

Spontaneous Subdural Empyema Secondary To Escherichia Coli Infection: Case Report

Pantangco, J. O. , Alvarez, R. M., Delfino, J. M.

Neurology Resident, Makati Medical Center, Department of Neurosciences, Section of Neurology,
Amorsolo Street, Legazpi Village, Makati, Metro Manila, Philippines 1229
jaopantangco@gmail.com

Consultant, Makati Medical Center, Department of Neurosciences, Section of Neurology,
Amorsolo Street, Legazpi Village, Makati, Metro Manila, Philippines 1229
benjiealvarez@yahoo.com

Neurology Resident, Makati Medical Center, Department of Neurosciences, Section of Neurology,
Amorsolo Street, Legazpi Village, Makati, Metro Manila, Philippines 1229
jeanpaolomd@gmail.com

Abstract: Subdural Empyema (SDE) is an uncommon intracranial infection commonly caused by staphylococcal or streptococcal infection via contiguous spread or intracranial surgery. This disease entity is not immediately considered in patients who develop a subdural lesion because of its non-specific clinical presentation and radiologic features. Our patient was managed initially as a spontaneous subacute subdural hematoma with concomitant E. coli urinary tract infection (UTI), and later treated as a case of E. Coli positive subdural empyema. Prompt diagnosis and adequate treatment is critical because SDE is a neurosurgical emergency associated with a high mortality.

Keywords: Spontaneous Subdural Empyema, E. Coli, Urinary Tract Infection

1 INTRODUCTION

Acute febrile illness with rapidly progressive neurological deterioration characterizes subdural empyema (SDE), which may be fatal if left untreated [1]. More commonly, SDE arises from local invasion. Streptococcus pneumoniae is isolated in empyema from mastoiditis or paranasal sinusitis, whereas Staphylococcus aureus is seen in post-operative and post-traumatic empyema [2]. We present a case of spontaneous E. Coli SDE managed initially as an acute subdural hemorrhage with concomitant urinary tract infection.

CASE REPORT

A 63-year-old male with a prior history of spontaneous non-traumatic T₁₀-T₁₂ epidural hematoma was brought to the emergency room presenting with sudden onset slurring of speech, right facial asymmetry and right-sided extremity numbness. He had no recent head trauma, and had treatment for urinary tract infection 6 months prior. Upon arrival at the emergency room, vital signs were stable. Patient was afebrile. Pertinent neurologic findings showed mild dysarthria, with right central facial paresis, and hypalgesia of the right sided extremities. Plain Magnetic Resonance Imaging (MRI) of the brain revealed a mixed diffusion restriction, T1 hypointense, T2-FLAIR hyperintense lesion along the left cerebral convexity compressing on the left cerebral hemisphere (Figure 1). Given his prior history of spontaneous epidural hematoma, he was managed as a spontaneous subdural hemorrhage and treated accordingly. On his second hospital day he developed a low-grade fever. Urinalysis was positive for a urinary tract infection, and was treated with IV cefuroxime.

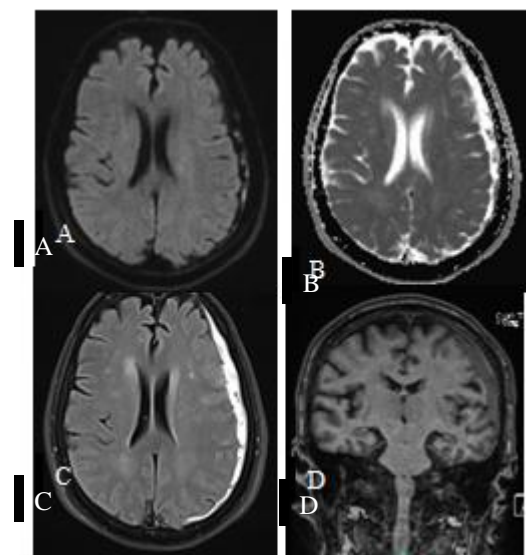


Figure 1: Plain Axial Cranial MRI images showing subdural fluid collection along the left cerebral convexity with a maximal thickness of 10 mm. This was shown to be mixed hypointense and hyperintense in Diffusion Weighted Imaging (DWI), A; Hyperintense in Attenuated Diffusion Coefficient (ADC) B; Hyperintense in T2-FLAIR, C; and Hypointense on coronal section of T1, D.

His symptoms transiently improved, but fever still persisted. A follow up cranial CT scan on the 3rd hospital day revealed no interval change in the maximal thickness of subacute subdural lesion along the left cerebral convexity (Figure 2). Urine culture was positive for E. coli, antibiotic was shifted to ertapenem.



