CASE REPORT

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A developing brain abscess as acute stroke mimic: presentation of case and review of literature

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Abstract

Whether brain abscess is a cause, or a complication of stroke has been a question that elicited differing opinions. We report a case of a child who initially presented with hemiparesis and seizures. Initial brain tomography did not show hemorrhage or infarction. He was managed as a case of childhood stroke. The neurologic manifestations gradually improved until the 17th day of hospitalization when regression of the motor strength and anisocoria occurred. A repeat brain tomography showed multiple masses and vasogenic edema consistent with brain abscess.

Keywords: Brain abscess, Acute stroke, Brain tomography.

INTRODUCTION

It has been a long-standing debate whether a brain abscess is a complication of stroke or is a cause of stroke-like manifestations. Majority of case reports on brain abscess either as a complication of stroke, or as a stroke mimic are in adults. Here, we present a case of a brain abscess in a child who presented with stroke.

CASE REPORT

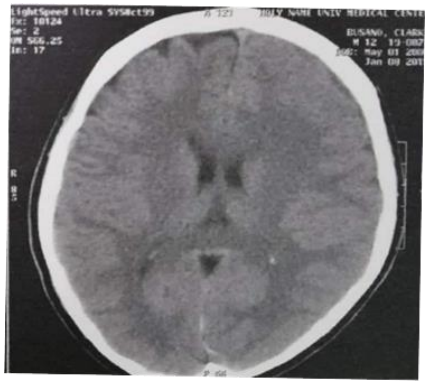
We report the case of a 12-year old boy who was admitted to our institution for sudden onset of right-sided weakness. Four days prior to admission he started to have low to moderate grade intermittent fever associated with occasional nonparoxysmal unproductive cough. He was prescribed with Clarithromycin, antihistamine, and ß2-agonist medications, with resolution of symptoms. The fever recurred 1 day prior to admission associated with 2 episodes of nonprojectile vomiting of previously ingested food, and a throbbing frontal mild to moderate headache. About 12 hours prior to admission, the patient could no longer move his right upper and lower extremities, prompting transport to the hospital where he had an episode of generalized tonic clonic seizures lasting for 10 seconds.

The past medical history revealed that the patient had a bout of fever and cough about 3 weeks prior to admission, for which he was prescribed Amoxicillin-clavulanic and Salbutamol, with subsequent improvement.

In the emergency room, the patient was examined conscious, coherent, oriented to 3 spheres, verbal but unable to move his right upper and lower extremities, and febrile at 38.6°C. The systemic physical examination findings were generally normal. However, the neurologic examination revealed a flattened right nasolabial fold, inability to lift his right shoulder, and deviation of the tongue to the right on protrusion. There was 0/5 motor strength of the right upper and lower extremities. Babinski sign present at the right. Hyporeflexia was noted on the right upper and lower extremities.

The patient was initially admitted as childhood stroke, and was worked up along this impression. There was neutrophilia seen in the complete blood count. This, together with the fever prompted administration of antibiotics. Hyponatremia at 127.5 mEq/L was seen. The prothrombin time was prolonged with an activity of 57.4% while the activated partial thromboplastin time was normal. The lipid profile showed low high density lipoproteins (HDL). The urinalysis was normal. The chest radiograph and the 2-dimensional echocardiography were normal.

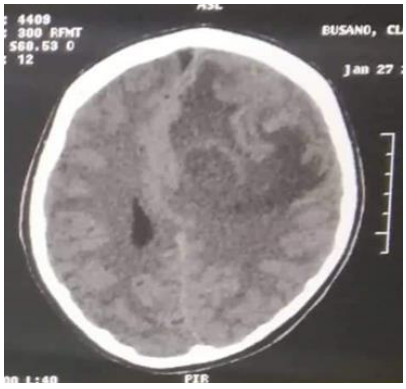
The initial non-contrast cranial CT scan only showed focal brain atropy at the right frontal lobe area without any sign of hemorrhage or infarction.



