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Intracranial gas containing epidural abscess in a closed traumatic head injury

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ABSTRACT

This paper is intended as an illustrative teaching case. It presents a prototype of a closed traumatic head injury with left frontal convexity extra-axial gas containing abscess, not conclusive on CT scan. The diagnosis was, however, made intra-operatively. It went further to discuss the diagnostic challenges and management pearls of such patients and concluded with a few pertinent take-home messages..

Keywords
aerocele,
non-foul smelly culture negative abscess,
traditional practice

CASE PRESENTATION

A 17-year-old right-handed immunocompetent Nigerian male, who hit his head against a hanging rail while playing with his friends 7 days prior to presentation, sustained injury to the scalp which was dressed with traditional concoction. He had no loss of consciousness, bleeding from craniofacial orifices or features of raised intracranial pressure (ICP). He however, presented with a 3-day history of altered sensorium, progressive right hemi-body weakness and right focal tonic-clonic seizure lasting 3-10 minutes, averaging 5 episodes per day, with subsequent increasing frequency, necessitating admission. No history of fever.

At presentation to Modibbo Adama University Teaching Hospital Yola, in northeast Nigeria, he was drowsy with Glasgow coma scale score (GCS) of 10 (E3, V2, M5), sluggishly reactive equal pupils, right supranuclear facioparesis and right spastic hemiparesis (with Medical Research Council -MRC- power grade 0). He had a 2x3cm irregular shaped contaminated left frontal scalp wound. However, his vital signs were within normal limits. We made a clinical diagnosis of moderate head injury (GCS 10), with right extra-axial collection likely subacute extradural haematoma.

Cranial computed tomography (CT) scan revealed left frontal scalp soft tissue swelling and aerocele, with no underlying calvarial fracture. There was no evidence of sinus or otologic infection. There was however, a left frontal convexity biconvex ring enhancing air-fluid 6.06

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cm mass, abutting on the calvarium, with perilesional oedema, 8.55mm ipsilateral subfalcine herniation and some effacement of the lateral ventricles. We made a diagnosis of left frontal convexity subacute haematoma and aerocele (Figure 1a to c) and the patient was scheduled for urgent left frontal mini-craniectomy and evacuation of left frontal epidural subacute hematoma.

However intraoperatively we found a left frontal scalp wound with purulent discharge, an underlying intact calvarium and egress of approximately 30 millilitres of non-foul-smelling yellowish purulent effluent under pressure following the craniectomy, which was evacuated. The cavity was irrigated with warm saline containing antibiotics until there was egress of clear fluid containing minimal debris. The wound was copiously irrigated and closed with interrupted mattress sutures. He was thereafter, placed empirically on triple parenteral antibiotics consisting of ceftazidime, vancomycin and flagyl for 4 weeks despite the culture yielding no growth and did well. He regained some power in the right lower limb (MRC of at least 3) on the second postoperative day and sat out of bed by himself on the third postoperative day. He was discharged upon completion of parenteral antibiotics on 2 weeks of oral antibiotics, with Glasgow coma scale extended (GOSE) of upper normal and modified Rankin score (mRS) of zero. The post-operative CT scan done at discharge revealed complete resolution of the abscess, with normal brain parenchyma and ventricles (Figure 1d to f). He was seen at 3 months post-discharge with no sequelae.

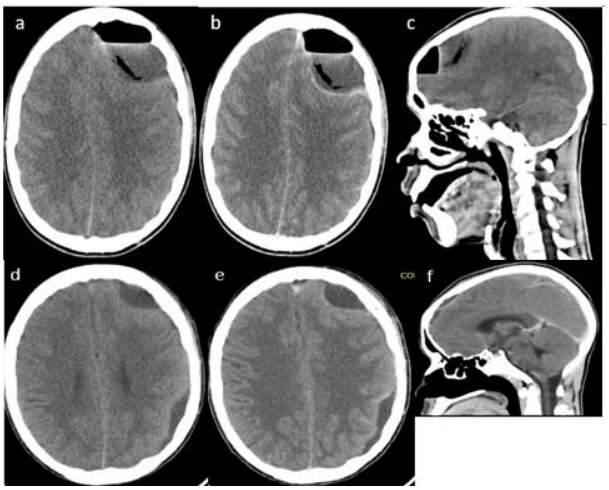

Figure 1. (pre-operative image-a to c)- Cranial CT of a 17-year old with moderate head injury, early focal right tonic-clonic seizure, right supranuclear facioparesis and right spastic hemiparesis showing ring enhancing right frontal air-fluid mass with midline shift. (a) axial pre-contrast, (b) axial post contrast, (c) sagittal pre-contrast. (2-weeks post-operative images-d to f) shows complete replacement of lesion with normal saline- (d) axial pre-contrast. (e) axial post-contrast and (f) sagittal pre-contrast.

