# Nicolás Achúcarro's 1905 communication to the Société Française de Neurologie

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**ABSTRACT**

Nicolás Achúcarro had the opportunity to travel and train in several neurology and psychiatry centres around Europe. The first of these experiences was in Pierre Marie's service at the Hospice de Bicêtre. According to his letters and biographies, he presented a communication to the Société Française de Neurologie at the end of his time working under Pierre Marie; however, its content was not known. The communication has now been found, and is reported in this article. In the communication, Achúcarro presents a patient with syringomyelia, which he considered to be of traumatic origin; however, given current understanding of the disease and Achúcarro's autopsy findings, it is likely that aetiology was non-traumatic, with trauma simply triggering the symptoms.

**KEYWORDS**

Achúcarro, hydrocephalus, paraplegia, Pierre Marie, spinal cord injury, syringomyelia

## Introduction

Nicolás Achúcarro has been the subject of several biographies[1] and numerous articles.[2,3] In fact, this journal published a short article summarising his life and his most important scientific work.[4]

Achúcarro had the good fortune to be born into a comfortable, forward-thinking family. His father was a prominent ophthalmologist and his mother was born into a family of Norwegian merchants who settled in Bilbao. The atmosphere of Achúcarro's childhood home was one of enlightenment: the family were lovers of music, travel, and languages; this was further reinforced in Nicolás when he was sent as a teenager to study in Germany, where he learnt the language.

Shortly after starting his degree in medicine in Madrid, he found himself disappointed with the poor intellectual and cultural level of the faculty (despite Cajal being the university's chair of histology) and decided to continue his studies in Germany. In Madrid, he benefited from private tuition from Luis Simarro, who gave lessons on his own initiative, introducing Achúcarro to the field of neurohistology. By the time he completed his degree, Achúcarro was a proficient speaker of German and French, and so decided to pursue further training in Europe, beginning in Pierre Marie's neurology department at the Hospice de Bicêtre (Figures 1 and 2), where brilliant figures including Bourneville, Charles Foix, and Dejerine had previously stood out.

Letters written by Achúcarro himself, collected in Vitoria's biography,[1] provide direct information from his time at the Bicêtre and other Parisian centres, beginning in 1905. Laín Entralgo's[5] article on Achúcarro erroneously notes that Pierre Marie was at the Salpêtrière that year, when in fact he had left the hospital in 1897. It was not until the death of his great rival Dejerine in

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**Figure 1.** Pierre Marie with his collaborators, including Nicolás Achúcarro (fine arrow) and his Italian friend Catola (bold arrow)

1917 that Marie was awarded Charcot's old chair. Laín Entralgo may be the original source of this error, which is reproduced by other authors including Vitoria.[1]

According to Achúcarro's letters to his mother,[1] his instructor Pierre Marie encouraged him to prepare a communication to present to the Société Française de Neurologie (SFN) in May 1905, but it was never presented due to a lack of space in the programme. According to Vitoria,[1] Achúcarro did not feel settled at Bicêtre, perhaps because his main interest was psychiatry (mental illness and its neuropathological basis) rather than neurology, or perhaps due to the lack of a systematic research programme that suited his scientific mentality.

The other two foreign assistants working under Pierre Marie at the Bicêtre, Catola and Lewandosky, invited Achúcarro to visit their respective countries, Italy and Germany. With this idea in mind, and led by his adventurous spirit, Achúcarro informed his mother in his last letter from Paris that he would return to Bilbao in late June. Vitoria[1] cites a secondary source stating that it was Pierre Marie who presented the communication to the SFN, in July 1905. Laín Entralgo[5] also mentions the hypothesis that Pierre Marie presented the communication in July because Achúcarro had returned to Bilbao in June. However, the official programme of the SFN[6] lists the communication in its June session; similarly, Pierre Marie's[7] collection *Titres et travaux* notes that the communication was presented on 7 June. Therefore, Achúcarro may have presented the communication not in May, but in June, prior to his planned return to Bilbao.

