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Symmetric Syphilitic Gummas of the Frontal Lobes

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Summary. An anatomo-clinical observation of symmetrical syphilitic gummas in the frontal lobes is described and some problems related to the endocranial localization of syphilitic gummas are reviewed.

The difficulty establishing a differential clinical diagnosis between cerebral syphilitic gumma and other intracranial neoformations is discussed. Neurosurgical intervention is still essential for a syphilitic gumma.

"Tentative" specific medical treatment is usually ineffective and entails serious risks. Post-operative specific medical treatment (following histological confirmation) is recommended since parenchymal inflammation and syphilitic arteritis can be improved.

Key words: Gumma, Syphilitic, Endocranial — Clinic and Pathology.

Zusammenfassung. Es wird eine klinisch-anatomische Beobachtung symmetrischer syphilitischer Gummata im Frontallappen mitgeteilt und die Problematik der endokraniellen Lokalisation syphilitischer Gummata diskutiert.

Die Schwierigkeiten der Differentialdiagnose zwischen syphilitischem Gumma und anderen intrakraniellen Neubildungen wird besprochen und die Wichtigkeit der neurochirurgischen Behandlung hervorgehoben. Klinische Versuche mit einer spezifischen Therapie bleiben meistens erfolglos und sind riskant. Die postoperative spezifische Therapie nach gesicherter histopathologischer Diagnose ist empfehlenswert und wirkt sich auf die parenchymale Entzündung und die luetische Arteritis aus.

Schlüsselwörter: Gumma, syphilitisches, endokranielles — Klinik und Pathologie.

Percentages quoted for the incidence of syphilitic gummas on expansive endocranial lesions vary considerably between early and recent statistics.

In 1910, Starr (in De Franco, 1940) quoted a percentage of 4.4%. However, with the expansion of neurosurgery, this percentage underwent a sudden drop. Cushing (1923) determined 0.6% in 2000 endocranial masses, Cl. Vincent (in Sezary, 1938) 0.2% in 1500 cases, Bagdasar (1929) 0.1% in 1550 cases and Roger and Paillas (1939) 0.5%. More recent statistics (cf. Marra, 1960) give 0.4—0.5%.

According to Sezary, the sudden drop in percentage was due exclusively to a more systematic operative check and improved diagnosis. Bianchi and Frera (1957), on the other hand, claim that it was real and

coincided with the more widespread application of chemio-therapeutic treatments.

The rareness of syphilitic gummas in cerebral tissues is ascribable (Sezary, Dujardin, 1921) to the scarce atypical participation of the latter in the allergic process, and more particularly, in its necrotizing activity. This particularity does not, of course, extend to the meninges or vascular structures.

Bagdasar rightly criticized the abusive diagnosis of syphilitic gumma in syndromes of the endocranial mass, but to-day the other extreme has been reached in that this diagnosis is no longer taken into consideration even hypothetically.

However, it should not be forgotten that treponemosis appears to have increased again during the last few years and that the percentage of endocranial granulomatous lesions in syphilis is not irrelevant to the problem. This percentage was given as 6.3% of 4000 cases by Fournier and 19% of 2023 cases by Among (in Oblu *et al.*, 1964).

The following anatomo-clinical case of multiple cerebral syphilitic gummas presenting a very unusual topography provides an interesting basis for a discussion of some of the specific problems involved.

Personal Observation

This observation concerns a working-class man (L. Ernst), whose case history was virtually uneventful, both from a family and a physiological standpoint. He was the youngest of 12 brothers. His physiological reactions, behaviour and integration into social life may be described as adequate in infancy, adolescence and when he was a young man. Between 10 and 14 years he is believed to have been subjected to carnal assaults by his elder brothers. At 22 years of age he married a healthy girl. No children were born of the marriage. There were no pathological precedents except a specific cervical lympho-adenitis that was cured without leaving after-effects.

At the age of 29 he suffered his first attack of convulsions which spread from a focal source (clonic contractions of the right muscles) and was followed by post-critical hemiparesis of the right side. During the 5 years that followed he suffered more attacks that were of the same nature but more frequent.

At the age of 34 he was admitted to the cantonal hospital of Berne. The cause of the critical symptoms was traced to an endocranial neoformation situated on the left side of the forehead. This was removed by a surgical intervention.

