Brain abscess of unknown etiology in a 2-year-old child: a case report

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Abstract
We present a case of brain abscess necroptically discovered in a 2-year-old child hospitalized in the Pediatrics Clinic of the “Filantropia” Municipal Hospital, Craiova, Romania. The family, with a poor financial situation, reports previous episodes that may be interpreted as convulsive crises. Clinically speaking, he presents a height-weight hypertrophy, vitamin D loss rickets, and psychomotor retardation. At the objective examination, we found a weight of 10 500 g (!), second and third degree mesocardiac systolic beat and cardiomegaly in the thorax-cardiac-pulmonary X-ray examination. Despite the intensive treatment, death occurs few hours after hospitalization. During the autopsy, there is observed a partial dehiscence of the cranial arch sutures, with a 6/5 cm oval cavity in the parietal lobe, containing approximately 200 mL of yellow-green serous liquid, with uneven walls, but with no hemorrhagic or pus infiltrates. The heart is enlarged (in comparison to the general somatic development) of 9/7/4 cm, without any cardiac malformations. The microscopic examination showed degenerative neuronal and ischemic lesions on the left-brain hemisphere. Comparing to the data from specialty literature, we consider it as a yellow brain softening (according to Rokitansky’s classification), most probably of an embolic cause.

Keywords: child brain abscess, leukoencephalitis, leukomalacia.

Introduction
Brain abscess is relatively uncommon but is serious in children [1], similarly to stroke [2, 3]. Brain abscesses occur infrequently but continue to be problematic for the pediatric neurosurgical community. About 25% of brain abscesses occur in children, mostly in the age group of 4–7 years [4, 5]. Brain abscesses in children were traditionally associated with congenital heart defects and with infections of the face, head or brain [6]. Intracranial parenchymal abscesses are a consequence of invasion by infectious microorganisms secondary to hematogenous dissemination from a remote infection, the spread of a contiguous infection, foreign material introduced through a penetrating traumatic injury, or as a postoperative complication. The most common risk factors that predispose a child to the formation of a brain abscess include congenital heart disease, sinus and otogenic infections, poor dental hygiene, infancy, immunosuppression, neurosurgical procedures, penetrating skull injury and comminuted fracture of the skull, congenital lesions of the head and neck, and as a rare complication of meningitis [7]. Of patients diagnosed with a brain abscess, 30–34% have underlying heart defects [8–10].

Our case presents a child with congenital heart disease and brain abscess.

Aim
This article aims to present severe pathological changes that accompany brain abscess in a child with poor nutritional status and comorbidities.
walls, but not hemorrhagic or purulent infiltrates have been observed (Figure 1c). Right hemisphere show only edema and stasis. Macroscopic examination of the other regions highlights only a 9/7/4 cm enlarged volume heart (relative to general somatic development), but no malformations (Figure 1d).

Histological examination performed after Hematoxylin–Eosin (HE) classical technique, reveal three areas in terms of histopathological lesions: (1) a marginal zone with interstitial edema and compression phenomena in the nervous tissue, (2) a transition zone with predominantly rich lymphocytic infiltrate, and (3) a deep zone of leukomalacia, with numerous neoformation vessels.

Adjacent nervous tissue injury is the white matter and submit nerve fibers, glial cells (oligodendrocytes particular) and blood vessels, whose appearance suggests phenomena flattened unidirectional compression exerted by cerebral abscess, especially that the limit of the nervous tissue and lymphocytic infiltrate is well defined (Figure 2a).

Intermediate area of transition between nerve tissue “intact” and deep area of leukomalacia is represented by a diffuse lymphoplasmacytic rich infiltrate but discreet subdivision trend, suggesting a pseudofollicular aspect (Figure 2, b and c).

Deep area of leukomalacia, presents on an eosinophilic background an obvious cell depletion in contrast to the extremely rich vasculature, pseudoangiomatous layout (Figure 2d). Obviously, there are neoformation vessels, because normally this region in depth hemisphere does not have a rich blood supply.

Discussion

Brain abscess may be asymptomatic for long times. Clinical findings may be mild and the classic triad characterized by headache, fever, and focal neurological deficit is seen in 9% to 28% of children [4, 11]. Therefore, lack of neurological symptoms in present clinical case is not at all unusual.

Figure 1 – Macroscopic aspects: swelling of the left cerebral hemisphere (a), with inner abscess-like aspect (b), and diffuse cerebral tissue as wall (c); relative enlarged heart (d).

Figure 2 – Histopathological aspects: marginal zone (a – HE staining, ×100), transition rich lymphoplasmacytic zone (b – HE staining, ×40).
Infectious etiology cannot be ruled out, although the incidence is relatively low, as shown above, and the lack of bacteriological confirmation occurs in 10% [12] up to 56% of the cases [6]. Consistent with this case are contributory factors, such as congenital heart disease and association with immunosuppression [4, 12–15]. Tuberculous etiology is unlikely, both in incidence [16], and lack of specific pathological lesions in the present case. Surprisingly or not, the brain is resistant to infection, due to the protective microglia [17, 18]. Lesions of cerebral abscess following stratigraphic characteristics: (1) a well-formed necrotic center; (2) a peripheral inflammatory zone; (3) a dense collagenous capsule; (4) a neovascular zone; and (5) a reactive zone with gliosis, and cerebral edema, external to the capsule [19, 20]. This description is in contrast to what we found in our case, where the lack of specific pathological lesions in the present case. Given the above, we consider as the most likely mechanism embolic stroke with cardiac source. The incidence of stroke in children is reduced, for example, in some US states is only 10.7/100 000; only in the presence of congenital heart disease incidence significantly increases this to 132/100 000 [21]. A similar study conducted in Hong Kong showed an incidence of pediatric stroke of only 2.1/100 000, but with a relatively balanced distribution of the first three etiologies: congenital heart diseases (15, 30%), vascular diseases (13, 26%), and hematological diseases (14, 28%) [2].

Now more than a century and a half, Karl Rokitansky and William Edward Swaine (1855) described encephalomalacia (softening of the brain) in three different forms, both macroscopic and etiologic: (1) white, in edema and hydrocephalus, (2) red in inflammation, and (3) yellow with questionable etiology, including nutritional deficiency. In ischemic cerebral infarction, the two pathologists observing that, as the core of the infarction disintegrates, endothelial cells from the periphery of the lesion proliferate and grow the capillaries within necrotic tissue. Astrocytes from neural tissue integrity neighboring forms a glial scar that limits and stabilizes the lesion. With the proliferation of capillary, the glial scar repair hematencephalic barrier [22].

Conclusions

Although the incidence of pediatric stroke is reduced in the case of pre-existing cardiovascular or blood disease, it can be a formidable complication. Structural peculiarities of the forebrain tissue determine the evolution of cerebral ischemia in a special manner, reaching a morphologically aspect, similar brain abscess. Therefore, it requires tracking children with cardiovascular congenital diseases and immunodeficiencies to assess them both neurological and clinical and, where appropriate, imagistic.

Conflict of interests

The authors declare that they have no conflict of interests.

References


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