The Dejerines and Jumentié were the first to describe a patient with subacute necrotising myelopathy, before Foix and Alajouanine

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ABSTRACT

Introduction. In 1926, Foix and Alajouanine reported two cases of young men affected by subacute paraplegia caused by a necrotising lesion to the thoracolumbar spine associated with proliferation of abnormal hypertrophic blood vessels ("angio-hypertrophique"). In 1931, Lhermitte et al. published a similar case, coining the term Foix-Alajouanine syndrome, which is used today to refer to subacute necrotising myelopathy with hypertrophic vascular lesions.

Material and results. However, a review of the 1914 volume of *Revue Neurologique (Paris)* identified an earlier description of a patient with the same syndrome, reported by Joseph Jules Dejerine, Augusta Dejerine-Klumpke, and Joseph Jumentié, which has not been acknowledged in the literature.

Discussion. The main reason for this is probably that the report by the Dejerines and Jumentié focuses on neuroanatomical aspects of the case, with less focus on the pathological basis. Specifically, they emphasise clinical-pathological correlation and the secondary degeneration of the sensory tracts of the spinal cord, which were poorly understood at the time. Studying degeneration was the method on which they had based much of their excellent work *Anatomie des centres nerveux*.

KEYWORDS

Dejerine, Foix-Alajouanine syndrome, Jumentié, necrotising angiohypertrophic myelopathy, arteriovenous malformations, arteriovenous fistulae

Introduction

In July 1926, Foix and Alajouanine[1] published an article entitled "La myelite nécrotique subaigûe," reporting the cases of two men presenting subacute paraplegia associated with spinal cord necrosis. The most striking histological finding was the presence of a large amount of abnormal blood vessels with hypertrophic walls ("angio-hypertrophique"). Several years later, in 1931, Jean Lhermitte et al.[2] published another study of necrotic myelopathy with angiohypertrophic gliosis, using the eponym Foix-Alajouanine syndrome, the term that has since become established in the literature. Other early cases are cited in the study by Brion et al.[3]

However, a more thorough review of the journal *Revue Neurologique* identified a contribution from Joseph Jules Dejerine, Augusta Dejerine-Klumpke, and Joseph Jumentié,[4] published 12 years before the work by Foix and Alajouanine, describing a patient with the characteristic symptoms and neuropathological lesions of subacute necrotising myelopathy. The study has gone unnoticed

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and is not acknowledged in the subsequent literature, with the exception of a passing reference in Walusinski's[5] biography of Jumentié, which does not address the study in detail.

Material and methods

The article by the Dejerines and Jumentié[4] in *Revue Neurologique* was found coincidentally, during a review of the 1914-1915 volume for another reason. The other sources used in this study are listed in the references section. From a historical perspective, the essential contributions are those from Foix and Alajouanine,[1] Lhermitte et al.,[2] and Brion et al.[3] Comparison of the findings of Dejerine et al.[4] and the basic, clinical, and pathological elements enabling Foix and Alajouanine to characterise their syndrome (flaccid and spasmodic subacute paraplegia and spinal cord necrosis with vascular abnormalities) reveals great similarities. The patient described by Lhermitte et al.[2] displayed symptoms differing somewhat from those described by Foix and Alajouanine, and presented vascular lesions that enabled the authors to assimilate both observations. The study by Brion et al.[3] is relevant as it marks a historical turning point between the time in which necrotising myelopathy with vascular malformations was an exclusively neuropathological diagnosis, often not suspected based only on clinical signs, and the phase in which it began to be diagnosed in living patients with angiography studies, just a few years later.

Results

The article reviewed here is based on a communication to the Société de Neurologie de Paris on 25 June 1914, a month before the outbreak of the First World War.