In any case, after leaving Paris, Achúcarro undertook his planned European tour, first staying briefly in Florence with Lugaro and Tanzi, then staying until September 1908 in Munich. There, under the direction of Kraepelin, and especially under Alzheimer, his true calling as a

**Figure 2.** Main entrance to the Bicêtre. Photograph taken by the author in 1971. The white marks at the lower part of the façade were caused by the cleaning of graffiti from the May 1968 protests. The neurology department was based in the wing named after Pierre Marie and was subsequently renamed for Pierre Lasjaunias, the pioneer of interventional neuroradiology who began his career at the hospital. Prof. Jean Lepresle's neuropathology laboratory was the same space that had been used by Charles Foix; its windows overlook the "Sibérie" courtyard.

neuropsychiatrist and neurohistologist was consolidated. Vitoria[1] indicates that he was unable to find any information on the content of Achúcarro's communication to the SFN. Laín Entralgo[5] was even more explicit, writing in a footnote in his article that "the text of this communication (what was it about?) seems to have been lost." However, the communication has now been found, in the proceedings of the SFN (Figure 3).[6]

The objective of this article is to report the discovery of Achúcarro's communication to the SFN,[6] in which he presents clinical and autopsy findings from a patient with post-traumatic syringomyelia, and to discuss his conclusions in the light of current understanding of this disease.

## Material and methods

Basic and general information was taken from the sources cited in the bibliography. The text of Achúcarro's communication to the SFN was found in the digital archives of *Revue Neurologique* (Paris), which are conserved in the inter-university library of Paris Descartes University (http://www.biusante.parisdescartes.fr/histmed/medica/cote).

## Results

The complete text of Achúcarro's[6] communication is as follows:

> We present a patient with post-traumatic syringomyelia with hydrocephalus.
>
> At the age of 12, the patient suffered major trauma after a one-storey fall from a ladder. When he regained consciousness, he was impotent in all four limbs, but after three weeks his condition had improved to the point that he was able to walk and to eat unassisted. He was unable to return to work and his disorders continued to progress gradually.
>
> During examination at the Bicêtre infirmary in 1904, he was impotent in all four limbs and presented

VIII. **Syringomyélie traumatique avec Hydrocéphalie**, par M. N. Achucarro (Bilbao).

(Travail du laboratoire de M. Pierre Marie.)

Il s'agit d'un cas de syringomyélie traumatique avec hydrocéphalie.

Notre sujet subit à l'âge de 12 ans un grand traumatisme; il tomba, en descendant un escalier, de la hauteur d'un étage. Quand il reprit connaissance il était impotent des quatre membres, mais au bout de trois semaines son état s'améliora au point de lui permettre de manger seul et de recommencer à marcher. Il ne put plus reprendre son travail et à partir de ce temps-là ses troubles suivirent une marche progressive.

Quand on l'examina à l'infirmerie de Bicêtre, en 1904, il était impotent des quatre membres, il présentait une scoliose très accentuée, atrophie musculaire aux membres, déformation de la main en pince et dissociation de la sensibilité. On diagnostiqua une syringomyélie. Il meurt, en 1904, à 56 ans à la suite d'une eschare trochantérienne.

La moelle présente une cavité unique s'étendant en longueur depuis les premiers segments cervicaux jusqu'aux derniers dorsaux. Elle siège à la région de la commissure grise et du canal central empiétant sur la substance grise des cornes. Très évidente à la région cervicale et dans les derniers segments dorsaux, elle est presque linéaire au niveau des premiers segments dorsaux. Sur les coupes faites à différentes hauteurs on voit que la cavité est en partie tapissée par des cellules épendymaires, formant une couche unique.

Une couche de gliose entoure la cavité et s'étend plus ou moins sur la substance grise et blanche. L'aspect histologique de la lésion est intermédiaire entre une hydromyélie et une syringomyelia gliosa.

Dans le cerveau, les ventricules latéraux sont considérablement dilatés. Leur capacité est de 73 cm³ pour le ventricule droit et de 65 cm³ pour le ventricule gauche. Le corps calleux est aplati.