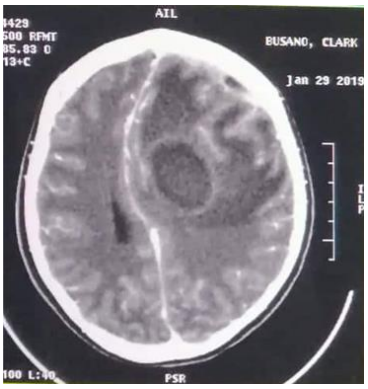
The fever resolved on the third day of hospitalization while on the antibiotics, but the motor deficits did not improve. Physical therapy was started.

By the sixth day of hospitalization, the patient's motor strength on the right upper and lower extremity was seen to be 2/5, which further improved to 3/5 the next day. Rehabilitation therapy and the antibiotics were continued.

The patient's condition was progressively improving, when on the 17th hospital day there was note of sudden regression of the motor strength of the affected side to 0/5. Anisocoria was also noted. A re-stroke was considered so a repeat noncontrast brain CT scan was performed; this time, multiple defined masses on the left frontal lobe were seen with vasogenic edema and mass effect, consistent with a brain abscess.



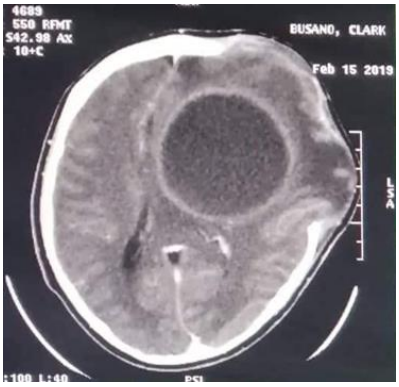
A neurosurgery referral was made, and a contrast-enhanced brain CT scan was proceeded, revealing multiple enhancing complex masses, with subfalcine herniation to the right.



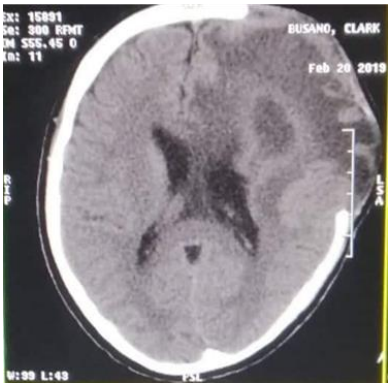
Medical and surgical decompression was done. Antibiotics were shifted to Metronidazole, Amikacin, and Meropenem per brain abscess guidelines. Post-operatively, the patient's pupils became isocoric, the motor strength in the right lower extremity was 1/5 while the motor strength in the right upper extremity was 0/5.

With the medications and rehabilitation, his condition gradually improved. By the 8th postop day, he was already able to gain full motor strength in the right leg while the right arm strength improved to 2/5.

A repeat contrast-enhanced cranial CT scan done on the 37th hospital day showed interval enlargement of the rim-enhancing fluid density in the left frontal lobe, surrounding vasogenic edema and expansile effect, right subfalcine herniation and left frontal craniotomy defect.



Aspiration of the abscess was performed. Culture of the abscess fluid did not yield any growth. Antibiotics were continued.



A repeat cranial CT scan on the 42nd hospital day showed interval decrease in size of rim-enhancing fluid density in the left frontal lobe and interval decrease in rightward subfalcine herniation, still with vasogenic edema. Medications were continued and further clinical improvement was noted.

Patient was discharged on the 45th hospital day already isocoric, with full strength in both lower extremities and left upper extremity, and residual weakness in the right upper extremity.

DISCUSSION

Childhood stroke is defined similarly as adult stroke: an acute onset neurological sign or symptom attributable to a focal brain infarction or hemorrhage [1, 2]. Just like in adults, childhood stroke can be divided into hemorrhagic and ischemic stroke. Hemorrhagic stroke may be intraparenchymal or spontaneous nontraumatic subarachnoid hemorrhage. Focal brain damage results from hemorrhage through localized mass effect and

ischemia of adjacent tissues. Cerebral infarction or ischemic stroke may result from arterial ischemic stroke (AIS), venous infarction, or cerebral sinovenous thrombosis (CSVT). Focal brain damage from ischemic stroke results when there is a loss of blood flow or oxygenation to an area of brain tissue [3].

