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RESEARCH ARTICLE

ACUTE LEUKEMIA REVEALED BY CEREBRAL HEMORRHAGE

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Abstract

Cerebral Hemorrhage is the most common neurological complication in acute leukemia and often leads to death. It may result from underlying leukemic effects on the immune or hematopoietic systems, or from antileukemic therapy. Several studies have been established to understand the pathophysiological mechanisms causing neurological manifestation in acute leukemia. Our case demonstrates that cerebral hemorrhage can occur as the first sign of this disease.

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Introduction:-

Acute leukemia is a malignant proliferation of hematopoietic tissue. It can frequently be complicated by neurological damage, but it is rare that it represents the initial manifestation of this disease.

Use of modern imaging techniques allows early diagnosis before the installation of irreversible disorders, thereby increasing the overall survival [1].

We report the case of a 2.5 years old child who presented a fatal cerebral hemorrhage revealing acute lymphoblastic leukemia (LAL).

Case Report:

We report the case of a child of 2.5 years old, with a history of cerebral palsy, admitted for apyretic impaired consciousness with a Glasgow coma score of 10.

The initial biological assessment showed a major hyperleukocytosis with white blood cells at 800 000 / mm³ and thrombocytopenia at 18000 / mm³ and hemoglobin at 10g / dl. The blood smear found 85% of blasts.

In front of this clinical aspect, a brain CT without contrast was urgently requested to look for a stroke due to leukostasis (Figure 1). It revealed multiple intra parenchymatous hyperdense lesions, well circumscribed, bilateral and diffuse, at the supra and infratentorial levels, with perilesionnel edema. The most voluminous one was located on the left parasagittal parietal level measuring 50x37 mm producing a mass effect with a compression of the adjacent lateral ventricle and a deviation of the cerebral midline by 3 mm.

We have concluded to multiple intraparenchymal hemorrhage, with mass effect.

Despite maximum medical care, the patient died in the intensive care unit at night from a subfalcine herniation caused by the exacerbation of his cerebral hemorrhage.

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Discussion:-

Acute leukemias are malignant overgrowths of precursors of blocked blood cells at an early stage of their differentiation.

There are two varieties of acute leukemia according to the cytological type of blastic cells: acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML) [2].

ALL represents about 80% of childhood acute leukemias, with a peak age between 2 and 5 years [3].

Among their most severe complications, the occurrence of hemorrhagic events, dominated by intracerebral hemorrhages, which represent an important cause of morbidity and mortality in patients followed for acute leukemia [4].

Several factors contribute to causing this hemorrhage as alterations in coagulation factors, thrombocytopenia, and the possible associated disseminated intravascular coagulation. Few of them can be treated, and some are the result of treatment directed against the disease process itself [4,1].

The platelet count plays an important role in determining the risk of bleeding, especially when the platelet count drops below 10000/mm³, but is not directly proportional to the degree of thrombocytopenia [5,1].

Furthermore, severe hyperleukocytosis, defined by a level of leukocytes greater than 100.10⁹ / L, leads to hyperviscosity of the blood, leukostasis, hypoxic vasodilation and sometimes the rupture of cerebral vessels. It is responsible for thromboses and haemorrhages due to capillary fragility. Leukostasis, particularly those with a leukocyte count over 300,000/mm³, can then be life threatening by neurological damage with cerebral anoxia and cerebral hemorrhage [1].

On the other hand, hemorrhage can also occur during chemotherapy or bone marrow transplantation. Some chemotherapeutic agents exacerbate the degree of thrombocytopenia and promote a breakdown of coagulation factors which leads to hemorrhagic diathesis [1,6].

Among the chemotherapy molecules used in the treatment of ALL, L-asparaginase is responsible for inhibiting synthesis of coagulation proteins. Its prothrombotic action is well described, but few cases of hemorrhage related to its use have been reported [4,1].

Therefore, nowadays there is no unanimity of opinion concerning the pathogenesis of cerebral hemorrhage in leukemia [5].

There are therefore three radiological profiles of cerebral hemorrhages linked to the different pathophysiological mechanisms: multiple hemorrhages by leukostasis, single and massive hemorrhage by disseminated intravascular coagulation, and hemorrhagic infarction by fungal embolism. They can be intracerebral, subarachnoid, and subdural or intra ventricular and are often fatal. Subarachnoid and intraparenchymal hemorrhages have a worse prognosis than subdural hematomas [2].