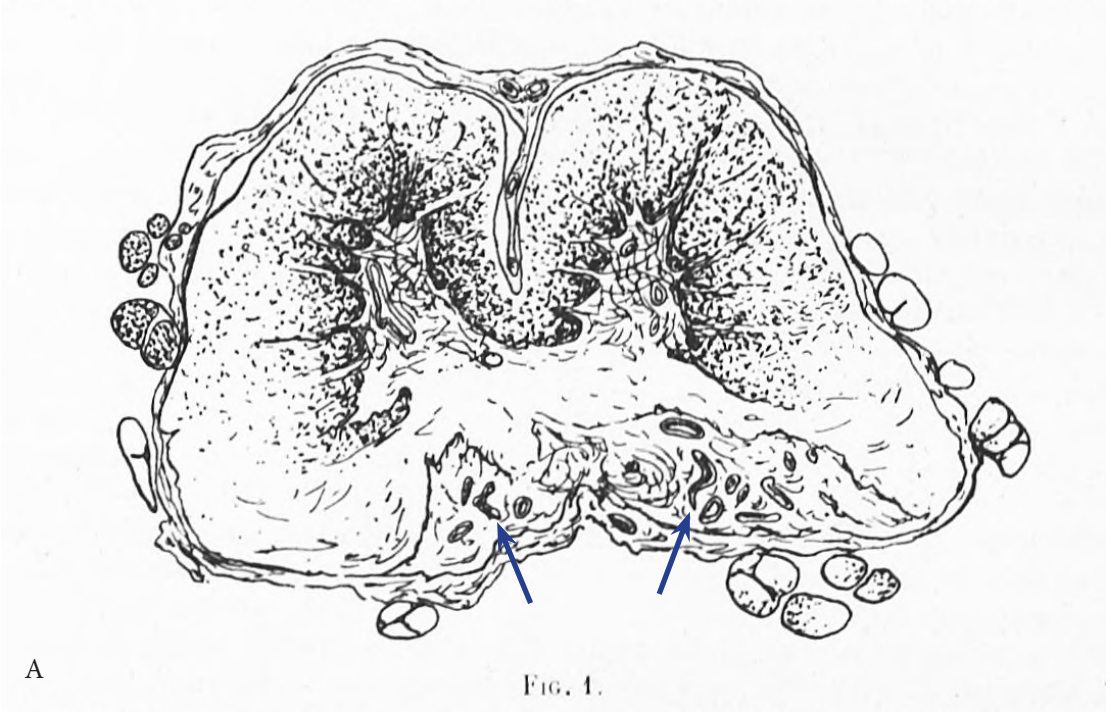
It begins with a description of the clinical case. The patient was a 22-year-old plumber who on 24 February 1910 was admitted to the Clinic for Nervous Diseases at the Salpêtrière due to paraplegia. He had no relevant personal history. Symptoms had begun in February 1909, during his military service, with pain and a pins-and-needles sensation in the feet; by 15 days, these symptoms ascended up both legs to the hip. Intensity of the discomfort oscillated, and generally worsened with effort. He was discharged from military service in July. At home, he developed progressive weakness, eventually losing the ability to walk. He was first admitted to Hôpital Beaujon, where physicians suggested a diagnosis of "double sciatica," and subsequently transferred to the Salpêtrière.

Physical examination of the head and arms identified no abnormalities. Paraplegia was not complete, and he was able to take a few steps with support. The motor deficit was predominantly distal and correlated well with abnormal electrical stimulation test results. With the exception of a somewhat pendular right foot, muscle tone was increased and patellar reflexes were intact. However, the Achilles reflex was abolished on the right side, but present on the left. The plantar reflex was clearly extensor in the left foot, and intermittently extensor in the right. The cremasteric and abdominal reflexes were weak. Pain and cramps persisted, particularly at night.

The finding that the authors most emphasise is the dissociated sensory loss: superficial sensitivity was only mildly affected, with mild hypoaesthesia to touch in the territory of the sacral and right L5 nerve roots, with errant and delayed perception. However, deep sensitivity was much more severely affected. Both positional and (vibratory) bone sensitivity were abolished below the knees. Examination of temperature and pain sensitivity in the right foot caused withdrawal/defence reflexes. The patient presented double incontinence. He later developed pressure ulcers, and died on 28 July 1911, after two-and-a-half years of progression.