Discussion

Intracranial abscess is a focal empyesis surrounded by a vascular capsule [1,2]. Intracranial abscess can be classified [1] based on location (such as frontal, parietal, temporal, occipital, cerebellar abscess, with frontal being the most common followed by cerebellar abscesses [2]), presence or absence of membrane or medullary covering (such as epidural -as seen in our patient-, subdural or intraparenchymal), mechanism (such as contiguous spread from otolaryngologic infection, paranasal sinus infection, mastoid sinus infection, or hematogenous spread from lung abscess, bacterial endocarditis, sequelae of cyanotic congenital heart disease, or iatrogenic or traumatic -as seen in our patient), volume (small <20 mls, medium 21 to 40 mls, large 41 to 60 mls and giant >60 mls), clinical phase (compensation, sub decompensation, moderate decompensation, severe decompensation and terminal phase), loculations (single or multiple), flow phase (acute, subacute or chronic) and number (single or multiple).

Before the advent of third generation cephalosporins and brain imaging, the risk factors for brain abscess are otitis media, paranasal sinus infection, trans mastoid, trauma and distant infection from hematogenous spread especially in children. However, with the advent of third generation cephalosporins and availability of radio imaging tools, there has been reversal of the risk factors [2–4]. The surgical risk factors for intracranial abscess [1] include poor surgical wound debridement following open head injury, late diagnosis of post-traumatic or iatrogenic cerebrospinal fistula, incomplete evacuation of intracranial haematoma or intracranial haematoma managed nonoperatively [5] and foreign body left in the wound (such as sponges or implants). However, in our patient the likely cause was contiguous spread from the overlying obvious scalp infection following traditional concoction application. It can also occur following acute bacterial meningitis [4].

Following the inoculation of the organism, there is initiation of vasculitis associated with arterial thrombosis leading to area of central necrosis [1], surrounded sequentially by zone of inflammatory cells, macrophages and fibroblasts, area of dense collagen capsule, an area of neovascularization in continuity with the outer area of cerebritis and reactive astrocytes, gliosis and area of oedema external to the capsule [2]. Classically, brain abscess evolves through the following stages viz: early cerebritis (1-3 days), late cerebritis (4-9 days), early encapsulation (9-14 days) and late encapsulation (>14 days) stage [2]. Due to the thinness of the capsules in the early stages, especially in the presence of immunosuppression, the abscess could easily spread to distant sites, making it present with multiple intracranial abscesses especially in the setting of brain abscess or subdural empyema.

Intracranial epidural abscess however, is an extramedullary (extra axial) focal empyesis occurring in the space between the periosteum and the periosteal dura. Furthermore, because of the firm attachment of the periosteal dura to the sutural line, the spread of an epidural abscess, similar to extradural haematoma is limited as seen in this patient.

The limiting nature of the epidural abscess, in the absence of subdural or deeper involvement, makes the presentation insidious in onset. However, our patient presented with acute onset of altered mentation, aphasia and right hemiplegia. Other common presentations which were absent in our patient are periorbital cellulitis and frontal oedema [3,6,7]. Most intracranial abscess occur in the second [2] to third decade [8], as seen occurring in the second decade in this patient. The absence of fever and normal white cell count which further confounded the diagnosis of an abscess, could be explained by the thick wall of the abscess seen on the CT scan.

Cranial CT is a cost-effective tool for diagnosing intracranial epidural abscess [6,7]. It usually will show a biconvex extra-axial mass either at the convexity, interhemispheric or skull base, with a thickened underlying dura which is visible following contrast infusion. It may reveal the focus of the infection if it is intracranial and either brain oedema and herniation as seen in our patient. Furthermore, in the case of open traumatic head injury as the risk factor, it can also reveal the overlying skull fracture and intracranial aerocele [9]. However, in our patient although there was obvious intralesional aerocele, there was no obvious calvarial or skull base fracture or intracranial focus of infection on cranial CT scan. Brain magnetic resonance image (MRI) also has a role in delineating the brain lesion interface and revealing thickened dura following gadolinium infusion [6,7]. Diffusion weighted imaging restriction could confirm the diagnosis [7].