These hemorrhagic complications are very well highlighted by the CT scan, which shows spontaneous hyperdense lesions often associated with edema and a mass effect [7].

Leukostasis can also cause microangiopathy which is responsible for infarction with microhemorrhage that can easily be detected by MRI [8].

MRI can also allow detecting small hemorrhages with small collars of edema in the subcortical white matter caused by disseminated intravascular coagulation [1].

It is true that MRI requires longer time compared to CT scan, but it is more sensitive to detect infarction and microhemorrhage caused by microangiopathy of leukostasis [8].

Therefore, CT scan should be the initial imaging modality in patients with leukostasis and MRI should be reserved for patients with neurologic manifestation after leukapheresis or the initiation of intensive chemotherapy [8].

The overall 5-year survival rate in children with LAA is about 90%[4].

Management of patients with acute leukemia must be carried out in a specialized pediatric environment after multidisciplinary concertation meeting. Recently, the introduction of new therapeutic methods, such as aggressive polychemotherapy, intrathecal cytostatic prophylaxis and cranial irradiation have improved the prognosis of acute leukemia, without denying that these treatments can sometimes themselves be responsible for neurological complications. Surgery is the last approach to care intracranial hemorrhage: craniectomy, evacuation of hematoma, shunt ventricular are gestures of last resort[4,1]. The results of a randomized trial indicate that surgical treatment fast does not offer an advantage compared to medical treatment for the management of cerebral hemorrhages. Bone marrow transplantation has also revolutionized the treatment of these patients [1].

Conclusion:-

Cerebral hemorrhage is a frequent and often fatal complication of acute leukemia, it occurs more frequently than is suspected clinically. It can sometimes be the initial manifestation of this disease. Several factors can be at the origin of this cerebral hemorrhage but few of them are treatable. However, some of them are due to the complications of the treatment directed against the disease itself.

Radiological imaging contributes considerably to the evaluation of hemorrhagic complications of acute leukemias, whether related to the disease or as a result of aggressive therapies.

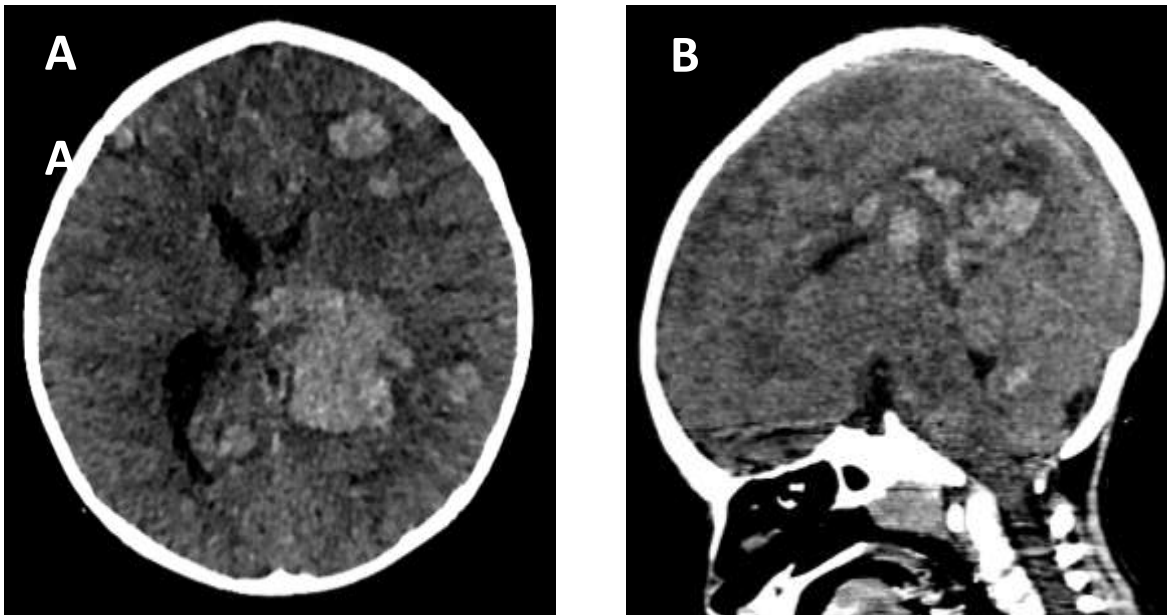


Figure 1:- Axial (A) and sagittal (B) CT without contrast shows multiple intraparenchymal hematomas with mass effect and midline shift with perilesional edema.

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