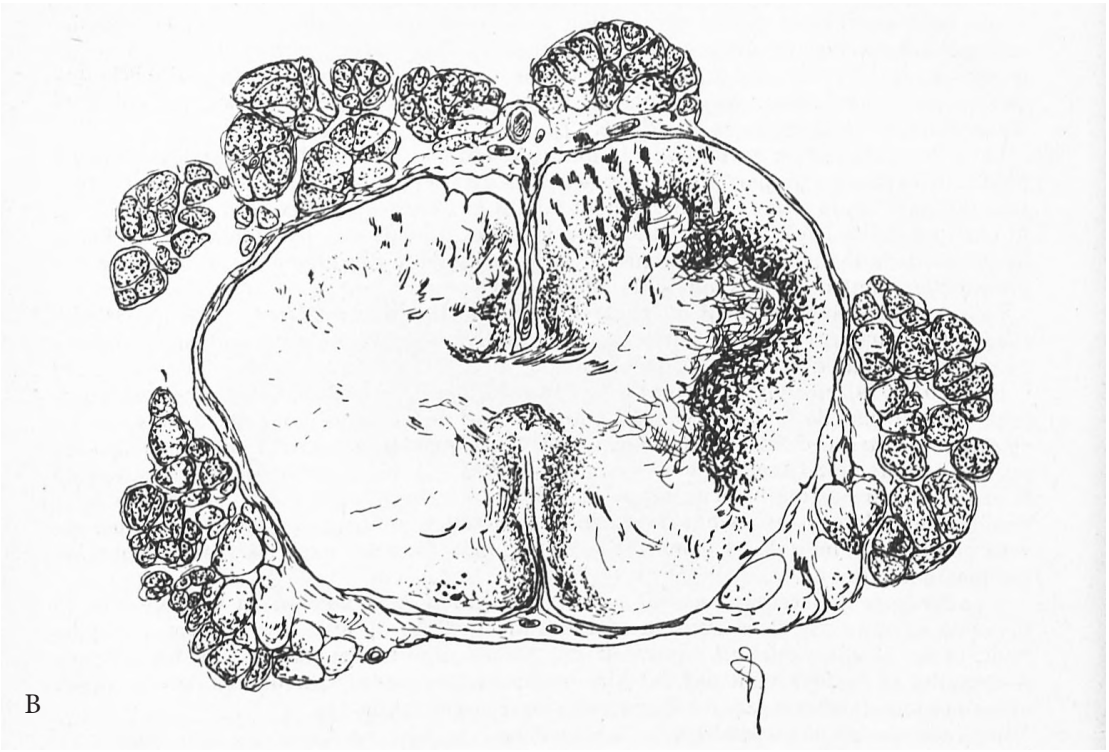
The article continues with a description of the neuropathology findings. The thoracolumbar spine was atrophic, and a section performed after fixing revealed a haemorrhagic appearance of the grey matter. Spinal specimens were fixed in potassium dichromate and processed in series. The authors also performed serial processing of the medulla oblongata and pons, to trace the ascending degeneration of nerve bundles from the spinal cord.

They identified two foci of "myelitis," one in the thoracic and the other in the sacral spinal cord. The thoracic lesion (Figure 1A) was at the T7 level, and predominantly affected the posterior columns, which presented destruction or voids ("évidés"), with tissue retraction and adherence of the thickened pia mater. Pal, carmine, and Van Gieson staining did not identify any nerve fibre. "There was no trace of nervous tissue. We observed blocks of amorphous tissue separated by numerous vessels with extremely thickened, infiltrated walls, some of which presented obliterating endarteritis." The lesion extended to the ventral portion of the spinal cord, but the anterior



A

Fig. 1.



B

Figure 1. Images from the article by the Dejerines and Jumentié[4]; in this view, the ventral horns and anterior columns are shown in the upper part of the image. A) Section from the thoracic lesion, showing complete destruction of the posterior columns. B) Section from the sacral segment, which was partially preserved above segments S3-S5, which were completely necrotic. Both images show silhouettes of the abnormal vessels, both intramedullary, in the posterior column (arrows in *A*), and extramedullary, mainly in the posterior column (both images). Source: *Revue Neurologique*, vol. 1 (1914). Available at: https://www.biusante.parisdescartes.fr/histoire/medica/resultats/index.php?do=chapitre&cote=130135x1914x01

horns were relatively well conserved. Both the lateral corticospinal tract and the dorsal spinocerebellar tract were degenerated in the lateral columns. The anterior columns were partially demyelinated, which the authors interpreted as secondary ascending degeneration.

The sacral focus of myelitis occupied the entire conus medullaris, completely destroying segments S3, S4, and S5. Above the S2 level, spinal cord morphology was partially preserved (Figure 1B). Nervous tissue could not be distinguished in the necrosed segments, which presented "thick intra- and extramedullary arterial lesions."

