Available online at www.elixirpublishers.com (Elixir International Journal)

Physiology and Anatomy

Elixir Physio. & Anatomy 94 (2016) 40425-40428

ISSN: 2229-712X

Importance of stereotaxic biopsy in definitive diagnosis of brain granulomatosis: Case of neurosarcoidosis an literature review

R. R. Olory-togbe, Y.Arkha, M.Boutarbouch, J.Asfara, A.Ouahabi and A.Khamlichi

Neurosurgery Department of HSR,IBN SINA Medical School of Rabat, Morocco.

ARTICLE INFO

Article history:

Received: 25 June 2015;

Received in revised form:

14 May 2016;

Accepted: 19 May 2016;

Keywords

Calcemia,

Neurosarcoïdosis,

ABSTRACT

The neurosarcoïdosis, is a rare granulomatosis, which causes serious neurological troubles. Until today, there is no consensus about its diagnosis. The stereotaxic biopsy appears to be the gold standard, for histological and definitivel diagnostic of this pathology. Our goal is to show through a case report the importance of the STB, in definitive diagnostic of cerebral granulomatosis; The case is about a young adult, who had clinical neurological disorders, going through an experimental therapeutic, and shows complications, and the STB corrects the diagnosis, so that without this gesture, the patient should have died.

Introduction

Clinical case:

This is a patient 29 years old, having An history of helicobacter Pylori gastritis on treatment, with good outcome. He presented two months before his admission in neurology department, with a cerebellar stato-kinetique syndrome, with swallowing disorders, associated with increased intracranial pressure, (headache, vomiting, decreased visual acuity) in a context of alterationof the general state and apyrexia .Cerebral MRI (done the 20.11.12)shows bulbo-pontine process with Sellaire location, fig1. Biological laboratory tests has been achieved, including a Blood count that shows a normal balance, (See Table 1 below) the rest of the blood and urine electrolytes without abnormalities. The skin test of the Tuberculin (IDR) was negative. Converting enzyme in the blood was normal at 18mg / l. HIV serology also negative.

Table 1

Calcemia	94mg/l	
Sodium	134mEq/l	
Phosphorus	24mg/l	
CRP	0.9mg/l,	
Platelets	517.000/mm³	
White blood cells		11.000/mm³
		(PNN= 87/mm³ ;
		Lymph=131/mm³)
Red blood cells		517.000/mm³
hémoglobin	▪	15.2 g/dl
sedimentation rate in the first hour blood	15mm à la première heure	
Rate of prothrombin	100%	
Converting blood Enzyme	18mg/l (normale)	

Lumbar puncture has not been achieved, due to increased intracranial pressure. The Lung X-Ray as thoracic CT scan shows no chest abnormalities. such chart evoke the posterior fossa syndrome Posterior. The mechanisms referred to, in first place as a hypothesis diagnostic, were

1-Tumor: lymphoma, or infiltrating glioma.

2- Infection: tuberculosis.

3- Inflammatory: sarcoidosis. Because of the negativity of biological laboratory tests, and Contrasting the alarming clinical chart, a Therapeutic treatment test for the Neurosarcoïdosis and tuberculosis was instituted.The patient was then given corticosteroid doses, with an antibacillary treatment. The clinical and radiological outcomes were very satisfying, with a spectacular, Improvement. The MRI 05/12/12 control objectified decrease in size of the process, fig 2. Followed by long-term corticosteroid, with gradual degression (20 mg isone three time by day) with the antituberculous treatment. The Clinical and radiological control was done regularly. The MRI control of the 21 january 2013, 45 days later, objectified recurrence of lesions mésencéphalo-diencephalic and cerebellar, Fig 3. Thus, a balance sheet tangible histological was needed to pursue adequate treatment. In our medical school, we use cortico-meningeal biopsy initially. However, in most of our patients, histological biopsy results of thoses cortico-meningeal proved not conclusive. This is why, in an optimization of Histological evidence We prefer stereotaxic biopsy. Stereotaxic of contrast enhancement of the Initial injury if seat depth. So after multidisciplinary discussions between neurologists, neurosurgeons, and neuroradiologists, we agree for a stereotactic biopsy.

