Hypertrophic Intracranial Pachymeningitis of a Tuberculosis Etiology: Case Report and Systematic Review of the Literature

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ABSTRACT

Background: Intracranial pachymeningitis is a rare neurological condition and tuberculosis as its etiology is uncommon. In the literature, little information is known about the epidemiological profile, clinical presentation, diagnosis, management, and outcome of patients with pachymeningitis of tuberculous origin.

Methods: The authors present a case of tuber- culosis intracranial pachymeningitis with a systematic review of the literature according to the Preferred Reporting Items for Systematic Reviews and Meta - Analyses guidelines. Relevant studies (up to June 2021) that reported patients with intracranial pachymeningitis of tuberculosis origin, were identified from the Google Scholar, PubMed, and Cochrane Library databases.

Results: This systematic review identified 19 patients of whom 11 were male and 8 were female. The mean value of age was 39.42 (Std. Deviation 14.54) years. Eleven patients had intracranial hypertension and hemiparesis while five presented with headache and blurred vision at admission. Surgery was performed in 18 patients. The presence of mycobacterium tuberculosis was not confirmed in one patient treated successfully with the antituberculosis drugs. Furthermore, 18 out of the 19 patients reported improved outcomes, only 1 patient died due to delay seeking health care, thus, delay management.

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Conclusion: Tuberculosis as etiology of intracranial pachymeningitis need to be searched in all patient presenting with this condition whether he is coming from a tuberculous endemic region or not.

Keywords: pachymeningitis, tuberculosis, intracranial hypertension, hypertension, management, case report.

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I. INTRODUCTION

Intracranial hypertrophic pachymeningitis was first described by Charcot and Joffroy in 1869. This rare disorder is characterized by inflammation and thickening of dura mater of diverse etiology. Infective cause includes tubercular, syphilis, fungal, cysticercosis, pseudomonas, Lyme disease, and human T- cell lymphotropic virus infection. Although idiopathic variety is seen in most cases it also has been reported with malignancy, after head trauma, intracranial hypotensinsion, autoimmune syndromes like rheumatoid arthritis, Wagner granulomatosis, Sjogren syndrome, sarcoidosis, Takayasu, and temporal arteritis**.** The main clinical manifestations may include fever, headache, intracranial hypertension syndrome, seizure, blurry vision, motor palsy, or cranial nerve (1–4). These mostly depend on the etiology (whether it is idiopathic or of known cause), the thickness of the abnormally inflamed dura causing compression of anatomic structures, and the topography of the lesion. Nevertheless, rare cases have been reported pointing out tuberculosis as the etiology of this condition.

Idiopathic and Immunoglobulin G4–Related Hypertrophic pachymeningitis confirmed by biopsy sample of the intracranial lesion have been managed with oral steroids and immunomodulators which suppressed the inflammatory markers

and serum biochemistry followed by the reduction in the degree of dural enhancement with clinical improvement in the patient's neurological symptoms. Refractory cases find solutions with rituximab (5–11). Hypertrophic intracranial pachymeningitis can be associated with a variety of medical conditions underlining the complexity of its management.

Some authors have reported cases treated successfully with antituberculosis drugs without any confirmation of mycobacterium tuberculosis isolation (1,12).

In this study, we performed a systematic literature review to evaluate all reported cases with intracranial hypertrophic pachymeningitis of tuberculosis origin and did a meta - analysis of the data. We also reported an illustrative case of a 65-year-old man that was successfully treated with antituberculosis drugs after surgical excision of the lesion.

II. CASE PRESENTATION

A 65-year-old man was admitted to the Neurosurgery Department of Military Teaching Hospital of Rabat with facial palsy. His past medical history was unremarkable. The magnetic resonance imaging (MRI) showed an affected right temporal dural hypertrophied isointense lesion with remarkable contrast enhancement on the T1-weighted and a hypointense lesion with hyperintense edges on the T2 weighted image combined with adjacent cortical thickening (Fig 3) indicating a dense fibrous tissue and inflammatory infiltrates allowing to conclude of a possible intracranial pachymeningitis.