Le troisième ventricule et les trous de Monro sont très dilatés.

La surface ventriculaire est ridée et présente en outre de petites granulations surtout visibles au niveau du prolongement occipital. Les coupes microscopiques montrent que la cavité est revêtue en partie par une rangée unique de cellules épendymaires manquant au niveau des granulations. Celles-ci sont formées d'une prolifération de la névroglie épendymaire (épendymite granuleuse).

Ce cas nous a paru intéressant, en premier lieu, au point de vue du rôle étiologique du traumatisme.

En effet, ce rôle est indéniable, étant donné que les accidents se sont installés immédiatement après la chute, et que, après un certain temps d'arrêt, ils ont suivi une marche progressive.

La seconde particularité intéressante de notre cas est la coexistence, avec la lésion médullaire, d'une hydrocéphalie avec épendymite granuleuse des ventricules latéraux, que l'on peut ramener à la même cause que les lésions médullaires, et nous serions portés à croire que le traumatisme a provoqué la syringomyélie et l'hydrocéphalie.

**Figure 3.** Reproduction of the text of Achúcarro's communication to the Société Française de Neurologie in 1905

very pronounced scoliosis, muscle atrophy in all four limbs, claw hand deformity, and dissociation of sensitivity. The patient was diagnosed with syringomyelia. He died in 1904 at the age of 56 as a result of a trochanteric pressure ulcer. The spinal cord presents a single cavity extending across all cervical and thoracic segments. The cavity is located in the area of the grey commissure and the central canal, extending to the grey matter of the horns. It is very evident in the cervical and lower thoracic regions, and almost linear in the upper dorsal area. Sections taken from different levels show that the cavity is partially lined with a single layer of ependymal cells. The cavity is surrounded by a layer of gliosis extending to different degrees through the grey and white matter. The histological appearance of the lesion lies between that of hydromyelia and that of gliotic syringomyelia.

Considerable dilation of the brain ventricles is observed. The right ventricle has a capacity of 73 $cm^3$ and the left a capacity of 65 $cm^3$. The corpus callosum is flattened.

The third ventricle and foramina of Monro are very dilated.

The surface of the ventricles is wrinkled and presents small granulations, particularly in the occipital prolongation (horn). Microscopic sections show that the cavity is partially covered by a single line of ependymal cells, which are not present over the granulations. These are formed by a proliferation of ependymal neuroglia (ependymitis granularis).

We considered this to be an interesting case, above all from the perspective of the aetiological role of trauma.

Essentially, the role of trauma is undeniable, given that symptoms manifested immediately after the fall and that, after a period of no change, it continued to progress gradually.

The second interesting detail of the case is the co-presence of the spinal injury and hydrocephalus with ependymitis granularis of the lateral ventricles, which we may attribute to the same cause as the spinal lesions: we are inclined to believe that both the syringomyelia and the hydrocephalus are of traumatic origin.

## Discussion

The communication describes a disabled patient who was admitted to hospital at the age of 56, in 1904, and died shortly thereafter (the same year) due to complications of a decubitus ulcer; at the time, this was a frequent and severe complication in patients permanently confined to bed.