The final part of the work is dedicated to the study of "degenerations," ie, the impact of the focal spinal cord lesions on the ascending and descending nerve tracts. This section spans nearly three pages, with extraordinarily thorough morphological descriptions. Above the thoracic focus, the posterior columns presented degeneration of the gracile fasciculus (where lumbar and sacral fibres ascend) until its nucleus in the medulla oblongata. Degeneration also extended to the Gowers tract and Clarke nucleus.

In the thoracolumbar segment of the spine, between the two necrotic foci, degeneration was complex, simultaneously ascending and descending, and involving both pyramidal tracts, for example. The anterior column presented complete degeneration immediately above the sacral necrotic focus, which was displaced to the ventral edge of the column in upper segments. Degeneration in the posterior columns resulted from the destruction of both the fibres ascending from the sacral focus and the descending fibres (endogenous/propriospinal and radicular cervicothoracic fibres) sectioned at the thoracic focus of myelitis. The description of the distribution of posterior column degeneration spans nearly a page, is impeccably thorough, and is challenging even for the most experienced anatomists.

The article ends with several conclusions synthesising the clinical-pathological correlations and the study of secondary degeneration:

1. The authors attribute the combination of clinical signs and symptoms of spastic paraplegia with other signs related to peripheral nervous damage (sphincter paralysis, abolished right Achilles reflex, distal hypotonia in the right leg, and degeneration reactions to electrical stimulation of muscles innervated by the sacral nerve roots) to the presence of two foci of spinal cord damage, in the thoracic and sacral segments.

2. The dissociated sensory loss, with mild involvement of superficial sensitivity and abolition of deep sensitivity, predominantly in sacral territories, with relative conservation of lumbar territories, was related to the intense damage to the thoracic posterior columns. The authors considered that the long fibres of the sacral spine were interrupted twice (at both foci), whereas fibres from the lumbar segment were able to reorganise, ascending via the anterolateral columns, which were intact at the thoracic level.

3. The two foci of myelitis originated at different times, with the sacral injury being more recent.

4. The study of degeneration demonstrated the presence of a "long endogenous (propriospinal) ascending tract in the anterior column, mainly originating in the crossed fasciculus and extending to all levels of the spinal cord."

5. In the thoracolumbar segment between the two foci, the descending endogenous (propriospinal) and radicular fibres reached the centrum ovale by three different pathways: the peripheral Hoche tract, the disseminated tract described by Nageotte and Esslinger, and a large number of fibres from a previously undescribed, cornucommissural and septocommissural tract.

Discussion

In 1914, the Dejerines (Figure 2), who have been the subject of extensive biographies,[6,7] were at the height of their career. However, they had little time remaining, as Joseph Jules died of kidney disease in 1917. Furthermore, these final years were not the most glorious. With the outbreak of the First World War, the Salpêtrière was largely converted into a military hospital, and its neurological tradition was only recovered slowly.