Stroke in children is reported to be common. It affects 1 in 1,600 – 4,000 neonates [3, 4], and 2.3 to 13 per 100,000 older children annually [5, 6, 7]. Hemorrhagic stroke accounts for 50% of pediatric stroke, with an annual incidence of approximately 1 – 1.7 in 100,000 children [6, 8]. The remaining 50% of pediatric stroke is accounted for by ischemic stroke, with a slightly higher annual incidence of 1 – 2 per 100,000 children in developed countries [8, 9]. In the Philippines, the registry of childhood diseases of the Philippine Pediatric Society, Inc., shows that there is a total of 701 reported cases of stroke in children from January 1, 2006 to December 31, 2019 [10]. In a country with roughly 42,311,222 children [11], the annual incidence rate of stroke is approximately 0.12 per 100,000 Filipino children. This number is much lower than that was reported for developed Western countries. However, this may not be a true reflection of the incidence of stroke among Filipino children as the PPS registry collects data only from their accredited training hospitals.

The most common symptoms of acute stroke in children are similar to those in adults. These include hemiparesis and hemifacial weakness (67 – 90%), speech or language disturbance (20 – 50%), vision disturbance (10 – 15%), and ataxia (8 – 10%). Nonlocalizing symptoms like headache and altered mental status may also be present. Seizures manifest in 15 to 25% of children with stroke [12-15]. Seizure serves as a sentinel sign of acute stroke in children, occurring in 31% to 46% of younger children with stroke [3]. Our patient presented with headache, vomiting, right-sided hemiparesis, and seizures. Clearly, our patient's clinical manifestations are compatible with acute stroke.

The nature of the stroke-like manifestations of the patient on presentation was a dilemma for a time because aside from the absence of an infarcted area in the brain on the initial cranial CT scan, the search for the more common etiologies like sickle cell disease, cardiac disorders, or prothrombotic conditions were negative. A suspicion for yet undetermined infection was considered based on the presence of fever and neutrophilia in the complete blood count.

Literatures show that only approximately 50% of patients with ischemic stroke will have known risk factors predisposing to stroke at the time of diagnosis. However, after thorough evaluation, 40% of children with ischemic stroke are found to have 1 risk factor, 50% have 2 or more factors, and 10% have no identified risk factor for stroke. These risk factors include arteriopathies, cardiac disorders, infection, acute head and neck disorders, acute systemic conditions, chronic systemic conditions, prothrombic states, chronic head and neck disorders, atherosclerosis-related risk factors, and others [16].

The pathophysiology of the stroke in our patient was made clearer when a repeat cranial tomography showed multiple defined masses on the left frontal lobe with vasogenic edema and mass effect, which showed enhancement with contrast. This finding was compatible with brain abscess.

Brain abscess is a focal infection in the brain parenchyma consisting of encapsulated pus and pyogenic bacteria. Less commonly, fungi, mycobacteria or protozoa cause brain abscess [17]. Brain abscesses are reportedly rare in children, but they can cause significant morbidity and mortality [18]. They occur in 0.4 per 100,000 children annually but may range from 0.3 to 1.3 per 100,000 children [17]. The registry of childhood diseases of the Philippine Pediatric Society (PPS), Inc. shows that there are 1,206 cases of intracranial abscess over 14 years [10]. This translates to an annual incidence of 0.20 per 100,000 Filipino children. This number is lower than the reported incidence and contrasts with the report by Gaskill and Marlin in 2008 which said that there is a higher incidence of brain abscess in underdeveloped countries [20]. They more common in males in the first two decades of life [21]. Children 4 to 7 years old have the highest incidence of brain abscess [19, 22]. The locations of abscesses in the brain, in decreasing frequency, are frontotemporal, frontoparietal, parietal, cerebellar, and occipital areas [23].