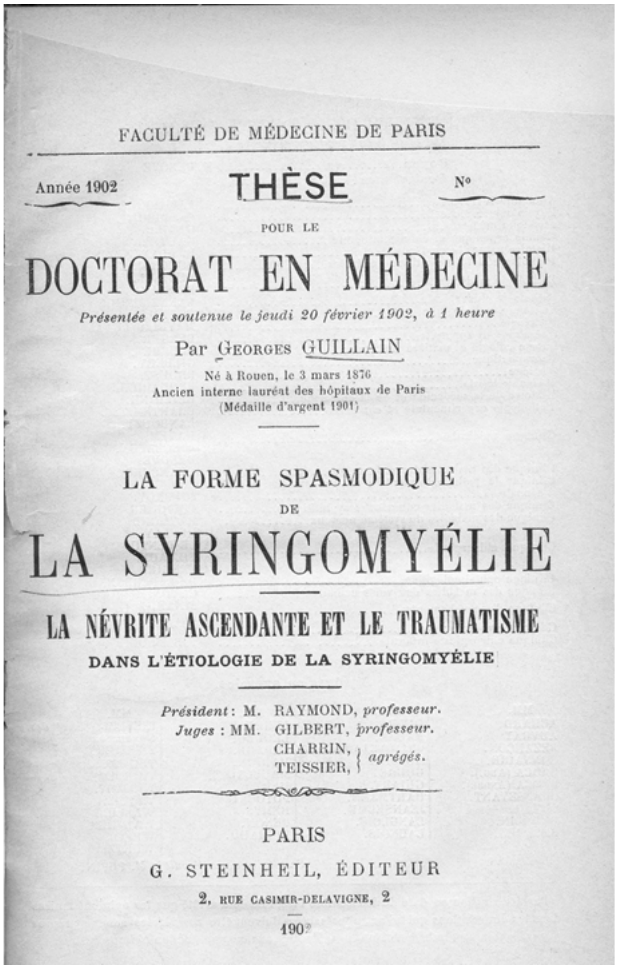
Pierre Marie generously permitted his young, foreign assistant to present the case alone in a communication to the SFN. This gesture was not unique: I have found numerous other communications by other visiting physicians at Marie's service that were also signed by a single author. This may confirm the opinions of Pierre Marie's disciples and biographers about his personality. All these voices agree that he was reserved, a man of few words and a disciplinarian who did not easily change his opinions; he was strongly confrontational but was both demanding and fair with his disciples.[8]

Taking into account the high caseload at the Bicêtre, a hospice for chronic and disabled patients where many were hospitalised until their deaths and subsequently underwent autopsy studies, we may ask why Pierre Marie selected this case and assigned it to Achúcarro for him to present. It is possible that post-traumatic syringomyelia was a subject of particular interest at the time, as it had been the subject of Guillain's[9] doctoral thesis several years earlier (Figure 3); Charcot's first successor, Fulgence Raymond, chaired the committee.

Achúcarro's clinical summary of the case is succinct. The patient was tetraplegic, with diffuse muscle atrophy, "claw hand" (the first three fingers extended and the remaining two flexed), dissociation of sensitivity, and highly pronounced scoliosis. Claw hand, semi-flexion of the arms, adduction of the shoulders, and barrel chest were the key semiological features in what Pierre Marie referred to as the "spasmodic" form of syringomyelia, which is described in detail in Guillain's[9] thesis. Pierre Marie could have used the same terms as Charcot to describe himself: "*Je suis un visuel*." To a great extent, semiology was based on visual observation.

Clinical diagnosis was confirmed by the autopsy findings. Up to this point, the case would be of very little interest. Encouraged by Pierre Marie, Achúcarro would have decided to present the communication to the SFN because he considered the cause to have been the trauma the patient suffered at the age of 12, and because of the associated hydrocephalus.

The proposed causal relationship between trauma and syringomyelia in this patient is based exclusively on the past trauma from his fall, with immediate onset of tetraplegia. However, this association is subject to debate, as the autopsy did not find any truly post-traumatic lesions in the spinal cord or column. The description of the histology study of spinal sections from various levels is succinct, but sufficient to infer that the study performed was thorough. However, no blood residue was found either on the meninges or inside the cyst; nor were there any arachnoid adhesions or foci of contusion in the spinal cord.



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LA FORME SPASMODIQUE
DE
LA SYRINGOMYÉLIE

LA NÉVRITE ASCENDANTE ET LE TRAUMATISME
DANS L'ÉTIOLOGIE DE LA SYRINGOMYÉLIE

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190?

**Figure 4.** Title page of Georges Guillain's doctoral thesis on post-traumatic syringomyelia

According to the description of the case, the boy was left "impotent" after the fall but subsequently recovered sufficiently to eat and walk. This progression is compatible with spinal contusion, with or without haematomyelia: after an initial spinal shock resulting in loss of spinal function, a gradual, partial improvement of symptoms is observed. However, the neurological symptoms progressively worsened from that time.

