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Epstein Bar virus encephalitis in a patient with T/NK non Hodgkin lymphoma

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Abstract:
We report the case of patient with nasal NK/T non-Hodgkin lymphoma, a rare and aggressive tumor. We described the evolution of the disease in this patient through imaging examinations such as 18F-FDG PET and CT Scan, as well as the treatments administrated. The results of the examinations showed an initial response to the treatment, but a relapse of the disease was noted with metastasis at the mediastinal and abdominal level. The patient underwent a salvage treatment with CHOEP, but developed complications and unfortunately died. Our scientific research highlights the importance of early analysis and regular follow-up of this rare and aggressive disease in order to improve the patient's chances of survival.

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I. Observation:

28-year-old female patient admitted for management of T/NK NHL of nasal location.
On ENT examination: ulcerated lesion of the soft palate with biopsy in favor of an angiocentric T lymphoma type NK nasal (CD3+ CD56+ GRANZYME +).
She had no peripheral tumor syndrome.

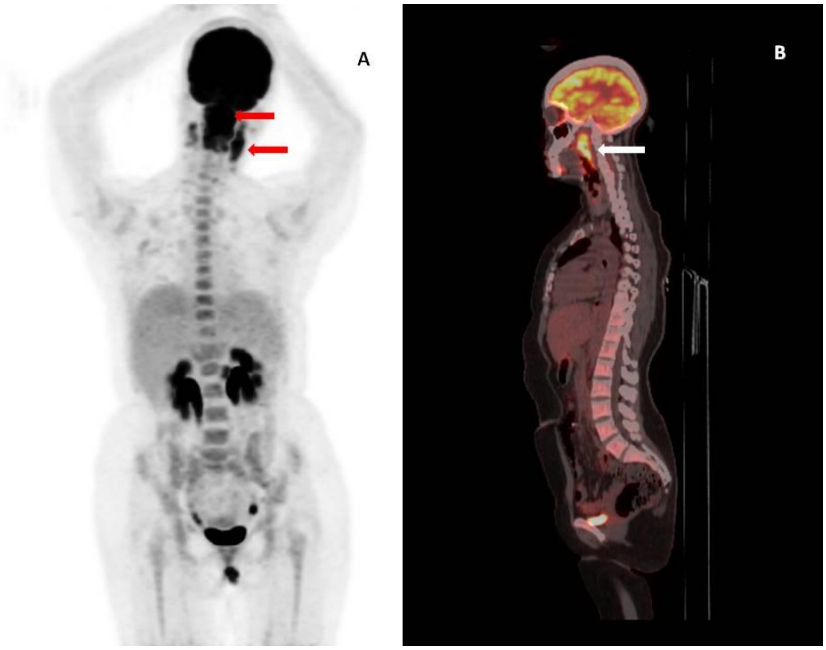

Figure 1: Initial 18-FDG PET scan (MIP on the left and sagittal slice on the right) showing foci of hypermetabolism in the tumor process of the cavum extending to the oropharynx associated with bilateral cervical hypermetabolism suggesting lymph node involvement