Microscopic examination revealed connective tissue with meshes of varying width that contained necrotic material and formed the greater part of the interwoven mass. This fibro-necrotic centre was surrounded by a loosely woven network of granulations containing fibroblasts, lymphocytes, plasma cells, a few giant cells and some groups of epitheloid cells. Vascularization was relatively abundant and the walls of the arterioles were often deteriorated. More excentrically, a florid gliosis with large astrocytes was observed, together with extensive perivascular infiltrations of round cells. No tubercular bacilli or spirochetes (Levaditi stain) were brought to light.

The histological diagnosis was *syphilitic cerebral gumma.*

After the operation the patient underwent an antiepileptic treatment. The critical symptomatology disappeared for three months. It then reappeared but the interval between the attacks was very much longer than during the pre-operative period.

From the start of the epileptic attacks psychic anomalies and lapses in behaviour began to appear and grew progressively worse. These were characterized by general physical deterioration accompanied by conceptive deceleration, impaired mnestic and critical functions, sudden bouts of aggressivity and obscene language and behaviour. During this same period sexual capacity began to diminish considerably.

After twice having been reported for sexual offences (between 44 and 49 years), a psychiatric examination was ordered. The judicial authorities suggested medical custody or, alternatively, surgical castration. The latter solution was chosen by the family.

During the 7 years that followed the psychic deterioration grew gradually worse. The antiepileptic treatment reduced the convulsions to a minimum of 3 per year. No more lapses occurred in sexual behaviour.

At the age of 56 the patient was admitted to the Psychiatric Clinic of the University of Berne. His wife reported that the attacks of convulsions had become frequent again, that his physical condition had deteriorated rapidly and that serious disturbances had appeared in deglution and in sphincteral control.

On admittance a serious deterioration was noted in the general state. The patient presented a marked global demential syndrome, predominantly verbal aphasia accompanied by dysarthria, oppositional resistance to the opening of the mouth, forced mimico-emotive explosions and slight facio-brachio-crural hemisyndrome of the right side.

A short-lived improvement was followed by a renewed deterioration in the neurological symptomatology. Hyperthermia appeared for no apparent clinical reasons and resisted antibiotic and antipyretic treatment.

Consciousness gradually decreased and the patient passed from a subconfusional torpor to a coma. Exitus occurred due to cardiorespiratory insufficiency 1 month and a half after admittance.

It should be pointed out that specific syphilitic reactions on serum and spinal fluid were always negative. Inquiry into the case history failed to establish at what period the venereal contagion had taken place.

The autopsy was limited to the cranial cavity.

Macroscopic Examination

A semicircular surgical osseous ridge, approx. 10 cm at the base, was present on the left temple. The dura mater adhered to the top of the skull in the region of the surgical ridge and the arachnoid to the dura mater in the vicinity of the pacchionian bodies. The pia mater was smooth and transparent throughout. The Willi's circle arteries were pervious and slightly elastic. A cicatricial retraction was clearly visible in the fronto-central cortical and subcortical regions of the left hemisphere.

1. Frontal Section through the Median Third of the Corpus callosum (Fig. 1)

Left Hemisphere (L). A demarcated crater-like defect appeared in the cortex and white matter of the first two frontal convolutions. The lesion