Although it has been established that intracranial extradural abscess is insidious in onset, gas containing intracranial abscess can cause rapid clinical deterioration and even death [9]. This explains the rapidity in onset of symptoms (occurring within 3 days) in this patient. Gas containing abscess following trauma is usually as a result of skull base fracture resulting in a potential fistulous tract communicating with the intracranial space [10]. Therefore, the location of the abscess is often in the frontal skull base [9].

However, in our patient it was located in the frontal convexity with no overlying calvarial or skull base fracture. Furthermore, gas containing abscesses are also associated with various gas producing organisms in the brain [10]. The reported causative microorganisms of gas-containing intracranial abscess include Clostridium perfringens, Klebsiella pneumoniae, Pepto streptococcus species, and Fusobacterium nucleatum [9]. Whilst Klebsiella pneumoniae is seen commonly in diabetic and immunosuppressed elderly patients following hematogenous spread, Clostridium perfringens are seen following traumatic inoculation of the pathogen in the young [9]. In order for Clostridium perfringens to multiply, they require exacting conditions of decreased oxidation reduction potential, thereby making them less frequently isolated from intracranial abscess, since the intracranial space has good arterial oxygenation [10]. This might explain the culture of negative abscess in this patient. The only recorded case of contiguous, non-fistulous and non-haematogenous gas containing abscess to the best of our knowledge was due to frontal sinusitis following sinus surgery [9]. This patient however, had no paranasal sinusitis, otitis media or mastoid sinusitis. Furthermore, posttraumatic fungal inoculation has also been recorded in a 21-month old child [11].

Different colours of the intracranial abscess such as white, yellow, green and red have been found to

be associated with causative organisms [8,10]. Grey coloured materials have been found in necrosis of malignant brain tumours and radio necrosis [8]. Reddish materials were associated with Clostridium perfringens in a child with open traumatic head injury, presenting with fever and operated within 48 hours of trauma [10]. However, our patient with gas containing yellowish abscess, had no organism seen nor cultured. Similarly, the study of 400 patients with brain abscesses by Yang et al revealed 171 patients without fever, 121 with normal white cell count and 116 with negative culture abscesses [12].

Because of the rapidity in deterioration and even death in patients with intracranial epidural gas containing abscess, surgery (consisting of either craniotomy and excision of abscess wall or burr hole and drainage of the abscess), microbiological and/ or histological analysis of aspirate and/ or abscess wall biopsy and extended empirical antibiotics of 4-6 weeks (consisting of at least 2 weeks of parenteral antibiotics) which could be reviewed following release of aspirate or biopsy results is usually the strategy of treatment, in all patients presenting with neurologic symptoms [9]. All empirical antibiotics are generally stopped before surgery to boost the yield of microbiology or histology results [2]. Although antibiotics were stopped in our patient, his aspirate culture returned negative and antibiotics were continued empirically since he responded well to the regimen. The type of surgery depends on the associated pathology seen on preoperative imaging [9]. Therefore, craniotomy will be decided when there is an identified fistulous tract needing excision, or frontal sinusitis as the likely aetiology of the abscess needing cranialization of its posterior wall [9]. However, when there are no additional indications necessitating craniotomy, it has been found that the outcome of burr hole drainage is better than that of craniotomy and excision [2]. Our patient however, since he had no identifiable frontal sinusitis and/ or fistulous tract, had mini-craniectomy and evacuation of abscess and did well on empirical antibiotics. Furthermore, in patients with paranasal sinusitis otolaryngologist collaboration should always be sought for a better outcome [8–10,12].

This case is unique in that it is the first case with gas containing abscess diagnosed intra-operatively in a patient with closed head injury and localised infected scalp injury, with no other known clinico-radiological risk factor. Although, a non-gas containing brain abscess has been reported in a diabetic patient with intraparenchymal haematoma following closed head injury and no discernible focus of infection, managed nonoperatively, resulting in abscess and subsequently needing craniotomy and excision of the abscess [5].

Conclusion

In a patient with previously reported lucid interval following trauma, presenting with acute onset of neurologic deterioration and an evidence as little as infected scalp injury and radiologic evidence of extra-axial air-fluid level lesion, a gas containing extra-axial traumatic abscess should always be considered as a differential diagnosis in the management of such patient. To achieve a good outcome, expedited surgical intervention and extended duration of antibiotics should be initiated.

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