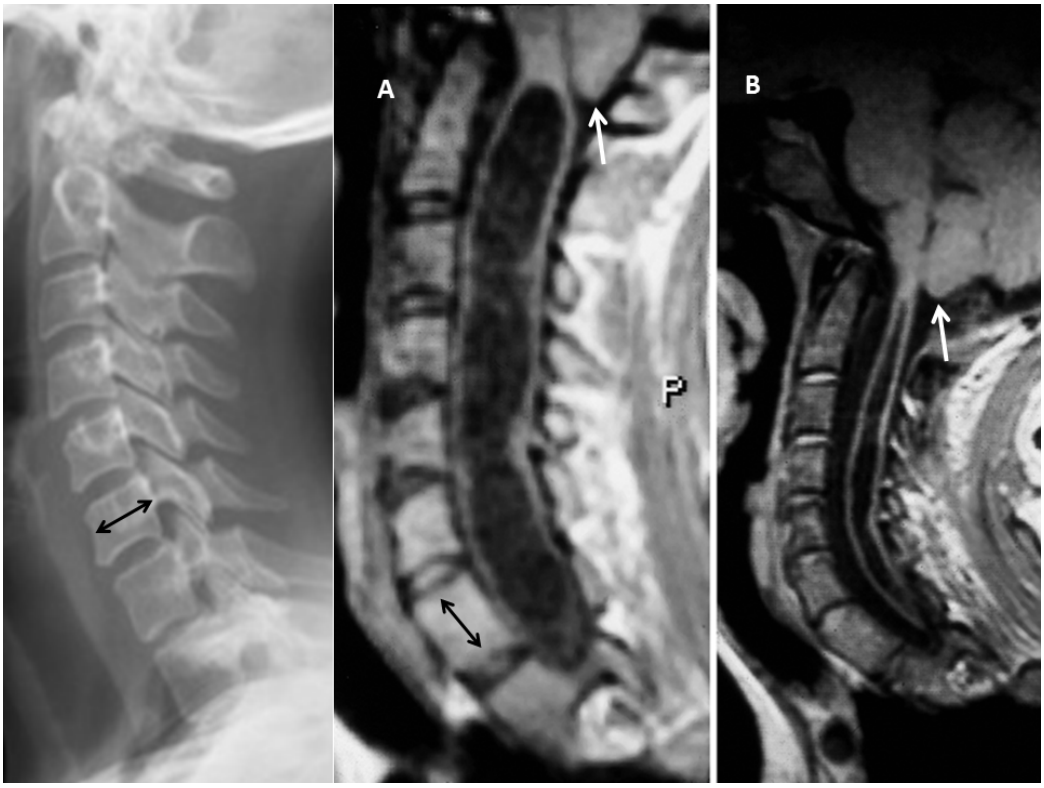


**Figure 5.** Radiography of the spinal column from a healthy control and neck MRI from a patient attended by the author. Several weeks after a minor cervical spine injury (whiplash injury), the patient consulted due to weakness and lack of sensitivity in one hand. The control radiography shows the typical rectangular shape of the vertebral bodies, with the horizontal diameter being greater. A) MRI from the patient showing descent of the cerebellar tonsil and marked syringomyelia. Progression time is long, considering the significant dilation of the cervical spinal cord and the remodelling of the vertebral bodies, with the vertical diameter being greater (double arrow). B) Post-surgical MRI. After decompression of the posterior fossa, the cisterna magna is enlarged, the syrinx collapses, and the length of the cerebellar tonsil is reduced (white arrow).

We may consider two hypotheses: firstly, that the patient had pre-existing, completely asymptomatic syringomyelia, which was decompensated and revealed by the trauma; and secondly, that the trauma was the true cause of the syringomyelia.

Alternative 1: trauma revealed pre-existing syringomyelia

This hypothesis was suggested many years ago,[10,11] and has been corroborated by experience. I have myself observed cases of syringomyelia after childbirth or minor neck trauma, in which imaging findings (Figure 5) clearly suggest a long progression time, with the trauma simply having decompensated and clinically revealed the condition. Evidently, the lack of radiology findings in Achúcarro's case represents a limitation for its proper interpretation.