The patient underwent surgery for excision of the right temporal lesion with a dural biopsy sample through craniotomy with a right temporal burr hole. The burr hole was enlarged to expose the maximum diameter of the infiltrated dura matter which was grayish dark in appearance and very tense. We proceed to the coagulation of the dura matter to avoid its bleeding during the procedure. We use a scalpel blade n°11 to open the dura in the crucifix form and milky color material, thick in consistency, pup out and was tightly adherent to the inner surface of the dura matter (Fig 4). The tissue material was sent to the laboratory for analysis and the direct bacteriological search for an infectious cause was negative. Histopathological examination of the tissue revealed necrotizing granulomatous inflammation (Fig 5), PCR for Mycobacterium tuberculosis DNA was positive on tissue. The patient improved with antituberculosis therapy.

III. METHODOLOGY SYSTEMATIC REVIEW

3.1 Search strategy

We followed the guidelines of the Cochrane Handbook for systematic reviews and metaanalysis of diagnostic test accuracy when conducting this systematic review and meta-analysis. This report was written in accordance with the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) guidelines (Fig 1), which were adapted for the current review to increase the comprehensiveness and transparency of reporting [13]. We aimed to identify all full-text, peer-reviewed publications pertaining to intracranial pachymeningitis of a tuberculosis etiology. Published studies in English and French pertaining to intracranial pachymeningitis with identification of mycobacterium tuberculosis as the causal agent were found by utilizing a thorough search strategy of the Google Scholar and PubMed databases from inception to June 2021, with no regional restrictions. The following search terms were used: Intracranial pachymeningitis, tuberculosis, facial palsy, management, outcome. We exclude all articles that are not pachymeningitis of intracranial location and those of non - tuberculosis origin.

3.2 Study selection

All the articles resulting from the search were exported into Rayyan (14), where duplicates were identified and deleted. Rayyan is professional research software that is widely used by collaborators for ease of study selection decisions. The study selection process consisted of multiple steps. Firstly, a minimum of two reviewers independently screened the titles and abstracts of the identified articles based on the predefined inclusion and exclusion criteria. Any disagreement bet-

ween the reviewers' decisions prompted further discussion. If a disagreement persisted, a third reviewer resolved the conflicts. The full texts of the remaining articles were also retrieved and screened by a minimum of two reviewers independently.

IV. DATA EXTRACTION

Data extraction was performed in two stages, a pilot stage followed by a proper stage. The pilot stage consisted of having multiple authors, each going through the same 12 selected articles to extract data. This was to ensure that all participant authors were able to extract data accurately to ensure homogeneity in the reporting of the data and to ensure the data collection sheet captured all relevant and important information from the included studies. Studies that met inclusion criteria were read in full, and the following data were extracted, summarized, and tabulated in an Excel proforma sheet: name of the first author, year of publication, age, sex, symptoms, past medical history of tuberculosis, etiology, imaging, the topography of lesion, management, and outcome.

IV. DATA ANALYSIS

Data were manually introduced into the IBM SPSS Statistics Data Editor v.27.0.1 Software and were pooled in the meta-analysis model using "the maximum likelihood ratio" in the Meta Disc software(15). Interregional and intraregional comparrisons were made using bivariate tests.

V. RESULTS

The comprehensive search returned 4416 studies and we screened 3538 articles (80.11%) after deduplication. The majority of papers were excluded at the title and abstract screening stage (n= 3247, 91.77%,), and the other 255 papers (7.20%) were excluded at full-text screening. Therefore, 12 articles for 19 cases remained for data extraction (0.34%) (Figure 1). The cases were published between 1977 and 2020 with a peak in 1997 (n=7, 36.80%). (Table 1). The patients were 39.42 years old on average (95% CI=32.41-46,43) and most were female 57.9%.