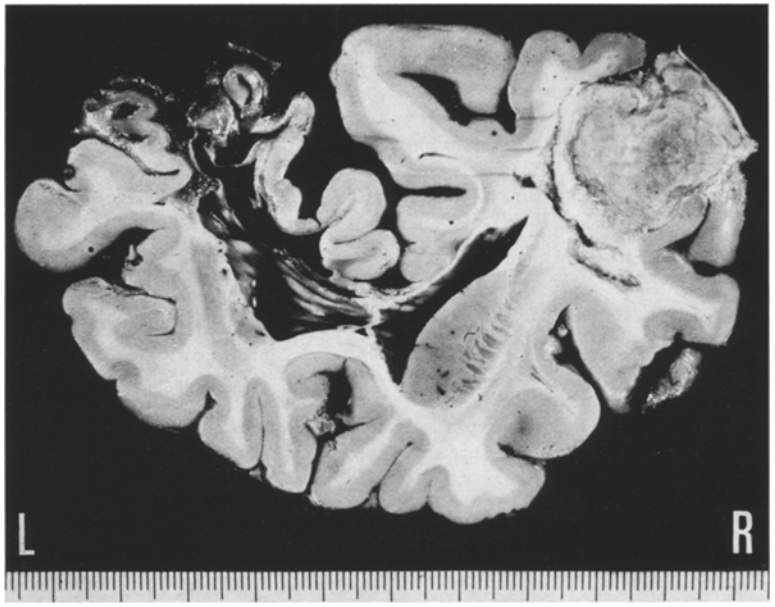

Fig. 1. Left hemisphere (*L*): crater-like defect with a retracted scar extending from the first two convolutions deep intu the lateral ventricle. Right hemisphere (*R*): an extraparenchymal mass in almost symmetrical position. Areas of softening of the white matter below the mass are visible

grew deeper rostro-medially towards the ependyma of the ventricular frontal horn and also affected the convolution of the cingulum.

Right Hemisphere (R). A compact roundish nodule the size of a cherry appeared in a position almost symmetrical with the controlateral cicatricial lesion. This appeared to be bounded by a fibrous capsule that was invested, from the cortical surface onwards, in the white matter of the centrum ovale. The white matter in contact with the mass appeared to have softened as far as the region of the ventricular ependyma.

2. Frontal Section through the Mammillary Bodies

Left Hemisphere. A cystic formation with smooth fibrous walls was observed in the white matter of the first frontal convolution, while the second and third convolutions were softened as in ischemic necrosis. The softening became deeper towards the caudate nucleus but, macroscopically, the putamen was intact.

Right Hemisphere. A transverse softening was observed in the dorsal white matter of the insula.

3. The *frontal sections* cut more caudally did not reveal any macroscopic pathological phenomenon.

Microscopic Examination

Specimens of myelin (Woelcke-Heidenhain, Weil, Weigert), cells (Nissl cresyl violet, hematoxylin-eosin), collagen and reticulin (van Gieson, Perdrau) were prepared, following fixation in 10% formalin.

The most significant frontal sections were those that passed through the genu corporis callosi.

Left Hemisphere (Scar). A retracted amyelinic scar appeared in the first and second frontal convolutions. There was also, in the centre of the centrum semiovale, a smooth-walled cavitary cyst (Fig. 2). Demyelination diminished gradually peripherical to the cyst (where a few spongious foci were observed) towards the white matter of the cingulate convolution. In the latter region some phenomena typical of ischemia of the white matter were noted accompanied by focal periarteriolar demyelination and relative immunity of the subcortical Meynert's fibres.

In the second frontal convolution the cytological specimens presented more evident symptoms of tissular damage. Here, the fibro-glial scar was very conspicuous. A slight, diffuse microglial reaction was observed both in the cortex and in the white matter. There were no perivascular in-

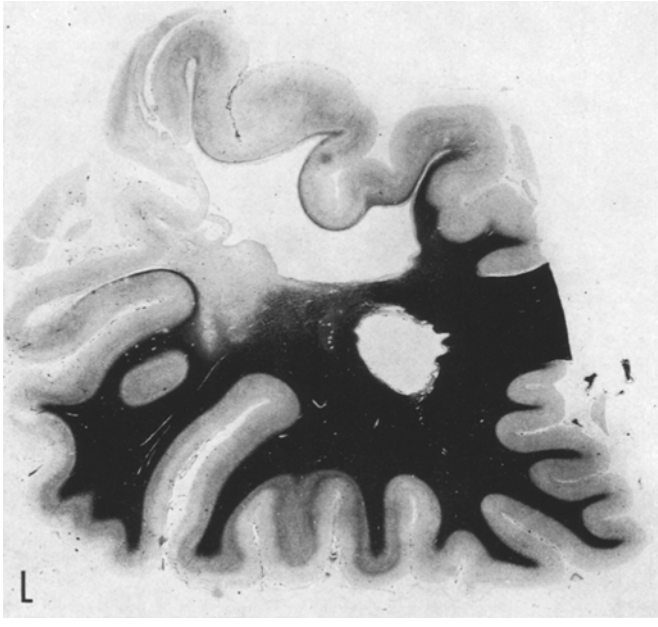

Fig. 2. Left hemisphere. An amyelinic scar in the first and second frontal convolutions and smooth-walled cyst are visible in the subcortex. Woelcke-Heidenhain stain