However, the patient presented suddenly, shortly before achieving its BST on 31-01-13, disturbances of consciousness, with a Glasgow coma score 11, was admitted to our emergencies, with increased intracranial pressure, with neck stiff, a Parinaud syndrome, papillary hyperhemia without deficit sensorimotor obvious to the four members. Cerebral CT performed in Emergency shows triventricular hydrocephalus, with trans-ependymal resorption signs and the presence of these mesencephalo-diencephalic lesions and Cerebellar lesions described above Fig 4. Patient was treated at first by an external ventricular shunt, in emergency, to relieve intracranial pressure. Then the chemical and bacteriological CSF laboratory test was

Tele:

E-mail address: regisolma@yahoo.fr

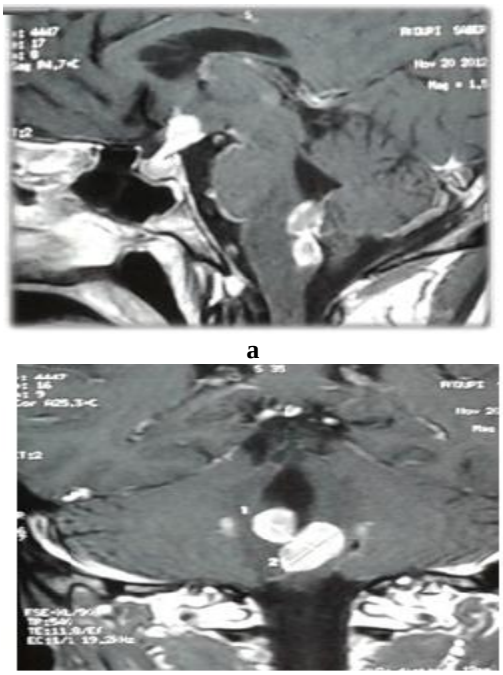
sterile. So, we proceeded to ventriculo-peritoneal shunt, in order to decrease the intracranial pressure. As part of the research the etiological diencéphalo-mésencéphalique tumor, and cerebellar. A stereotactic biopsy (TSB) was Realised, the 11.02.13 on the midbrain lesion. We used for this purpose, a Leksell frame type G, Stockholm (Sweden), the trajectory has been studied on the Leksell SurgiPlan® . A sequence 3DT1 injected, sub-millimeter, multiplanar, with MPR reconstruction was carried out in stereotaxic reconstruction, fig 5, fused to a bone CT-scan, to correct possible distortions. Three fragments were collected in rosette at the Target, histology reveals a morphological and immune-histochemical Aspect corresponding to an inflammatory granuloma compatible with neuro-sarcoidosis origin. The patient was therefore awarded in Full corticosteroid dose, in association with immunosuppressive treatement.

The clinical course in the short, medium and long term is satisfactory. Currently with a period of 30 months, the patient evolved well, with total cleaning of all MRI lesions, fully autonomous, aware of his actions, has restarted working one year ago.

Discussion:

Sarcoidosis is a granulomatous multisystemic, of Unknown etiology. It is a disease of young adults Beginning usually between 20 and 40 years. It usually presents as a bilateral hilar lymphadenopathy, a lung infiltration and skin or eyes lesions, but any organ can be affected. Its impact ranges from 7 to 20 per 100 000 and its prevalence is of the order of 40 per 100 000 [6], [7]. It is more common among black individuals, and female patients. Clinical neurological Achievement occurs in 1 to 5% of cases depending on the series [8], [9] [1]. However, the autopsy series found sarcoid granulomas in the central nervous system approximately 14% of patients with a known sarcoidosis, demonstrating more frequent subclinical reached [10]. She has a great predilection for lung. It is a cold and humid country diseas, with a depressive gradient between the north and the South. (3) Parenchymal involveme of central nervous system is the most serious, responsible sometimes of extremely serious sequelae, sometimes fatal. (4).