Intracranial hypertension and Hemiparesis were the most common clinical presentation n=11 (57.9%), followed by Headache and blurred vision n=5 (26.3%). Only one case had a past medical history of tuberculosis and mycobacterium tuberculosis was found in almost all of the reported cases n=18 (94.7%). The primary neuroimaging modality was magnetic resonance imaging (MRI) with Hypointense T1 - Hyperintense T2 – weighted in n=9 (47.4%) and Isointense T1 - Hypointense T2 – weighted in n=10 (52.6%). All patients had solitary lesions evenly distributed in occipital n=6 (31.6%), skull base n=5 (26.3%), and frontoparietal n=4 (21.1%). (Table 2) shows the management and outcome distribution of intracranial pachymeningitis of tuberculosis origin, followed by the correlations displays in (Table 3) and (Figure 2).

A Pearson product-moment correlation coefficient was conducted to evaluate the null hypothesis that there is no relationship between the management and outcome of the 19 cases of intracranial pachymeningitis published in the literature (N = 19). Preliminary analysis showed that there were no violations in the assumptions of normality, linearity, or homoscedasticity (see Figure 2 Scatterplot and Table 3). There was significant evidence to reject the null hypothesis and conclude that, there was a strong, positive association between management (M = 0.94, SD = 0.22) and outcome (M = 0.05, SD = 0.22), $r(19) = 0.31$, $p < 0.01$. The combination of surgical and medical management of intracranial pachymeningitis of tuberculosis origin is associated with the favorable outcome.

VI. DISCUSSION

To our knowledge, this is the first systematic review mapping the intracranial hypertrophic pachymeningitis of tuberculosis origin. We identified and extracted data from 19 cases of 12 studies on the name of the first author, year of publication, age, sex, symptoms, past medical history of tuberculosis, etiology, imaging, the topography of lesion, management, and outcome. There was a female patient predominance in most studies. Most tuberculous intracranial hypertrophic pachymeningitis was diagnosed with a head MRI and

was located in the occipital, skull base, and fronto parietal region. The preferred treatment modality was a surgical couple with a targeted medical treatment with antituberculosis drugs with a favorable outcome and the patients were discharged after few days of in-hospital care, to continue oral drugs therapy. Only one death was reported due to delay in seeking healthcare. A Bivariate Pearson correlation allowed us to conclude there is a strong correlation between the choice of surgical and medical management of intracranial pachymeningitis of tuberculosis origin and the outcome.

From the 11 cases of hypertrophic cranial pachymeningitis reviewed by *Shobha et al.,* only one was confirmed to be caused by mycobacterium tuberculosis, and it was a female of 34 year old. This female predominance find in our study is also reported in many studies whether of tuberculosis origin or not. Moreover, most of the patients were young adults less than 45 years old (12,16–18). But the MRI findings are very diverse from one study to another. We found isointense T1-weighted and heterogenous T2 weighted lesion while the four previous authors found T1-weighted hyperintense and T2 weighted hypointense lesion. The heterogeneous aspect of our illustrative case can be explained by the presence of calcification inside the lesion significant to understand the chronicity of the lesion. Most authors agreed on the enhancement of the lesion on T1-Gadolinium. The perilesional edema and the bright enhancement with gadolinium contrast can make the imaging confusing with other intracranial tumors like meningioma, mainly when there is obvious dural attachment (12,16,19,20). Nevertheless, the thickening of the dura is usually found in focal and rarely diffuse pachymeningitis. Focal pachymeningitis appears iso signal T1, iso to hyposignal T2, whereas diffuse pachymeningitis is rather hypersignal T2 (17). Our illustrative case is of a category of focal pachymeningitis but we have heterogeneous T2. Likewise in our series, 50% of cases fall in hyperintense T2 while the other 50% showed hypointensity on T2. In magnetization transfer sequences, the visibility of meninges in T1 MT without gadolinium injection is highly suggestive of tuberculous meningitis. There is a significant difference in the magnetization transfer ratio (MTR) in the different etiologies. MTR in meningeal thickenings is different in tuberculous origin compared to pyogenic, fungal, or viral etiologies. In T1 MT, the percentage of the difference between the two types of disease was different in signal intensity between meninges and adjacent brain parenchyma (T2 and normal MT) is significantly elevated (>20%) in the tuberculosis group compared to the nontuberculous group and may explain the difference in the visibility of the dura (17,21). The imaging diagnosis of pachymeningeal tuberculosis is important because these patients have been shown to respond well to antitubercular treatment, thus avoiding any surgical intervention (22,23). Many of these lesions share similar imaging characteristics as meningiomas; however, some distinctive imaging findings, specific history, and supportive laboratory findings often assist in reaching an appropriate diagnosis.

