the larger published series[7,22]. There have been five cases reported in the literature regarding brain abscess caused by Streptococcus pyogenes and all had a better prognosis than those caused by the more common CNS pathogens. None of the reported cases had a history of penetrating injury to the cranium[10,11,14,17,21]. We believe that penetrating skull injury has a high risk regarding development of an intracranial abscess. The clinical condition of the patient may worsen within days once intracranial penetration occurs and lead to mortality in cases with no proper and immediate treatment. Evacuation of the abscess, repair of the defect and proper antibiotic therapy should be employed together in patients with a penetrating skull injury. In addition, any skull penetration with an object (such as a wooden stick) showing a connection with the ethmoidal sinus should arise the suspicion that this object may transport the microbial flora of the sinuses to the cerebrum. Miller et al.[20] reported 42 cases of intracranial injury following periorbital wounds and half of these patients died because of a brain abscess. Bursick et al.[8] reported 21 cases of intracranial penetrating injury and pencils were the cause in 15 of these cases. Staphylococcus aureus was the most frequently microorganism in this series. Bert et al.[6] reported a brain abscess due to penetrating periorbital injury by a wooden stick caused by Bacillus macerans, a very unusual bacterial etiology[5]. We found five brain abscess cases caused by Streptococcus pyogenes with a Pub-

Med-based search. Two had otitis media while three had no predisposing factors or trauma[10,11,14,17,21]. Our case is the first reported brain abscess caused by penetrating skull injury. The abscess culture produced Streptococcus pyogenes following penetrating skull injury. The traversing object was directed toward the ethmoidal sinus and lamina cribrosa, which is very thin and allows easy access to the frontal lobes. The organism may have been present on the wooden stick, or may have come by contamination from the skin or ethmoidal sinus, and the microorganisms can be transported into the cerebrum. Streptococcus pyogenes is rarely seen, and is grown in only 2% of sinusitis and otitis media cultures. We believe that our patient had this organism within the ethmoidal sinus and that the wooden stick became contaminated and carried the organism to the cerebrum. The noncontrast cranial magnetic resonance imaging showed a 14 mm shift from the left to the right at the left frontal lobe. The lesion size was at 5 × 4 × 4 cm and it was hypointense on T1WI and hyperintense on T2WI images on the fourth day of the incident (Fig. 1B). Cranial tomography performed on the fifth day of the incident, during the patients admission, showed no contrast enhancement but there was a hypodense lesion and edema around the lesion (Fig. 1A). The patient's clinical condition progressively deteriorated within five days. Brain abscess caused by Streptococcus pyogenes as a result of penetrating skull injury may show progressive deterioration. Preventing ascending infection to the cerebrum through the lamina cribrosa and the ethmoidal sinus is as important as evacuating the abcess formation. The cranial defect connected with the ethmoidal sinus may be a source of ascending infection in cases with dural and arachnoid tearing. We repaired the defect with bone and glue to prevent such an ascending infection. All infection parameters and fever were reduced after the operation. In addition, the patient's clinical condition improved dramatically and he was treated with antibiotics for 43 days. We correlated this clinical improvement with serial contrast enhanced tomographies over time. The contrast enhanced tomography images showed a gradually decreased abscess size and edema formation with the last cranial tomography on the 43rd day of antibiotic therapy showing no contrast enhancement and little edema formation. We believe that any cranial bone sinus defect connected to the brain requires attention to prevent recurrent infection by transmitting a source of infection. The defect should be repaired initially, and this may be followed by abscess evacuation. In addition, brain abscess caused by Streptococcus pyogenes in patients with a penetrating skull injury may show a fulminant clinical course and have a fatal outcome.

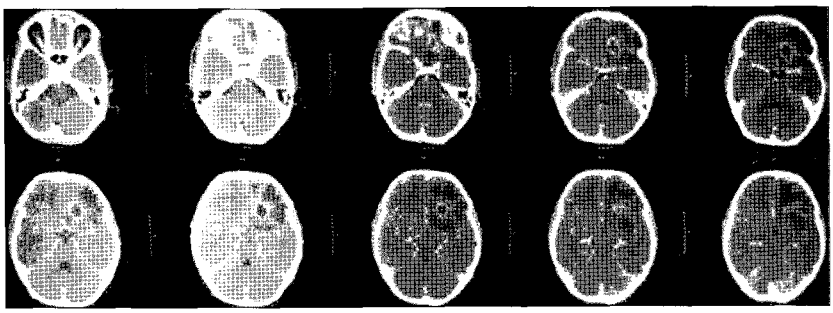

Fig. 9. Contrasted computed cranial tomography scan on the 22 nd day of the operation showing contrast enhancement around the abscess cavity and edema formation in the frontal lobe and no shift.

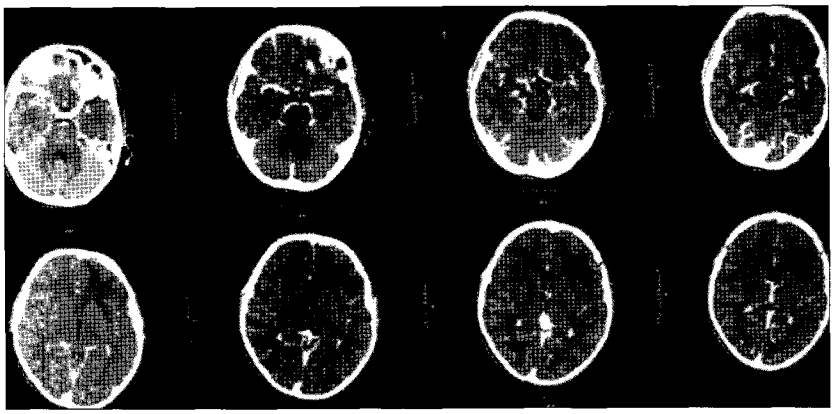

Fig. 10. Contrasted computed cranial tomography scan on the 32 nd day of the operation showing contrast enhancement around the abscess cavity and edema formation in the frontal lobe and no shift. In addition, the edema formation decreased.

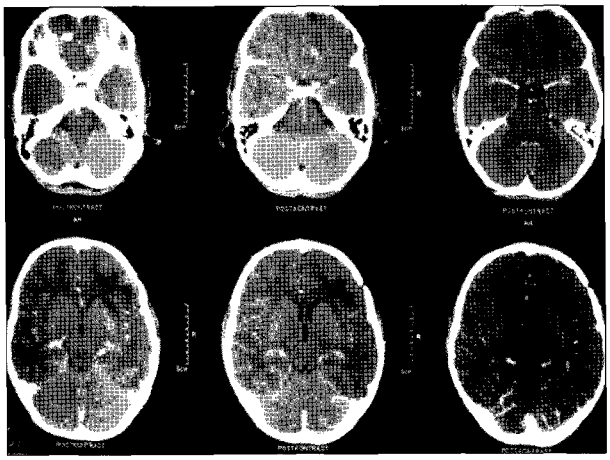

Fig. 11. On the 43rd day of the operation, contrasted computed cranial tomography scan showing no contrast enhancement and no abscess cavity, but very little edema formation at left frontal lobe.

CONCLUSION

In contrast to previous reports, brain abscess caused by Streptococcus pyogenes may show a fulminant course and create a life-threatening clinical condition especially in patients with a penetrating skull injury. All brain abscess should be evaluated promptly and possible cranial defects associated with cranial bone sinuses should be repaired as soon as possible to prevent an ascending source of infection.

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