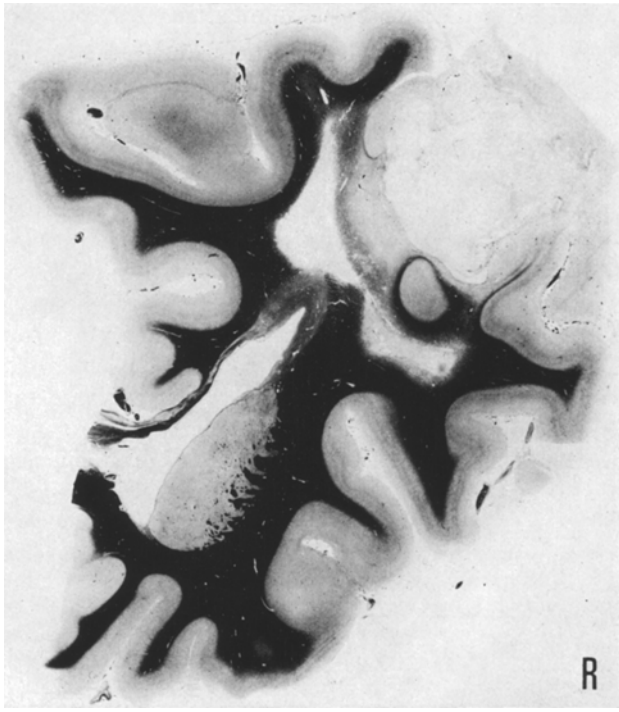

Fig. 3. Right hemisphere. Extraparenchymal mass embedded in the first and second frontal convolutions. Note a large crescent-like area due to the ischemic softening in the underlying white matter. Heidenhain-Woelcke stain

filtrations but a thickening appeared in the arteriolar walls and affected the tunicas.

In the demyelinated white matter gliosis was accompanied by a fibrolastico-collagenous formation of a perivascular source. The aforesaid meningeal reaction was hyperplastic.

Right Hemisphere (Granuloma). In the myelin specimens (Fig. 3), the cuneate extraparenchymal mass was clearly visible in the second and third frontal convolutions. Adjacent regions presented pronounced demyelination. Below the mass, but not in topographical continuity with it, a semilunar band of demyelination appeared in the white matter of the second and third frontal convolutions but, here again, the subcortical Meynert's fibres had been spared at several points. The characteristics of the latter lesion resembled those of ischemic necrosis. Another centre of ischemic demyelination ran from the angle of the lateral ventricle dissecting the fibres of the corpus callosum longitudinally.