Histopathological examination of the tissue revealed necrotizing granulomatous inflammation with central areas of caseous necrosis bordered by granulomas with giant cells and lymphocytes in most cases reported. Only one case was described with status epilepticus as a symptom presented at the admission. There were no specific clinical findings related to intracranial hypertrophic pachymeningeal tuberculosis. The patient was given antitubercular treatment and showed complete resolution of the neurologic findings including the cranial neuropathy. All of the patients improved markedly with antitubercular medication and some authors added steroids for at least six weeks. The medication for tuberculosis was continued from 9 to 12 months with a good response (1,24). In our series, the surgical indication was not only for biopsy to get a sample for laboratory analysis, but the resection of the thickened dura is a way of decompression and reduction of the cortical irritation, thus, participating in the quick relief of the signs and symptoms of the patients. This should explain the tight correlation between the management (surgical and medical) and the good outcome. Moreover, the radiologic evidence of pulmonary tuberculosis was confirmed in only one case in our series; meaning the patient's past

medical history of tuberculosis or contagion is not significant enough to decide whether or not he may develop tuberculous pachymeningitis.

Some authors deal with cases that were managed as tuberculous pachymeningitis just on the basis of strong suspicion since the patient is living in a tuberculous endemic area or based on the clinics and radiological findings. The results are quite acceptable even if it is not an evidence-based practice (8,25–27). This raises the issue of having many more cases of tuberculous pachymeningitis undiagnosed and untreated properly; making our series not exhaustive. Another systematic review could be done to clarify the case of the non-tuberculous pachymeningitis that responded to antitubercular therapy.

Despite a systematic and extensive literature search, the quality of conclusions that can be drawn from this study is limited by the available literature, which was extremely sparse. However, this lack of available literature has served to highlight the underreporting of intracranial hypertrophic pachymeningeal tuberculosis. Moreover, we were only able to include articles published in English or French. This means literature published in alternative languages such as Spanish and Arabic will have been omitted from our analysis.

Further to the limited number of included articles, only data from Google Scholar and PubMed were captured in this study.

VII. CONCLUSION

There is no specific clinical presentation of intracranial hypertrophic pachymeningitis of tuberculous origin and the iso signal T1 and hyposignal to heterogeneous T2 on the MRI with a thickened dura is a keynote to redirect our diagnosis to intracranial hypertrophic pachymeningeal tuberculosis based on the clinical presentation and to be confirmed by histopathological findings. Surgical excision of the lesion coupled with a 9 to 12 months course of the antitubercular drug is necessary for a better outcome.

Informed Consent: The patient gave his informed consent to publish his case.

Conflicts of Interest: The authors declare that they have no conflicts of interest.

Table 1: Characteristics of intracranial tuberculous pachymeningitis in the literature

Characteristic	Frequency (Percentage)
First Author	
Aggarwal	1 (5.3)
Akhaddar	1 (5.3)
Fonseka	1 (5.3)
Goyal	7 (36.8)
Jacques	1 (5.3)
Kettani	1 (5.3)
ParneyIan	1 (5.3)
Sharma	1 (5.3)
Shobha	1 (5.3)
Tariq	1 (5.3)
Voider	2 (10.5)
Yamashita	1 (5.3)
Publication year	
1977-1997	11 (57.9)
1998-2008	2 (10.5)
2009-2019	5 (26.3)
2020	1 (5.3)
Symptoms	

Facial palsy	1 (5.3)
Headache - blurred vision	5 (26.3)
Fever - Seizure	1 (5.3)
Facial palsy - Seizure	1 (5.3)
Intracranial hypertension - Hemiparesis	11 (57.9)
Imaging findings	
Hypointense T1 - Hyperintense T2 - Enhancement	9 (47.4)
Isointense T1 - Hyporintense T2 - Enhancement	10 (52.6)
Location of lesion	
Frontal	
Fronto-parietal	1 (5.3)
Temporal	4 (21.1)
Occipital	1 (5.3)
Skull base	6 (31.6)
Fronto-temporal	5 (26.3)
	2 (10.5)