If the hypothesis that pre-existing syringomyelia was decompensated by trauma is correct, then the aetiology must be some other, non-traumatic cause. Unquestionably, the most frequent is type I Chiari malformation.[12] This association had already been described when Achúcarro presented the communication, but was probably not well known.

Both syringomyelia and type I Chiari malformation, and particularly the latter, were difficult to identify with lipiodol myelography, which requires transport of contrast with the patient placed supine in the Trendelenburg position; therefore, diagnosis of these conditions was infrequent until the development of gas myelography and subsequently metrizamide CT myelography and magnetic resonance imaging (MRI).[13,14] It is not implausible that a type I Chiari malformation

may go unnoticed in an autopsy study not targeted to identify this entity.

Two significant details of the case reported by Achúcarro support the hypothesis of a type I Chiari malformation: presence of hydrocephalus and pronounced scoliosis. Hydrocephalus is not described in any of the numerous studies addressing post-traumatic syringomyelia.[11,15-25] Neither is scoliosis described in these patients, although kyphosis and deformities due to vertebral fractures have been reported. On the contrary, both hydrocephalus and scoliosis are frequent in patients with syringomyelia associated with type I Chiari malformation.

Alternative 2: syringomyelia was truly post-traumatic

The prevalence of post-traumatic syringomyelia is difficult to establish. In the follow-up of patients at centres specialising in spinal injuries, its frequency is estimated at 3.2% in one series,[11] whereas another series reports a rate of 3.4% in patients with complete paraplegia and 2.3% in those with incomplete paraplegia, in whom the time to symptom onset was greater.[19] Other authors also report higher rates of post-traumatic syringomyelia in patients with complete spinal cord lesions.[24] These series probably only include the most severe, symptomatic cases. In fact, long-term MRI follow-up of all patients with spinal cord trauma detects syringes in 21%-28% of patients at 30 years of follow-up, although many patients are asymptomatic.

The most characteristic form of post-traumatic syringomyelia is caused by a focus of compression due to the spinal fracture and deformation; in these cases, the syrinx may ascend, descend, or extend in both directions (Figure 6). The pathogenesis of these cysts is related to the blockage of cerebrospinal fluid (CSF) flow at the focus of compression, causing the fluid to enter the spinal cord through the perivascular spaces. This is caused by pumping and aspiration mechanisms during changes of pressure in the subarachnoid space (eg, cardiac systole, Valsalva manoeuvre).[22,23,26,27] Changes in pressure are transmitted to the interior of the spinal cord. High pressure in the cyst may explain the rostral MRI hyperintensity observed in the spinal cord and medulla oblongata in some patients due to transudation of CSF from the cyst, which is reversible with surgical treatment.[28]

It is undisputed that severe trauma with vertebral fracture and compression of the spinal cord is not a necessary condition for secondary syringomyelia. For example, mild to moderate trauma without vertebral fracture is known to cause a focus of haematomyelia or spinal cord contusion; symptoms are initially reversible but subsequently progress. This lesion focus is reabsorbed, leaving a small cavity that may subsequently grow due to the same CSF pumping and aspiration mechanisms mentioned above. This progression can be caused by arachnoiditis surrounding the focus of contusion, which limits the circulation of CSF and promotes its passage into the cyst. In fact, varying degrees of arachnoid adherences and thickening are the main morphological