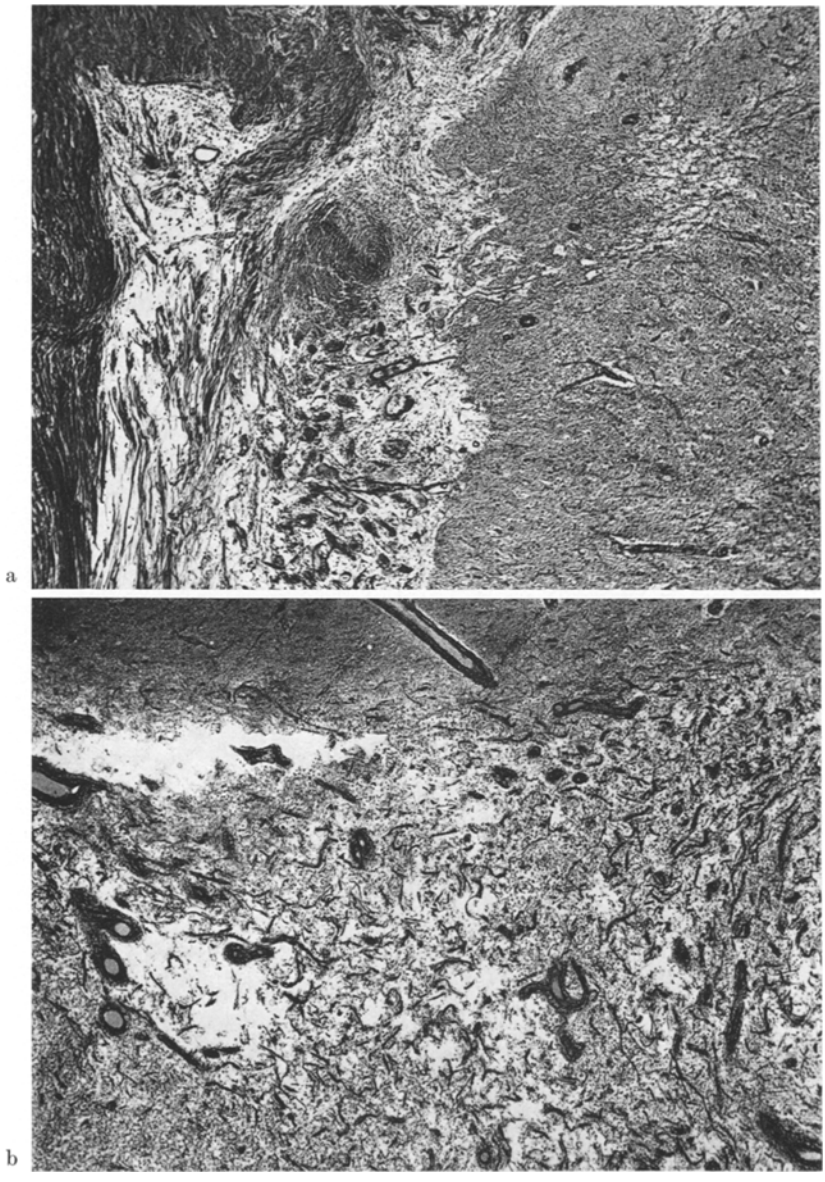

Fig. 4. a Passage between syphilitic granuloma (left) and cerebral tissue. Collagenous bands, perivascular infiltration, necrotic and spongious areas of the parenchyma are clearly visible. Van Gieson stain. b A higher magnification showing typical features of the granulomatous tissue. Van Gieson stain