Figure 2: Follow up Axial Plain Cranial CT image showed no interval change in the maximal thickness of subacute subdural hemorrhage along the left cerebral convexity.

On the 5th hospital day, still febrile, patient developed recurrent episodes of aphasia. His sensorium also began to deteriorate. Repeat plain cranial MRI done revealed no change in size of the subdural lesion. The patient eventually developed focal motor tonic-clonic seizures of the right-sided extremities. Repeat imaging showed no progression of the subdural lesion. Anti-epileptic medications were started but seizure control was difficult. A decision was made to surgically evacuate of the left subdural lesion. On the 7th hospital day, left subdural hemi-craniectomy was done which visualized the empyema collection (Figure 6).



Figure 6: Intraoperative findings of purulent material originating from the subdural space during craniotomy.

Post-operatively, the patient's seizures and fever continued which were eventually controlled in the Intensive Care Unit. Blood culture done was negative. Culture of the empyema was positive for E. coli and empiric treatment was shifted to Cefuroxime and Fosfomycin. He was eventually brought out of the ICU on the 16th hospital day and discharged on the 25th hospital day with dysarthria as the only residual deficit.

2 DISCUSSION

Subdural Empyema (SDE) is an intracranial purulent infection in the subdural space. This disease is more common in children but may infrequently occur in adults. In about 90% of cases of SDE, the initial prominent symptom is headache. Focal neurologic signs develop afterwards, with more than 85% of cases develop contralateral motor deficits. Aphasia may occur when the dominant hemisphere is involved. If infection is left untreated, rapid neurologic

deterioration would occur manifesting as focal or generalized seizures, decreasing sensorium, and ultimately coma and death [1]. Our patient initially presented with headache, right-sided neurologic deficits, and eventually developed fever, progressing to aphasia and persistent seizures. SDE in adults frequently occurs either as a complication of meningitis, direct extension from sinusitis or otitis media, cranial surgery or traumatic injury [4]. Spontaneous SDE is rare. In about 40% of spontaneous SDE, distant infection with the same bacteria is seen, hence a hematogenous route is considered. In most cases, the etiologic agents are Streptococcus or Staphylococcus [2]. To date, there are only a few reported cases wherein E. Coli is the offending agent of a spontaneous SDE, making E. Coli a rare, but important causative agent for SDE [5]. Contrast-enhanced MRI is the imaging study of choice because it has a higher sensitivity for detection of small subdural fluid collections. SDE are isointense on T1-weighted imaging, hyperintense on T2-weighted image and are rim-enhancing. SDE has high signal intensity on DWI and low signal intensity on ADC maps. Viscosity of the purulent material in empyema is thought to cause restriction in diffusion [6]-[7]. Contrast-enhanced CT is not as specific as MRI, but may also be done, showing rim-enhancement [7]. In our patient, plain MRI revealed hypointense on T1 and hyperintense on T2-FLAIR, but without diffusion restriction in DWI. Given his prior history of a spontaneous epidural hematoma and initial lack of fever, we were led to believe the initial imaging was a subacute spontaneous subdural hemorrhage. The limitation in our case was the lack of contrast-enhancement. Surgical evacuation of empyema and culture-guided use of intravenous antibiotics for 4-6 weeks is the treatment of choice [5]. Delay in surgery leads to clinical worsening and poor outcomes. Seventy percent of patients who did not receive surgical intervention within 72 hours developed significant disabilities [1]. Though our patient's operation had been done on the 7th day of presentation, he was discharged with minimal neurologic deficit.

3 CONCLUSION

SDE is an uncommon but rapidly fatal occurrence which needs to be ruled out in patients with focal neurologic deficits and fever. Clinical suspicion should be made even if there is no previous trauma, evidence of adjacent infection or with incompatible imaging findings, as delays in management can cause significant morbidity or mortality.

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AUTHOR PROFILE



Pantangco, Jose O. received the B.S. in Biology in Ateneo De Manila University, Philippines in 2008 and M.D. in Medicine and Surgery in University of Santo Tomas, Philippines in 2012. He is currently a 4th year Neurology resident in Makati Medical Center, Philippines.



Alvarez, Raquel M. Received her B.S. in Psychology and her M.D. in Medicine and Surgery in the University of Santo Tomas, Philippines in 1984 and 1988, respectively. She is a member of the Philippine Neurologic Association since 1998 and is currently the head of the Acute Stroke Unit and Neurology Intensive Care Unit in Makati Medical Center, Philippines.



Delfino, Jean M. is an M.D. graduate from Our Lady of Fatima – College of Medicine in 2013. He is currently a 2nd year Neurology resident in Makati Medical Center, Philippines.