Table 2: Management and outcome of tuberculosis pachymeningitis in the Literature

Management (Mean = 0.97)		Outcome (Mean=0.05)		Lower limit 95% CI of mean	Upper limit 95% CI of mean
Medical	5.3%			0.83	1.05
Surgical & Medical	94.7%				
		Favorable	94.7%	-0.05	0.16
		Non-favorable	5.3%		

Table 3: Correlations

		Management	Outcome
Management	Pearson Correlation	1	,056
	Sig. (2-tailed)		,821
	N	19	19
Outcome	Pearson Correlation	,056	1
	Sig. (2-tailed)	,821	
	N	19	19

Fig. 1. Intracranial pachymeningitis of a tuberculosis etiology search strategy using PRISMA flowchart

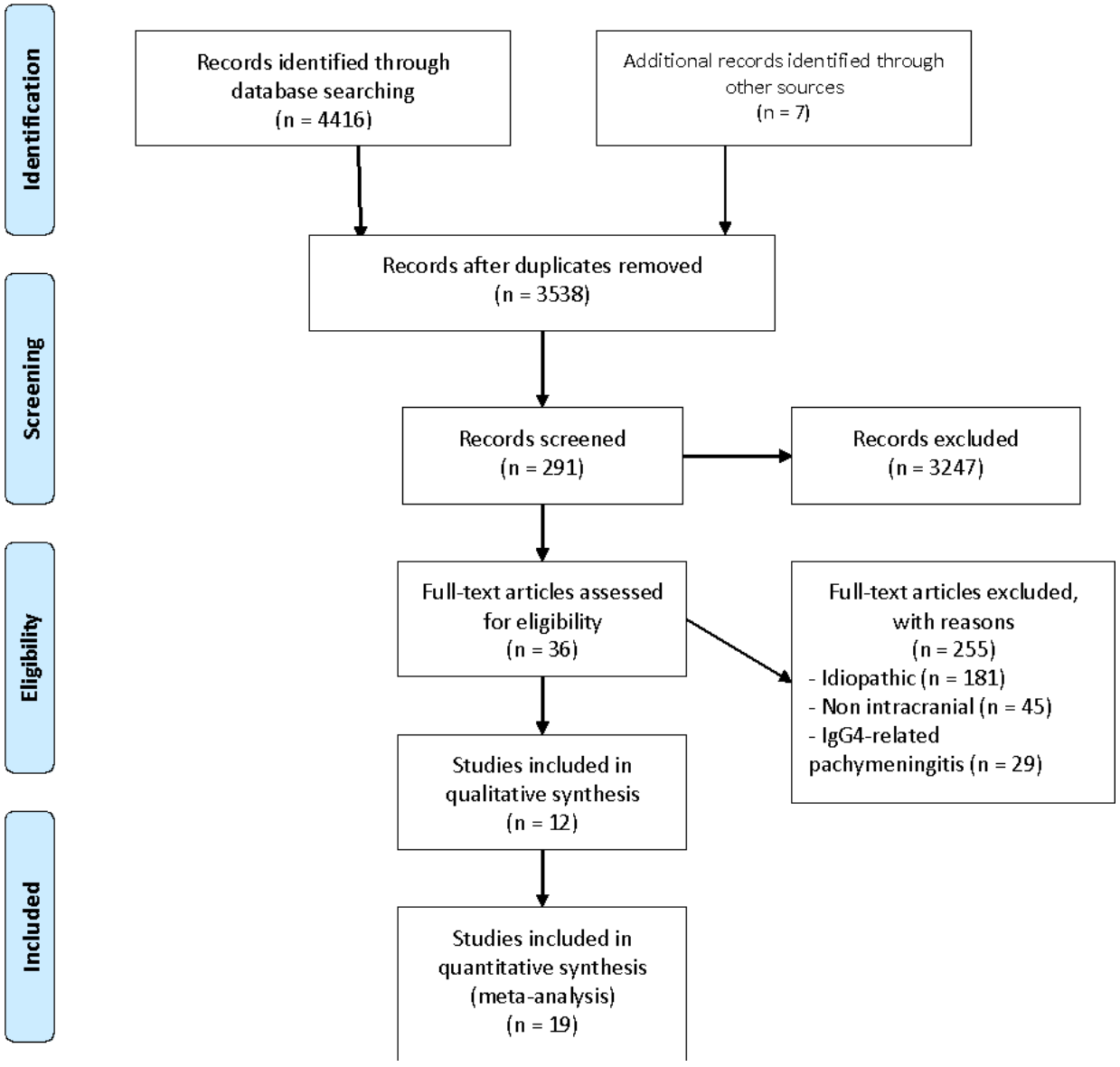


Figure 1: Search Strategy of PRISMA flow chart

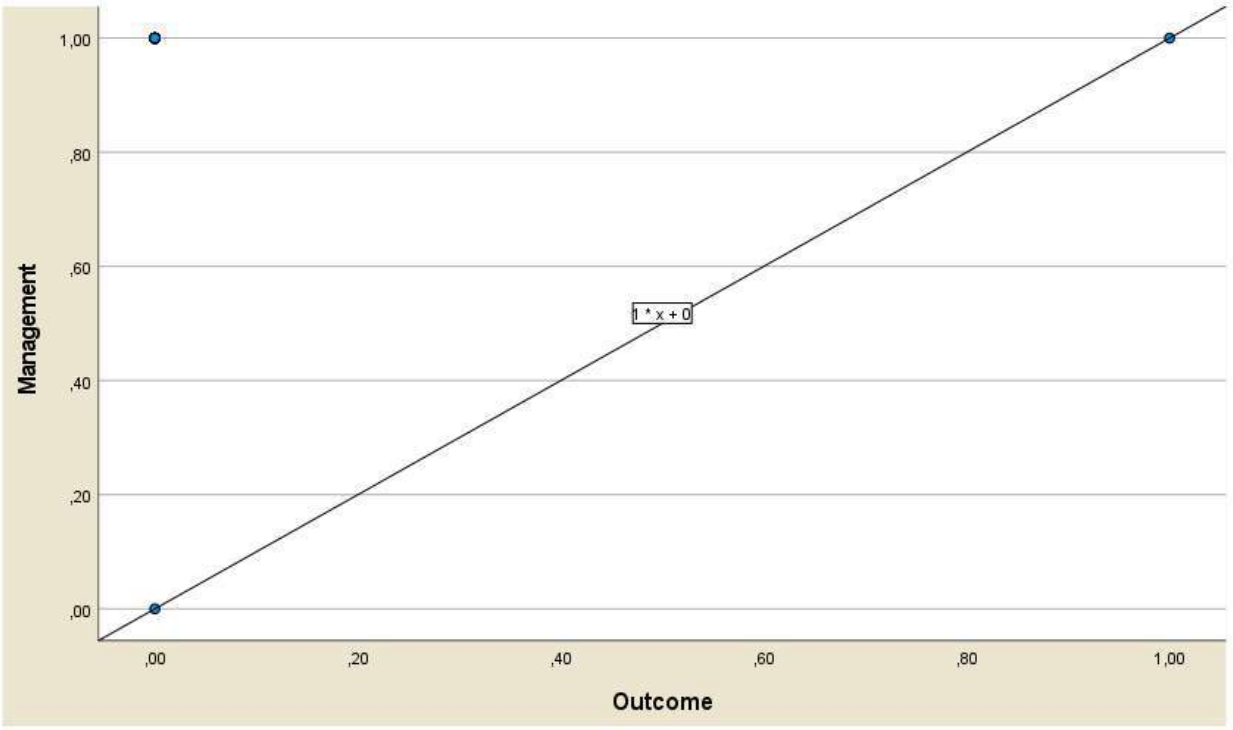
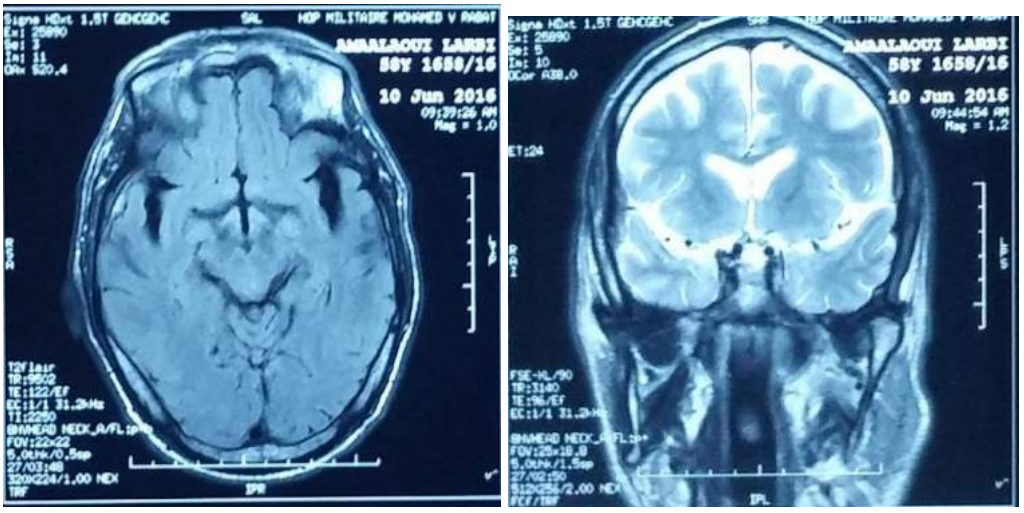