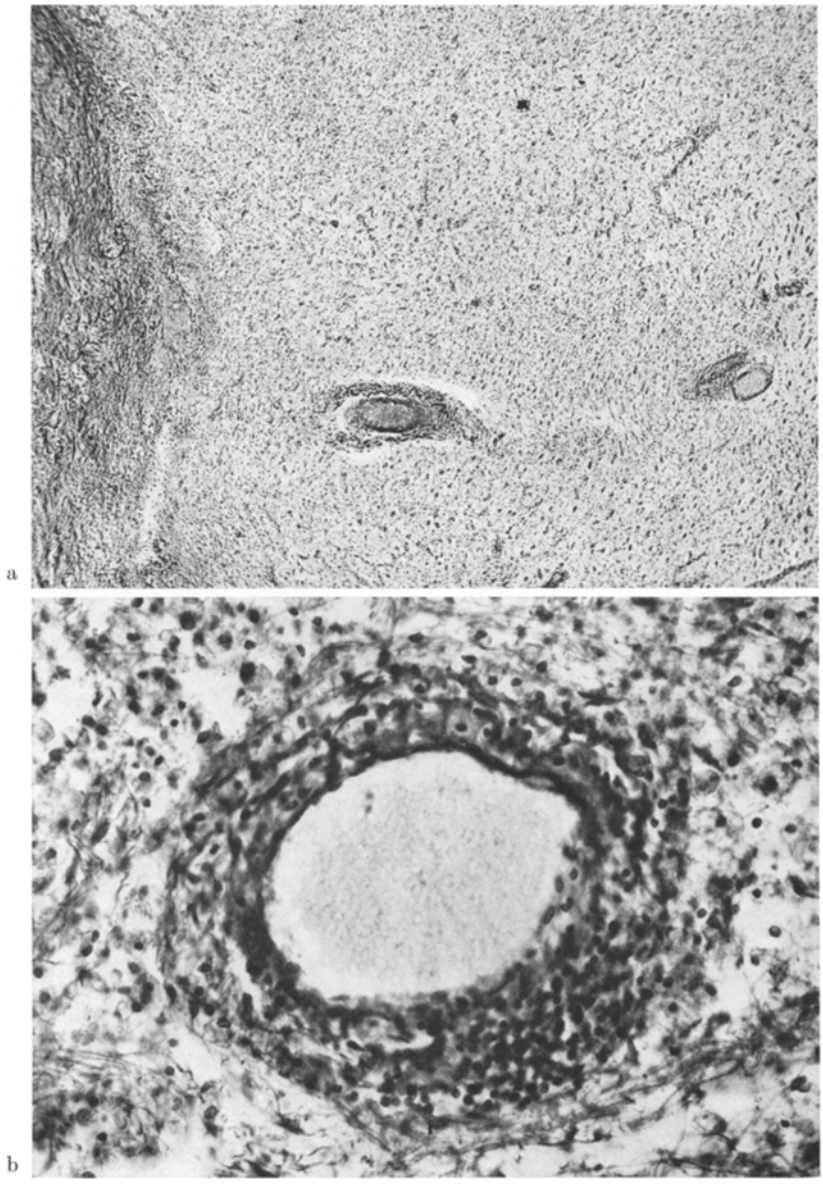

Fig. 5. a Perivascular infiltrates in the cerebral tissue adjacent to the granuloma. Hematoxylin-eosin stain. b Higher power view of the specific arteritis. Hematoxylin-eosin stain

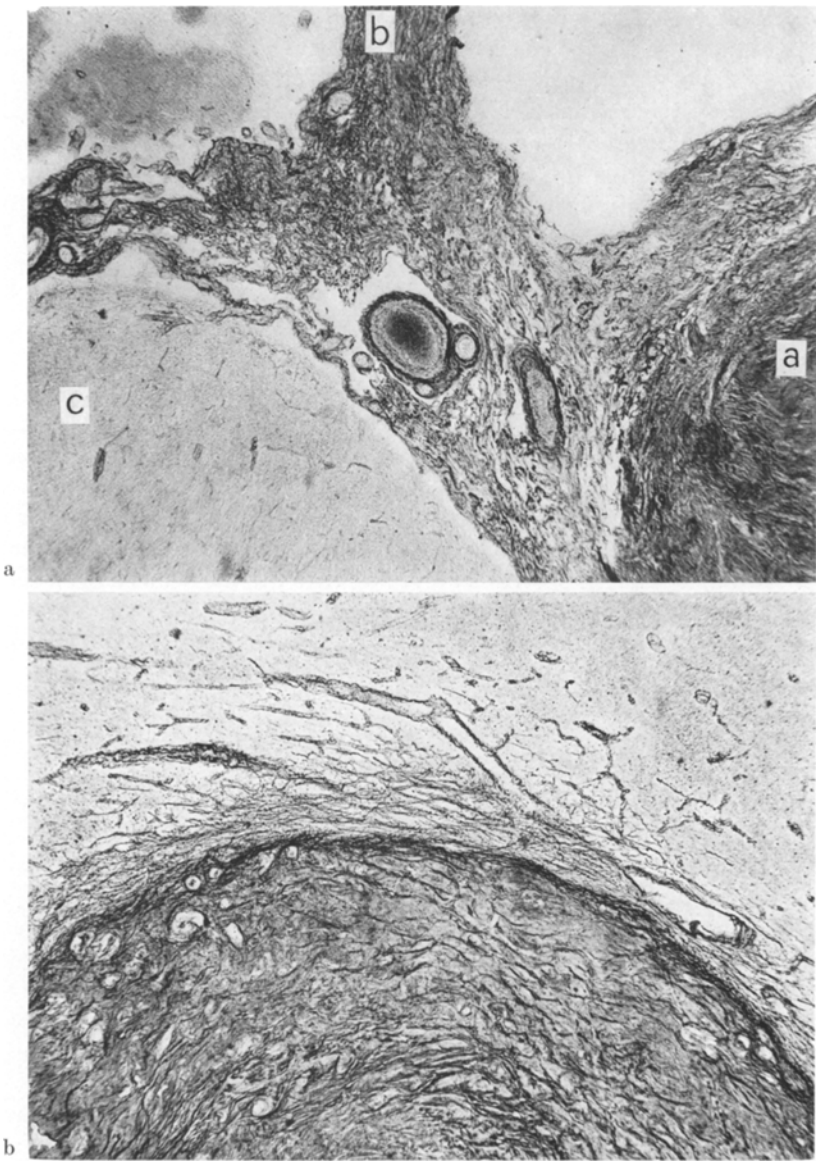

Fig. 6. a Relationships between the fibrous structure of the granuloma (*a*), meninx (*b*) and cerebral tissue (*c*). Perdrau stain. b View of the well defined fibrous wall around the granuloma. Perdrau stain