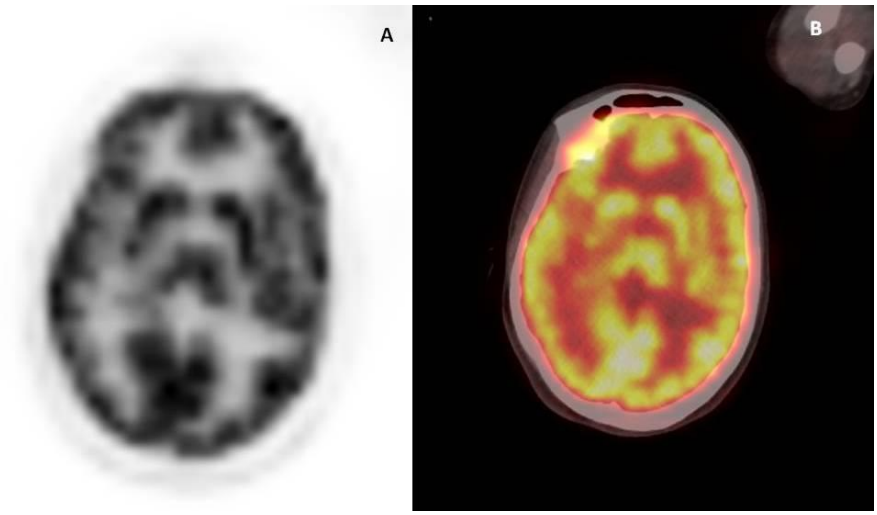

Figure 2: Initial 18-FDG PET scan (PET alone on the left and PET/CT fusion image centered on the brain in axial slice on the right) showing normal uptake of the glucose analogue on the cerebral cortex and basal ganglia.

The initial 18F-FDG PET scan showed intense hypermetabolism from the nasopharynx to the oropharynx (Suv max: 13.5), bilateral cervical adenopathy (Suv max between 3.1 and 8.1), mesenteric lymph node hypermetabolism (only 1 focus, Suv max 5.5)

The evolutionary and pre-therapeutic work-up was normal, therefore the disease was classified as a Stage II NK Lymphoma of the cavum. The patient received 2 courses of Aspa-Dexa-Metho (L-Asparginase-Dexamethasone- Methotrexate)

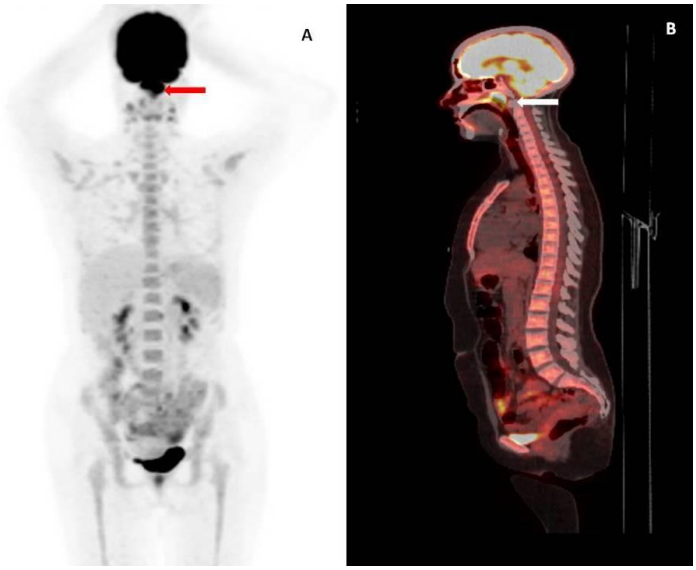

Figure 3: 18-FDG PET scan of treatment evaluation after two treatments (MIP on the left and sagittal section on the right) showing the persistence with a clear decrease of the previously described hypermetabolic foci in the cavum tumor process and cervical lymph node involvement.