Figure 2: Correlations of management and outcome



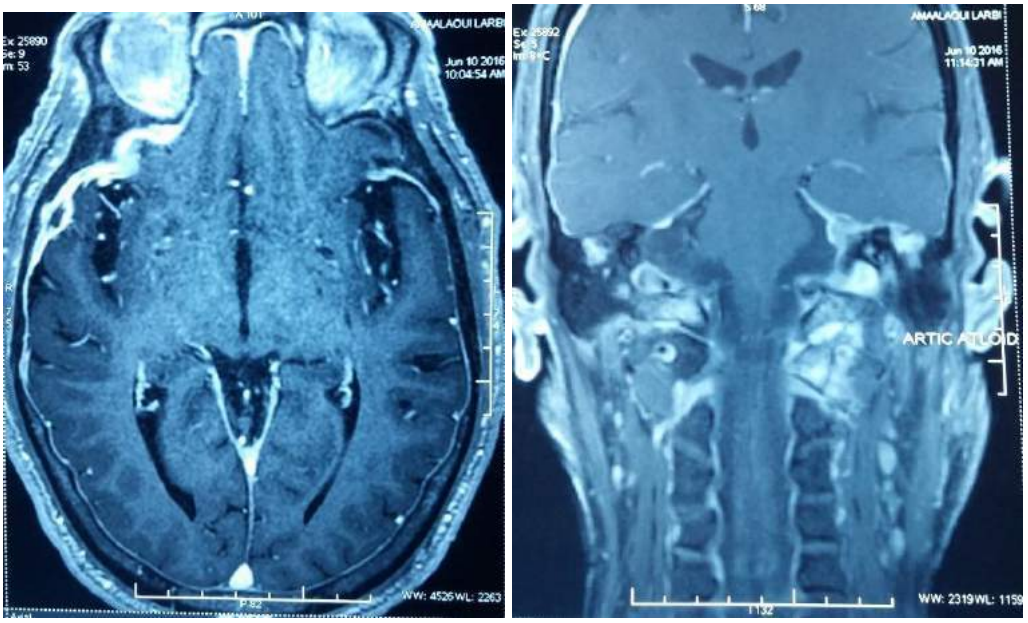


Figure 3: Facial palsy and MRI

A/ Facial palsy; **B-C-D-E/** Brain MRI showing (white arrow) respectively T1-weighted temporal low intensity lesion, T2-weighted heterogenous lesion, T1-Gadolinium highly enhanced lesion, and heterogenous FLAIRE