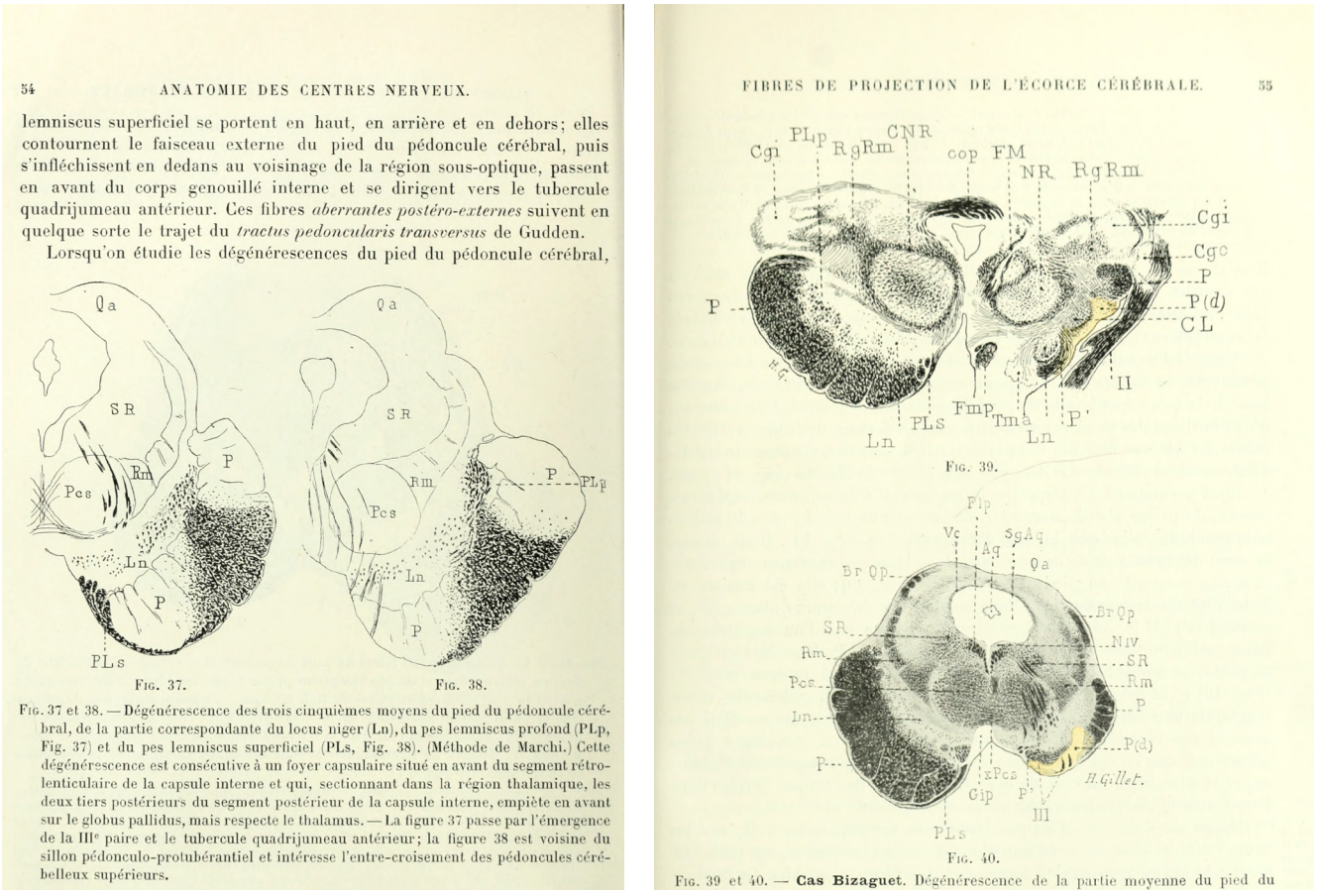
Joseph Jules had been appointed to Charcot's chair at the hospital in 1911. However, he was a disciple of Vulpian, and his selection for the chair was at the expense of Pierre Marie, a favourite student of Charcot's; thus, his appointment was highly controversial and exacerbated the old rivalry between the two men, which had dire consequences. Dejerine's death in 1917 led to the return of Pierre Marie, who liquidated and violently expelled all of his rival's followers and archives from the hospital, a truly regrettable decision.

Figure 2. Joseph Jules Dejerine and Augusta Dejerine-Klumpke in their latter years. Both hold in their hands preparations of large serial sections from the whole brains on which they based their extraordinary neuroanatomical and neuropathological oeuvre.[8] Source: Wikipedia, the free encyclopedia (Wikimedia Commons). Available at: https://en.wikipedia.org/wiki/Joseph_Jules_Dejerine

In turn, Joseph Jumentié was forgotten, until the neurologist's memory was recovered in a recent article by Walusinski.[5] He was an outstanding student of, among other great masters, Babinski and the Dejerines, with whom he had close ties. He published over 100 works, and had particular interest in certain types of brain tumour. However, his career was interrupted firstly by the First World War and secondly by the unsuccessful attempts to be selected as a clinical lecturer, which prevented him from projecting his ideas into academia and creating a school. He died suddenly at the young age of 49 years. Walusinski insists that Jumentié's support was highly important in the Dejerines' scientific contributions, due to his mastery of the pathological method of serial sections and in the study of secondary degeneration; the latter point is particularly relevant to the study discussed in this article.