This parenchymal involvement, She is often indicative of sarcoidosis, and observed in 36-66% of neurosarcoïdose cases; it is due to the extension of granulomatous process Affecting the leptomeninges. According to Chapelon-Abric, deep nodular lesions, in MRI imaging are due to cerebral demyelination, while those diffuse subcortical and micronodular would be more caused by an arteriolar reached. So in the case of central nervous system damage, the granulomas Favoritely involve in leptomeninges at two Floors: peri-medullary space, and basal cisterns. The existence of meningeal granulomas may cause neurological deficits through several mechanisms: either by infiltration or compression of cranial nerves, vessels or the ventricular system; either by extension of granulomas along perivascular spaces of Virchow-Robin explaining intracerebral and intramedullary locations [11]. The direct vessel wall reache is rare, and mainly concern encephalic vascular structures, of small and medium size and can cause stenosis or vascular occlusions, responsible of ischemia in corresponding areas. [12], [13] . Hydrocephalus is not frequently observed in 6-30% of Cases (1).



a

b

Fig 1. a) sagittal sequence, with gadolinium injection: bulbo-pontine and sellaire process.
b) coronale séquence with gadolinium injection: bulbo-pontine and sellaire process : homogen enhancement.

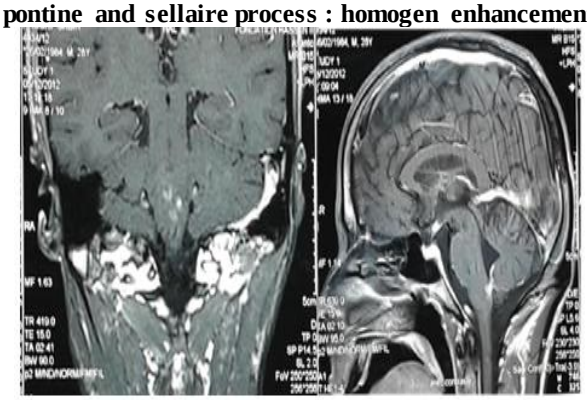


Fig2 a **fig2 b**

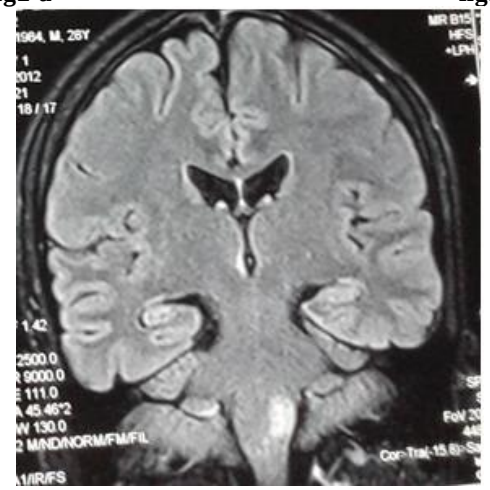


Fig2 c

Fig 2. a,b,c, : significant reduction of tumor volume on trial treatement.