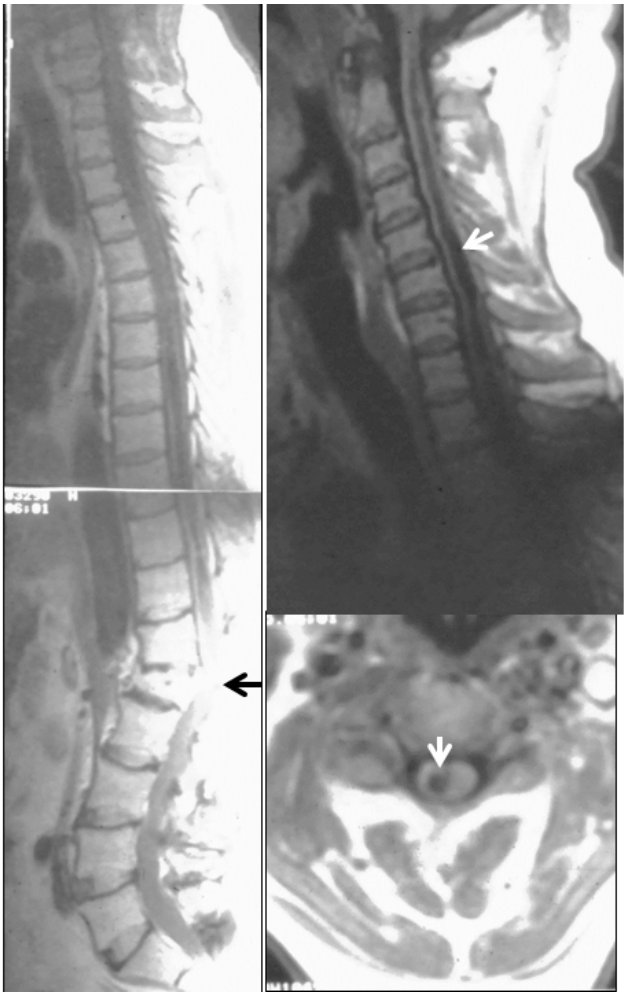

**Figure 6.** Post-traumatic syringomyelia. Several years after an accident leading to incomplete paraplegia, the patient began to notice loss of strength and sensitivity in the hands. MRI detected a fracture of the L1 vertebra, with compression of the conus medullaris (black arrow), and the syrinx, which ascends to the level of C1 (white arrows).

findings described by neurosurgeons operating on patients presenting post-traumatic syringomyelia without fractures/spinal cord compression; surgical outcomes are uncertain and inconsistent.[17,21,26,29-31] However, it seems unlikely that the lesions observed during surgery would go unnoticed in the macroscopic examination of the spinal cord during the autopsy study of Achúcarro's patient, or that they would not be noted in the histological description presented.

The French neuropathological tradition distinguished between syringomyelia, pseudosyringomyelia, and hydromyelia.[32] Pseudosyringomyelia referred to residual cysts secondary either to the reabsorption of old haemorrhagic or necrotic foci or to cystic tumours (eg, haemangioblastoma). Syringomyelia was characterised by loss of the ependymal wall of the cyst and formation of gliosis of the walls, extending in different directions. Hydromyelia was defined as simple dilation of the spinal canal, with the ependyma remaining intact. After reporting the histology findings, Achúcarro concludes that the patient presented an intermediate condition between hydromyelia and syringomyelia. This is the most frequent observation in patients with syringomyelia associated with type I Chiari malformation, in which the ependyma remains intact in some spinal cord segments but is fragmented, with a marked glial reaction, in others. The same phenomenon constitutes the basis of the ependymitis granularis described in the ventricular walls in Achúcarro's patient. Distension of the ventricular walls gives rise to rupture of the ependyma and the formation of small granulations due to the astrocytic reaction. Ependymitis granularis is observed in patients with hydrocephalus of all aetiologies, not necessarily only infectious or post-traumatic hydrocephalus.

In conclusion, retrospective analysis of the case that Achúcarro presented to the SFN in 1905 as post-traumatic syringomyelia raises questions about the aetiology, given the lack of supporting data from the neuropathology study, which did not identify scarring, arachnoiditis, or foci of contusion. As an alternative interpretation, syringomyelia may have been associated with undiagnosed type I Chiari malformation. This hypothesis is also supported by the association with scoliosis and hydrocephalus. Finally, the trauma may have revealed underlying syringomyelia, and may have contributed to its progression, but probably was not the cause.

**Conflicts of interest**

The author has no conflicts of interest to declare. This study has received no public or private funding.

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