The oeuvre of the Dejerines is truly vast, reaching great heights both in the clinical setting and in the fields of neuropathology and neuroanatomy. In 1914, they published the first edition of their nearly unsurpassable *Semiologie des affections du système nerveux*,[8] which was followed by several posthumous editions, and was studied by generations of neurologists worldwide. In the field of neuroanatomy, one of their greatest works is *Anatomie des centres nerveux*,[9] dedicated to Dejerine's master Vulpian.

Dejerine made it clear in the introduction to this treatise that "L'anatomie du système nerveux central est avant tout une anatomie de texture," with "texture" referring not to consistency, but to structure. He continued, "our aim should be to follow the fascicles that comprise it, to establish their origin and termination." To that end, he highlighted the two methodological tools on which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lemniscus superficiel se portent en haut, en arrière et en dehors; elles contournent le faisceau externe du pied du pédoncule cérébral, puis s'infléchissent en dedans au voisinage de la région sous-optique, passent en avant du corps genouillé interne et se dirigent vers le tubercule quadrijumeau antérieur. Ces fibres *aberrantes postéro-externes* suivent en quelque sorte le trajet du *tractus pedoncularis transversus* de Gudden.

Lorsqu'on étudie les dégénérescences du pied du pédoncule cérébral,

Fig. 37. Fig. 38.

Fig. 37 et 38. — Dégénérescence des trois cinquièmes moyens du pied du pédoncule cérébral, de la partie correspondante du locus niger (Ln), du pes lemniscus profond (PLp, Fig. 37) et du pes lemniscus superficiel (PLs, Fig. 38). (Méthode de Marchi.) Cette dégénérescence est consécutive à un foyer capsulaire situé en avant du segment rétro-lenticulaire de la capsule interne et qui, sectionnant dans la région thalamique, les deux tiers postérieurs du segment postérieur de la capsule interne, empiète en avant sur le globus pallidus, mais respecte le thalamus. — La figure 37 passe par l'émergence de la III[e] paire et le tubercule quadrijumeau antérieur; la figure 38 est voisine du sillon pédonculo-protubérantiel et intéresse l'entre-croisement des pédoncules cérébelleux supérieurs.

FIBRES DE PROJECTION DE L'ÉCORCE CÉRÉBRALE. 55

Fig. 39.

Fig. 40.

Fig. 39 et 40. — **Cas Bizaguet.** Dégénérescence de la partie moyenne du pied du

Figure 3. Example of the Dejerines' methodology for studying the tracts of the central nervous system, based on the degeneration caused by focal lesions (taken from *Anatomie des centres nerveux*[8]). Left: degenerated tracts (black dots) of the pes pedunculi in the substantia nigra, the pes lemnisci profundus, and the superficial pes lemniscus, secondary to a lesion to the posterior limb of the internal capsule (Marchi staining). Right: massive descending degeneration of the pes pedunculi (myelin staining).

his method was based: *a)* serial processing of whole brains, both healthy and diseased (at great cost in terms of materials and laboratory technicians' time), and *b)* following lesion-induced degeneration to trace the afferent and efferent connection pathways of different structures (Figure 3), a similarly immense task for the neuroanatomist. This method enabled him to go further in his study of distant connections than was possible with Golgi staining of small sections from embryos or young animals, which provided information on the independence of neurons (he paid homage to Ramón y Cajal) and the internal structure of each nucleus and its local, but not distant, connections. Many decades later, the introduction of markers of anterograde and retrograde axonal transport enabled great advances in the hodology of the brain.

The Dejerines' and Jumentié's[4] interest in neuroanatomy is clear in this study of subacute necrotising myelopathy, as a significant part of the text describes not the primary lesions but the secondary degeneration, attaining a level of detail that is beyond the reach of all but the most experienced anatomists. The study's fundamentally anatomical approach probably explains why neither the authors nor subsequent researchers took notice of the characteristics of the lesions underlying the patient's paraplegia, contributing to the article having been overlooked. The authors' interest in neuroanatomy is also reflected in their analysis of the patient's dissociated

sensory loss and their correlation with the lesions, as the controversy regarding the path of sensory tracts through the spinal cord was not fully resolved at that time.