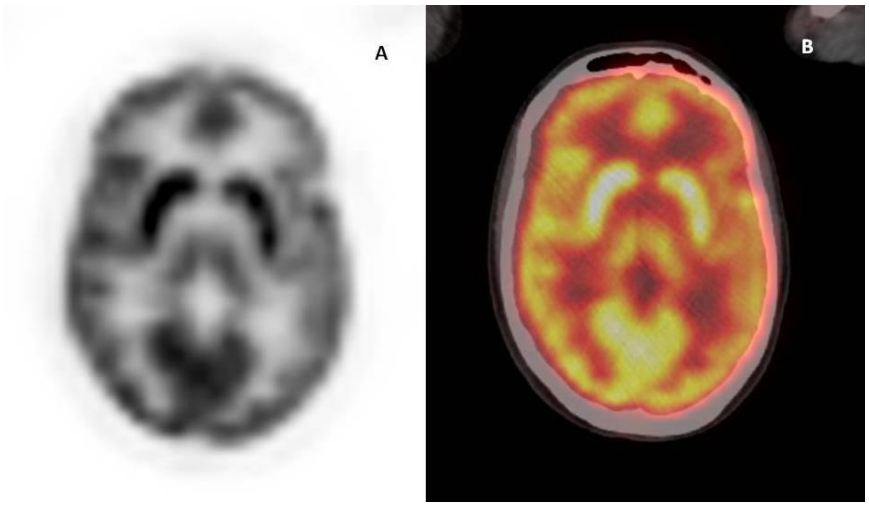

Figure 4: 18-FDG PET scan for treatment evaluation (PET alone on the left and PET/CT fusion image centered on the brain in axial section on the right). Cortical uptake of the glucose analogue begins to decline.

Re-evaluation 18F-FDG PET performed in September 2015 shows a clear decrease in previously described hypermetabolism in the cavum (Suv max 12.5) and cervical and abdominal adenopathy. Further analysis of the encephalic floor already showed a decrease in cortical uptake compared to basal ganglia
The patient subsequently received 50 Gy of cavum irradiation followed by 3 other Aspa-Metho-Dexa treatments. The last one was complicated by hepatic toxicity (cholestasis, cytolysis, ascites) without any disorder of consciousness.

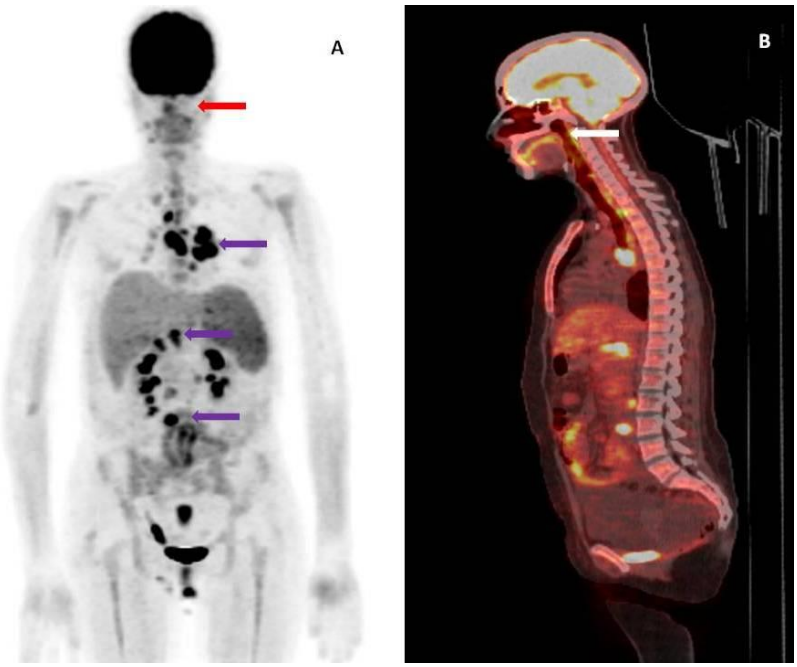

Figure 5: 18-FDG PET scan of treatment evaluation after five courses and external radiotherapy (MIP on the left and sagittal section on the right) showing the disappearance of the previously described hypermetabolic focus at the level of the cavum tumor process, but the appearance of new supra- and subdiaphragmatic lymph node involvement