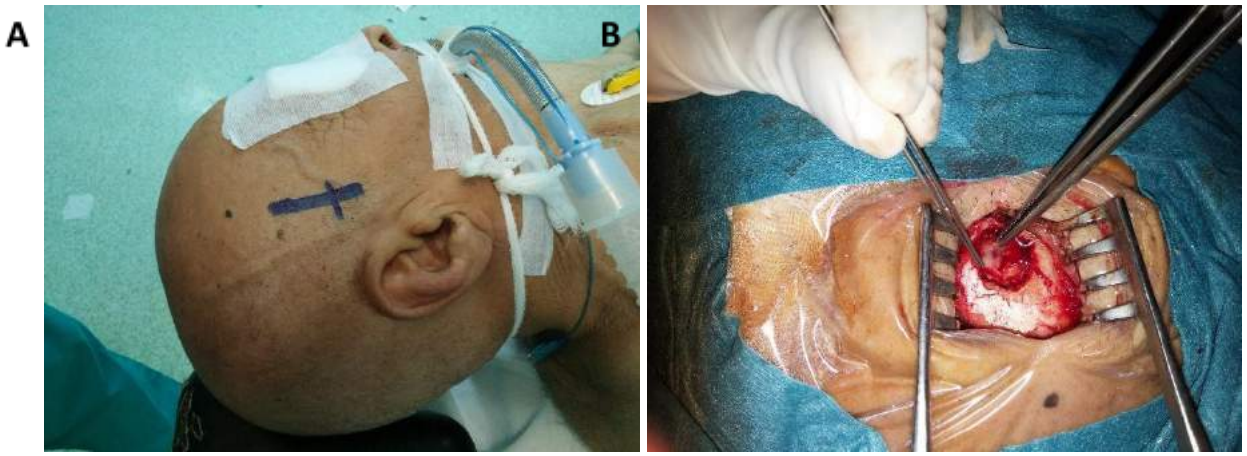


Figure 4: Per-operative excision of the lesion

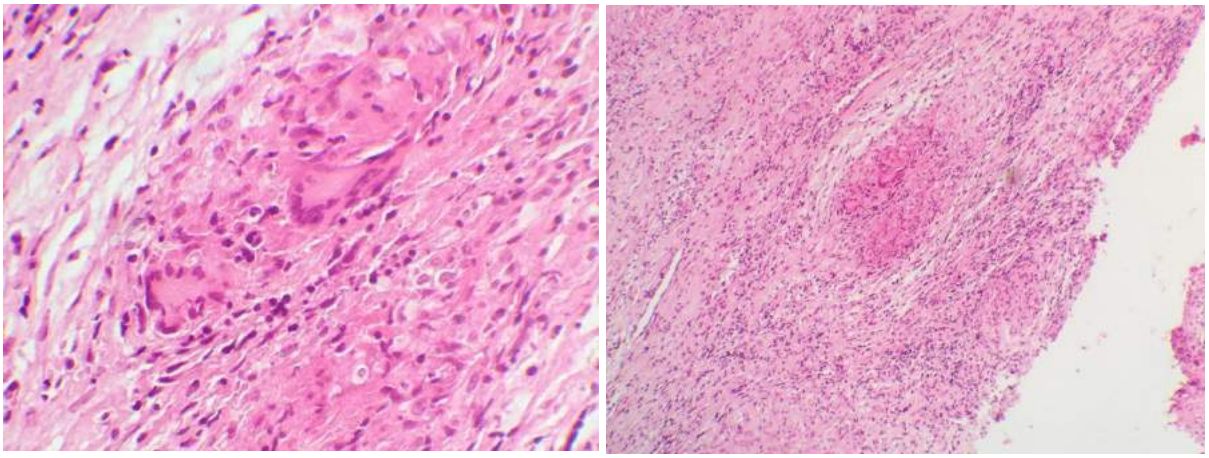


Figure 5: Histopathological result

A/ (haematoxylin and eosin) X 100: photomicrograph of the surgical specimen showing perivascular and diffuse lymphoplasmacellular infiltrates and vascularised fibrous tissue surrounding meningothelial proliferates. **B/** Necrotizing granuloma with giant cells in a dense fibrous tissue infiltrated by polymorphs, eosinophils and plasma cells (X 100).

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