Another clinical detail they highlight is the copresence of pyramidal and lower motor tract lesions: in addition to signs of "pyramidal" spasticity (hypertonia, exaggerated patellar reflex, Babinski sign), the patient presented such other signs as hypotonia, abolished Achilles reflex, and mild distal amyotrophy in the right leg. Later, Foix and Alajouanine[1] considered this combination of symptoms, changing over time, to be so revealing that they highlighted it in the title of their work, indicating that the paraplegia was initially spasmodic and subsequently flaccid, with amyotrophy. However, not all cases present this combination of symptoms. For instance, the patient reported by Lhermitte et al.[2] presented spastic paraparesis that did not progress to the flaccid form until the final stage, after a failed surgical intervention. However, this order of appearance of symptoms is not a necessary condition: signs of the "peripheral" lesion with segmentary paresis, hypotonia, or amyotrophy may be pronounced initially, or predominant over pyramidal signs, which would account for misdiagnoses. For example, the patient reported by the Dejerines and Jumentié was diagnosed with "double sciatica" at the first hospital he visited. The same has occurred in some of my own patients, and a recent article drew attention to this possible misdiagnosis.[10]

Regarding the lesion in the patient reported by the Dejerines and Jumentié,[4] we cannot establish a conclusion on what type of arteriovenous malformation or fistula was the underlying cause.[11,12] It was clearly a vascular pathology, given the presence in the spinal cord of "numerous vessels with extremely thickened, infiltrated walls, some of which presented obliterating endarteritis" at the level of the thoracic lesion and "thick intra- and extramedullary arterial lesions" at the sacral focus. In all likelihood, these were veins with arterialised walls due to hypertension. The existence of two lesion foci rules out a purely intramedullary malformation, as well as a selective malformation at the conus medullaris. The lesion was probably an intradural or epidural fistula.[11,12] Given the authors' extraordinary prior experience in the field of neuropathology, it is very striking that, while they dedicate several pages to describing the secondary degeneration, Dejerine et al.[4] limit themselves to describing the vascular lesions without offering a single comment on their possible nature. The opposite is the case in the articles by Foix and Alajouanine[1] and by Lhermitte et al.,[2] in which the bulk of the discussion focuses on the vascular lesions. Foix and Alajouanine[1] suggest a possible inflammatory aetiology, hence their use of the term myelitis. Lhermitte et al.[2] rule out this hypothesis and are more inclined towards a proliferative gliotic process ("gliose angéio-hypertrophique").

Foix and Alajouanine were extraordinary neurologists worthy of being remembered for their numerous great contributions; however, their link with the first description of necrotic myelopathy secondary to an arteriovenous malformation or fistula is a historical error, if we conclude that the Dejerines and Jumentié merit this attribution. It is also somewhat ironic that the eponym Foix-Alajouanine syndrome was coined by Lhermitte et al.,[2] who disregarded the article by their masters, the Dejerines, especially considering that Lhermitte worked at the Dejerine Foundation, created to defend their heritage after their violent expulsion from the Salpêtrière due to Pierre Marie's undisguised bitterness.

Independently of eponyms, subacute necrotising myelopathy continues to be a relevant topic today. Its pathogenesis was debated for decades, as autopsy studies were unable to identify the origin of the abnormal vessels, whether the lesion was congenital or acquired, or whether it was of inflammatory or other aetiology. Contrast myelography enabled in vivo study of some patients with subacute myelopathies, revealing the silhouette of the tortuous, dilated vessels described in post mortem studies.[13] However, this advance contributed little to diagnosis: as late as 1952, spinal arteriovenous malformations or fistulae were rarely suspected, and only discovered in post mortem examination.[3] Thanks to the introduction of spinal angiography by selective catheterisation, it became possible to observe the relationship between subacute myelopathy and arteriovenous malformations or fistulae,[14-17] paving the way for greater clinical understanding of these lesions and approaches to their endovascular or surgical treatment.[18-24] Both treatments are efficacious, and multidisciplinary collaboration will ideally enable selection of the most appropriate treatment, or combination of the two. Early diagnosis is essential to successful treatment. Magnetic resonance imaging findings of extensive centromedullary hyperintensity, with some meningeal or intramedullary contrast uptake and perimedullary points of signal void, corresponding to dilated veins, should lead to the decision to perform the

definitive angiography study. Poor treatment outcomes are largely explained by late or incorrect diagnosis, when the spinal damage has become excessive.[24,25]

Conflicts of interest

The author has no conflicts of interest to declare.

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