Cytological stains: the centre of the mass was composed of a homogenous acidophilic caseous substance. Thrombosed vessels that gave rise to fibrous bands and coils were indistinctly perceived. The mass adhered to the meninx which presented marked deterioration with fibrous thickening and perivascular infiltrations of round cells. The regions bordering on the cerebral parenchyma, on the other hand, were relatively clear. The marginal cortex was necrotic at several points and presented spongious areas (Fig. 4).

A web of granulation with a neoformation of small vessels, fibroblasts, plasma cells, epitheloid cells, some giant cells and an irregular network of collagen and reticulin was observed at the edges of the mass (Fig. 4, a and b).

More excentrically (Fig. 5a), infiltrations arranged in more clearly defined perivascular layers appeared. Here, some distinctly arteritic symptoms were identified (mostly meso-arteritic with endothelial reaction; Fig. 5b).

In the demyelinated semilunar area below the mass, the histological picture was typical of ischemic necrosis of not very recent date. The reticulin and collagen specimens revealed that the mass was supported by a thick web (Fig. 6a) that continued into the aforesaid fibrous formation of the meninx and condensed into a relatively clearly defined capsule in the region of the cerebral parenchyma (Fig. 6b).

Discussion

In view of the considerations set out in the introduction, it is clear that interest is to be gained from the collection and discussion of the general, anatomo-pathological, clinical and therapeutic data related to this field of pathology.

Case histories rarely help the diagnosis since such a long period elapses between the primary infection and the development of the gumma, and the granulomatous lesion develops so slowly. Both periods are usually calculated in terms of years. Cases of early development and rapid evolution are rare and are only found in meningeal gummas (François and Verriest, 1955).

Often the only way to trace the origin of the venereal contagion (Salles and Pecker, 1963) is to enquire more closely into the case history "a posteriori".

Many authors (Nonne, 1915; Houston Merritt *et al.*, 1946) have postulated that traumatic events may assist the various manifestations of neurosyphilis during a general treponemic infection, a hypothesis that has been given various pathogenic interpretations (Babonneix and David, 1917; Houston Merritt *et al.*).

The general opinion is that the typical origin of syphilitic gumma is in the meningeal and vascular structures. The syphilitic granuloma may appear as such from the start, or it may take the form of circumscribed chronicity of a specific diffuse meningitis.

Less frequently the productive lesion develops in the cerebral parenchyma and implants itself on the perivascular structures with a mesenchymal matrix.

A frequent place of origin is the base of the cranial nerves or the base of the infundibulo-hypophysial region where large vessels are affected (Walsh, 1947; Cohn, 1923; François and Verriest). Localizations on the convex face of the hemisphere are also frequent (Salles and Pecker; Bianchi and Frera; Bagdasar). Other places of origin such as the 3rd ventricle (De Franco), the corpus callosum (Donat, 1939), the cerebellum (Rigotti, 1950; Dufour, 1913) and the spinal cord (Oblu *et al.*), etc. are rarer.

Since the histological characteristics of syphilitic gumma are classic, only a few particular observations will be described.

Some cerebral lesions with necrotic centres have been observed in progressive paralytics submitted periodically to malariotherapy. Such lesions have often been identified as syphilitic gummas, probably in accordance with the hypothesis that malariotherapy is a means of modifying the allergic reactivity of the nervous tissue. It is probable that this also explains the high statistic incidence of cerebral syphilitic gumma claimed in the case histories prior to the 1930's.

To avoid erroneous interpretation, it is today unanimously agreed that the identification of caseous material in the histological definition of syphilitic gumma is indispensable, although not specific.

Meningeal gummas tend to invest the cerebral tissue. The cleavage plane towards the latter may be clearly visible (Salles and Pecker), but it is more often not very well defined (Roger *et al.*).