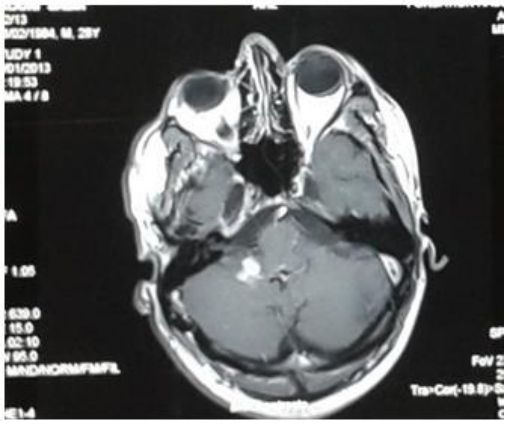


Fig3 a

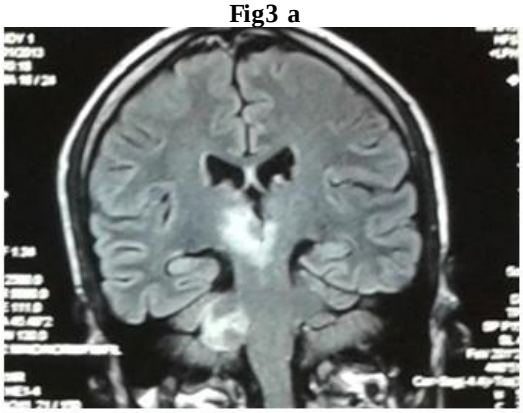


Fig3 b

Fig:3: a: axial sequence MRI T1, showing enhancement of gadolinium injection at the cerebellum level; b:coronal sequence MRI Flair showing reappearence of dincéphalo-mésencéphalic, et cérébellous lesions

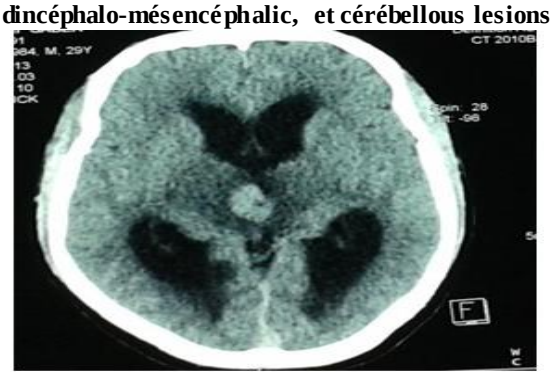


Fig4 a

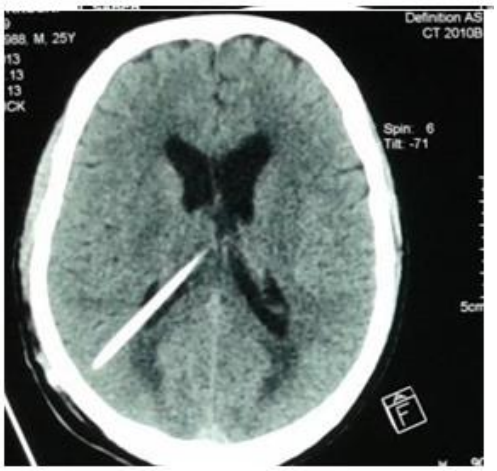


Fig4 b

Fig 4. a. CTscan C- showing acute hydrochephalus, with mesencephalo-diencephalic process. b: CT scan C- after VP ventriculo-peritonéal shunt.