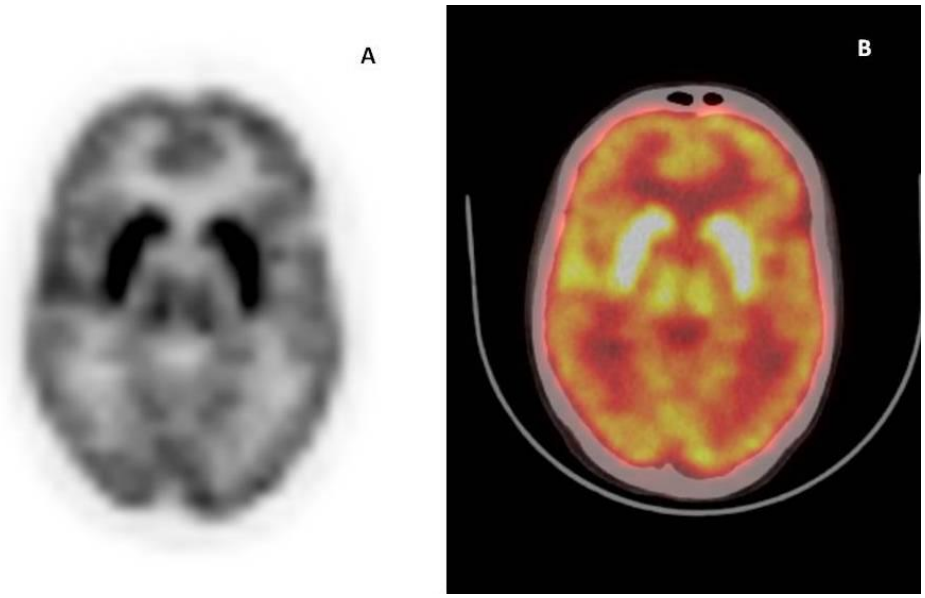

Figure 6: Third 18-FDG PET scan for treatment evaluation (PET alone on the left and brain-centered PET/CT fusion image in axial slice on the right). Cortical uptake of the glucose analogue continues to decrease.

The PET scan at the end of the treatment showed tumor progression with the appearance of new foci at the mediastinal and abdominal levels with visualization of abundant ascites without foci of hypermetabolism. At the encephalic level, the cortical uptake was obviously collapsed in comparison with that of the basal ganglia

The therapeutic decision was to treat the patient with CHOEP (Cyclophosphamide, doxorubicin, etoposide, vincristine, prednisone). She subsequently presented with consciousness disorders, with a positive Epstein bar PCR at the lumbar puncture, she unfortunately died in the following days.

II. Discussion :

EBV infects B cells latently in 90% of the population [1]. It is not neurotropic but can cause encephalitis by two mechanisms, classical cytolytic infection [2] or an excessive inflammatory host response [3] .

Clinical presentations are numerous, asymptomatic or sometimes acute. The classic erythemato pulmonary angina of infectious mononucleosis may be associated with central nervous system involvement revealed by meningitis, encephalitis, encephalomyelitis, myeloradiculitis, cerebellite or peripheral nervous system involvement. EBV is responsible for 2.3-6% of encephalitis in children and 1-1.5% in adults [4], [5], [6]. Chronic forms are associated with neoplasia such as nasopharyngeal carcinoma, Hodgkin's disease, non-Hodgkin's lymphoma, Burkitt's lymphoma or smooth muscle tumors in immunocompromised patients [7].

Striatal hypermetabolism has been reported in three patients with limbic encephalitis [8]. Limbic encephalitis can be paraneoplastic, following a viral infection, or idiopathic. In another case with anti-CRMP5 antibody-associated paraneoplastic chorea, PET showed hypometabolism of the bilateral caudate nucleus [9].

PET studies in LE usually report bilateral medial temporal hypermetabolism, as well as hypermetabolism of the frontotemporal lobes, brainstem and cerebellum [10-11].

Striatal hypermetabolism, however, has been associated with other autoimmune diseases, such as Sydenham's chorea, systemic lupus erythematosus, and primary antiphospholipid antibody syndrome [12-13].

Fujimoto et al [14] report ten cases of EBV neurological involvement: four encephalitis, one cerebellar ataxia, two acute disseminated encephalomyelitis, one myelitis and two meningitis. Finally, primary EBV infection has been reported as a cause of acute encephalitis in young children [15]. In any case, the treatment of neurological forms is not codified. Acyclovir is not very effective in vitro and is not a treatment for infectious mononucleosis. The efficacy of gancyclovir [16] should be considered with caution, for a condition that is likely to heal spontaneously [17].

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