In some cases it may not be easy to differentiate it from tubercular granuloma. The diagnosis of the latter is deducted from development at a younger age, the preferred subtentorial localization, smaller dimensions, the immunity of the osteo-meningeal tissue and inauspicious surgical prognosis.

From the histological standpoint it should be pointed out that in syphilitic gummas the connective tissue formation develops from the start and does not merely represent, as in a tuberculoma, a cicatricial repair. The peculiar characteristics of syphilitic arteritis also help in this respect (it is equivalent to a panarteritis in which the tunica elastica is unaffected and duplicated and the outer coats are sclerosed).

Clinical and Instrumental Diagnosis

In most cases, the clinical symptomatology of cerebral syphilitic gumma does not differ to any great extent from that of any other slowly developing extraparenchymal endocranial mass.

The endocranial hypertension syndrome is not precocious (Pussepp, 1920) and it is usually preceded by deficitary or irritative symptoms localized focally in the cerebrum. Partial or Jacksonian epileptic manifestations are extremely frequent.

The usually associated specific arteritis explains the frequent satellite symptomatology of a cerebro-vascular nature. This may render a differential clinical diagnosis even more difficult in the case, for example, of endocranial meningioma.

A symptom as significant for the diagnosis of neurosyphilis as that of Argyll-Robertson is rarely met in the case of cerebral gummas.

The data obtained from serological and spinal fluid tests are only indicative in the specific sense of this particular condition.

An investigation of the *Treponema* or its culture is of little practical assistance since the spirochete rarely appears histologically in cerebral gumma lesions (Belloni and Ambrosetto, 1936; Sheps and Simon, 1943; Bagdasar, 1929).

Wassermann's reaction on serum and spinal fluid may, as already noted, be negative in the case of neurosyphilis (cf. for example Garcia, 1953; Nordmann, 1925; Martin, 1929). It may, and this has also been observed, be positive in various conditions of a general nature and, deceptively in so far as the problem under discussion is concerned, in endocranial neoplasia (Newmark, 1912; Moersch, 1928; Stern, 1919; Lotmar, 1921; Oppenheim, 1910; Marburg, 1910; Cl. Vincent).

In five cases described by Bagdasar (cerebral syphilitic gummas), Wassermann's reaction was twice positive and three times negative on serum, once negative and once positive on spinal fluid.

Aita (1964) reported that in the presence of cerebral syphilitic gummas, Wassermann's reaction was positive in 75% of the cases on serum, and in 50% of the cases on spinal fluid.

An angiographic study can only provide indirect elements for the diagnosis (cf. for example the cases described by Bianchi and Frera, Paillas *et al.*, 1950; Salles and Pecker), such as vascular displacement from an extraparenchymal mass that does not have its own vascular system (sometimes merely with an evanescent arterio-capillary capsular halo) and general arteritic symptoms.

Therapy

The treatment par excellence for cerebral syphilitic gumma is surgical excision. This, contrary to the inevitably inauspicious outcome of surgical

intervention in the case of tubercular granuloma, and its varying results in cases of cerebral neoplasia, is usually successful.

Opinions differ as to the efficacity of medico-chemiotherapeutic and antibiotic treatment.

Houston Merritt *et al.* and Horsley (1910) advise a "tentative" medical treatment limiting surgical intervention to cases in which the results achieved by the latter are unsatisfactory, or to those in which the symptoms of focal lesions and endocranial hypertensions attain alarming proportions.

Many authors, on the other hand, are against "tentative" medical treatment for an endocranial mass of an undefined character since it may have serious consequences of a general nature (Noethe, 1912; Morrison and McKinley, 1927; Bagdasar; Moersch), perhaps due to the colliquation of tissues in a pathological condition.

Recent observations (Salles and Pecker; Bianchi and Frera) are in favour of introducing medical treatment *after* the surgical removal of the granuloma.

Surgical intervention would not only (Bagdasar; Dufour, 1924) eliminate the cause of endocranial hypertension and local parenchymal compression, but also improve local circulation (affected by the mechanical compression and general arteritic complications) thereby rendering it more permeable to pharmacological impregnation.

On the other hand, it is not improbable that a large part of the efficiency of the post-operative medical treatment in these cases depends on a beneficial effect on the specific arteritis.

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