Eighty percent of children with brain abscess have predisposing factors while 20% of cases are cryptogenic [17]. Among children with predisposing factors, 30 – 50% result from contiguous focus of infection like otitis media, mastoiditis, sinusitis, orbital cellulitis [17], or dental infections [24]. Approximately 10% of cases occur in cases where the body's natural barriers are disrupted like in cases of neurosurgical procedures or head trauma [17, 25, 26]. Hematogenous spread associated with bacteremia and immunosuppressive conditions accounts for 40% of cases of brain abscess in children [17]. Direct spread of the organism from a contiguous focus of infection usually causes solitary brain abscess [24] while bacteremic spread typically causes multiple brain abscesses [27]. Based on the history and the physical exam which did not show evidence of probable contiguous foci of infection, neurosurgical procedure or head trauma, and the multiple abscess lesions seen in the cranial tomogram, the brain abscesses in our patient must have resulted from a bacteremic route. However, despite work-up, there was no underlying condition that was identified. This absence of an underlying condition or a primary site of infection has been reported in 20 – 40% of patients with brain abscess.

The signs and symptoms of brain abscess are initially nonspecific [28, 31]. Headache is the most common symptom, occurring in 69% of cases. Fever occurs in 45 – 53% of patients, seizures in 25%, and focal neurologic deficits in up to 50% of children with brain abscess [32, 33]. Individually, each of these signs and symptoms is not a reliable basis for the diagnosis of brain abscess. Even the classic triad of fever, headache, and focal neurologic deficits that suggests brain abscess is present in only 20% of cases [34]. Thus, a high index of suspicion is recommended. Our patient presented with fever, headache, and hemiparesis. This should have alerted us to the possibility of brain abscess on admission; however, in the absence of abscess lesions on the initial cranial tomogram, the diagnosis of brain abscess was not considered on admission.

Hemiparesis in cases of brain abscess is reported to be more common in children than in adults. In a review of literatures on brain abscesses in children, the incidence of hemiparesis ranged from 19.2% to 44.06% [35, 36, 37]. This high incidence was demonstrated to result from the involvement of the eloquent areas of the brain with seeding of metastatic abscesses [37].

The question that we asked ourselves in the management of this patient was that is this a case of the brain abscess being a stroke mimic or is the brain abscess a complication of stroke. Unfortunately, there is a dearth of literatures related to this topic in pediatrics. However, there have been reports of brain abscess as complication of stroke in adults. To date, there are 20 case reports of brain abscess after stroke that were published [38-57]. All of these are reported in adults. The abscess is reported to develop 12 days to 14 months after the stroke incident [58, 59]. Our patient was diagnosed with brain abscess 17 days after the stroke.

The pathogenesis of brain abscess after a stroke involves the disruption and loss of integrity of the blood brain barrier around the ischemic area [43], making it vulnerable to microbial seeding when bacteremia occurs during a systemic infection [38].

However, brain abscess as a complication of the ischemic stroke could not be explained by the afebrile state of our patient at the time of or immediately before the diagnosis of brain abscess was made. This made us consider an alternative scenario where the developing brain abscess acted as a stroke mimic.

There have also been reports of brain abscesses as stroke mimics [60-70]. Unfortunately again all these reports are in adults. The exact mechanism how a brain abscess presents with stroke-like symptoms is still unclear. One hypothesis is the development of paroxysmal septic emboli that causes embolic infarction which becomes the nidus for the abscess formation [61]. However, the septic work-up in our patient did not reveal any focus of infection that could have thrown a septic embolus to the brain. The authors hypothesize that the stroke-like manifestations in our patient, not accompanied by an apparent infarct in the cranial tomogram, may be due to the cerebritis stage of abscess formation and its accompanying inflammatory vasculitis or in-situ thrombus formation, and that the brain abscess in our patient could have been cryptogenic in nature. It has been reported that the cranial tomography of the brain may be negative or may show only subtle nonspecific findings during the early stages of evolution of a brain abscess [71]. It could have been better if a magnetic resonance imaging of the brain was done as it is the preferred imaging method in the initial examination. Sadly, our institution lacks this technology for now.

The most important lesson that this patient taught us, regardless of whether the brain abscess is a complication of a stroke or is a stroke mimic, is that a high index of suspicion for the possibility of a brain abscess in a patient with stroke-like manifestations is quite necessary for early diagnosis and treatment to prevent significant morbidity and mortality.

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