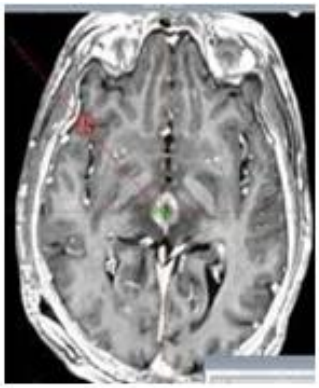


Fig5 A

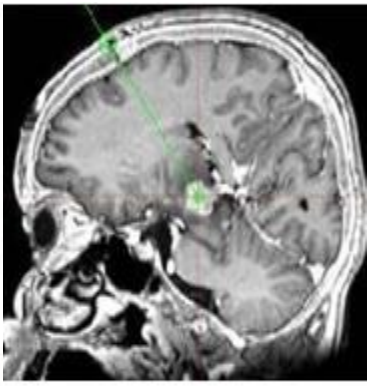


Fig5 B

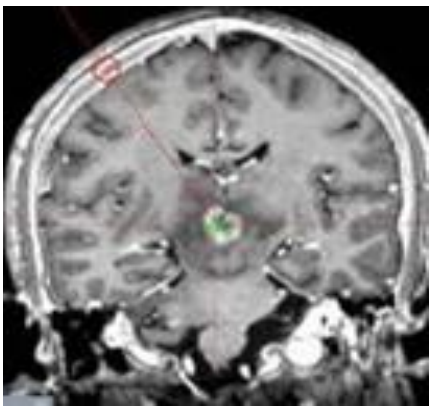


Fig5 C

Fig 5 A, B C : Stereotaxique planning of mesencephalic lesion.

Various mechanisms may the result: the granulomatous infiltration of ependymal and chroroïdes plexus. Chronic meningitis with obstruction of subarachnoid space, or the presence of intraventricular granulomas (1). The rarity of the disease and its Clinical polymorphism lead inevitably to somes difficulties, To evoke and confirm the diagnostic hypothesis in front of an atypical event. Outside of a proof Obtained by histological biopsy of the nervous system, there is no examination who can prove formally the diagnostic neurosarcoidosis. This is why, the stereotactic biopsy finds its place essential as an initial procedure in the definitive diagnosis of this disease, to avoid a trial treatment which is not documented. In our case, the patient's prognosis was at stake, which explains the saving character of the stereotaxique gesture, leading to adequate therapeutic, consistent in. As said previously, cortico-meningeal biopsies are not often conclusive, while the stereotaxic (TSB) offers more reliability as to its sensitivity. This is the reason why, in purpose of optimization of histological evidence, we prefer stereotactic biopsy for lesions that sit deep, on the areas of contrast uptake.

The diagnostic approach must respond to four imperatives: make clinical and laboratory arguments of neurosarcoidosis, search clinico-radiological picture compatible with neurosarcoidosis, having an Histological confirmation of the presence granuloma, noncaseating (2) and finally, eliminate other differential diagnoses, including cerebral tuberculosis and cerebral lymphoma, etc. In our case, TSB supplemented by histology followed By immunohistochemistry are the key Of definitive diagnosis, revealing the look morphological and immunohistochemical in favor of an inflammatory granuloma compatible with sarcoidosis origin.

Current treatment includes Immunosuppressive associated with steroids, according to a defined protocol, in use in our training hospital. The introduction of immunosuppressive treatment Is usual if occurrence of side effects related to corticosteroids, to supplement the therapeutic effect and to minimize the adverse effects of corticosteroid. In conclusion, the BST impose itself as initial gesture for diagnosis of brain granulomatous because of its low morbidity, its sensitivity to avoid complications of treatment test Unconfirmed, which in many cases can Be inefficient, and in our case, would Have been fatal. With the immunosuppressive treatment the patient recovers totally, clinically and radiologically, within a period of 30 months, and now restart working.

References:

(1)-Oksanen V.(1986).Neurosarcoidosis: presentations and clinical course in 50 patients. Acta Neurol Scand 73: 283-90.

(2)-Cacoub P Sbai A, Hausfater P et al. (2000). Reaching revealing medullary sarcoidosis. Rev Neurol (Paris), 156: 896-900.

(3)-Delaney P. (1977). Neurologic manifestations in sarcoidosis. Review of the literature, with a report of 23 cases. Ann Intern Med, 87: 336-45

(4)C.Abric -Chapelon-severe phenotypes of sarcoidosis (excluding lung disease). Rev Prat 008; 58: 1077-1080

(5) D. Ferriby, J. de Seze, T. Stojkovic, E.Hachulla, B. Wallaert, S. et al Blonde Rev Neurol (Paris) 2000; 156 /: 11, 965-975

(6)Christian J, Stanislaus Leguern G, JC Saltiel. Sarcoidosis. Systemic diseases. Flammarion, ed., Paris, 1995.857 to 84.