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Post-traumatic syringomyelia and post-traumatic spinal canal stenosis: A direct relationship: Review of 75 patients with a spinal cord injury

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This study aims to demonstrate predictive factors for post traumatic syringomyelia (PTS), and in particular to correlate the role of insufficiency of reduction of a spinal fracture with the occurrence of syringomyelia. One hundred and twenty-eight spinal cord injured patients (SCI) were studied during the years 1992 and 1993. Among them, 75 underwent a complete and reliable evaluation including: review of the initial vertebral lesion, and of the surgery report, and a radiological study of the lesion site with standard X-rays, a CT scan, and an MRI. The CT Scan included slices in sagittal reconstructions and in the axial plane at the site of injury with the calculation of a percentage of canal stenosis in the two planes of the space. An MRI was carried out with T1 and T2 weighted images, including sagittal entire cord images in addition to sagittal and axial slices centred on the site of injury. A syrinx was diagnosed in 28% of the patients. The occurrence of a syrinx is significantly correlated with spinal canal stenosis in the sagittal plane (ΔD) with a $P<0.001$ and in the axial plane (ΔS) ($P<0.05$). This present study demonstrates the major role of the insufficiency of reduction of the vertebral lesion in the genesis of a syrinx. The quality of the initial treatment of the vertebral injury is the first step in the prevention of a syrinx. The treatment of a syrinx, besides techniques of drainage, must also take into account the spinal realignment.

Keywords: spinal cord injuries; post-traumatic syringomyelia; post-traumatic canal stenosis; magnetic resonance imaging; CT scan

Introduction

First described by Bastian[1] in 1867, post traumatic syringomyelia (PTS) has only been considered a real entity since the years 1950–60 after the experimental works of Freeman[2] in 1953 and the first report of Barnett and Jousse in 1966.[3] The incidence of PTS is increasing and is evaluated differently in the recent literature depending on whether it was diagnosed by clinical means (3.2%,[4] 4.45%,[5] 3.43%[6]) or by neuroradiological examinations.[7] The unforseeable nature, and the seriousness of this complication, raise the problem of pathogenic mechanisms. The most attractive pathogenic theory concerning extension of the syrinx is that of Williams,[8–10] in that it gives a central role to an increase in epidural venous pressures, causing intra cordal fluid movements (theory of 'slosh' and 'suck'). These phenomena would seem all the more important in so far as there is a blockage of subarachnoid flow at the level of the lesion. In 1993, at the International Medical Society of Paraplegia (IMSOP) Ghent meeting, we presented 13 cases of PTS, underlining the cord compression by the insufficiency of reduction of the vertebral lesion in ten patients and the existence of an arachnoiditis in three patients. This data corroborates the Williams's theory.[11]

This study aims to demonstrate, from a population of spinal cord injured patients, followed yearly, that predictive factors for the constitution of the cavity do exist, and in particular to correlate the role of the insufficiency of reduction of the spinal fracture as well as the phenomenon of the tethered cord with the occurrence of syringomyelia.

Materials and methods

In order to eliminate any selective bias, we analyzed all 128 spinal cord injured patients with old or new spinal cord injuries, followed, or hospitalized in our spinal cord unit during the years 1992–93. We asked all of these patients to have clinical and neuroradiological examinations. Among them, 53 were excluded for various reasons; previous partial neuroradiological examinations (15), examinations programmed too late (15), refusals (8), distance (9), death (3), metal artefact (3). Thus the population studied consisted of 75 SCI patients. The analysis of the sex ratio shows 62 males, 13 females; average age at time of study 41 (range from

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15–81). The duration from injury to the time of the study ranged from 1 month to 28 years with a mean of 8 years. The aetiologies consisted of 44 road traffic accidents, 18 falls, ten direct traumas, and three had a diving injury. The level of the vertebral injury was cervical in 19 patients, cervicothoracic in two, thoracic in 36, thoracolumbar in 16 and lumbar in two. The neurological status was evaluated in accordance with the ASIA-IMSOP classification. Twenty-one were tetraplegic (nine complete, 12 incomplete), 54 paraplegic (45 complete, nine incomplete). The distribution, according to Frankel's grading showed 54A, 7B, 5C, 2E. The analysis of clinical syndromes revealed 52 transverse syndromes, eight anterior cord, eight Brown-sequard, four central cord, and three conus medullaris syndromes.

The method involved three stages: (a) A clinical analysis at the time of the study: This indicated the upper and lower limits of the initial lesion-syndrome, sought for symptoms and signs of PTS, and risk factors of venous hyperpressure (ie voiding by abdominal pressure, obesity, gait, weightlifting or sports); (b) An analysis of the initial bony injury: This included a review of the initial plain films and of the surgery report. In this way we defined: at the cervical level fractures or luxations, depending on whether instability was bony or ligamentous; at the thoracolumbar level, we used the Denis's classification,[12] distinguishing the lesion as fracture (failure of anterior column), burst fracture (anterior and middle columns), seat-belt fracture (middle and posterior columns), and fracture dislocation (failure of the three columns). By convention, three types of vertebral lesions were defined: luxations, burst and fracture. During the reading of the surgery report, anatomical peroperative observations, if a laminectomy was done, and if the dura was opened, were noted; (c) A radiological analysis of the lesion site at the time of the study: This included standard X-rays, a CT scan, and MRI. From the standard X-rays, we judged the quality of osteosynthesis, and calculated the angle of kyphosis. The CT scan included slices in sagittal reconstructions and in the axial plane at the site of injury. The failure of the posterior wall and the existence of intra canal fragments were noted. The degree of impingement in the two planes of space was evaluated by the calculation of the percentage of canal stenosis following a formula adapted from Hashimoto[13] and Keene.[14]

When measured on the sagittal reconstructions, ΔD represents the percentage of canal stenosis in the sagittal plane (midline sagittal slice) and is obtained by the formula:

$$\Delta D\% = \left[1 - \frac{\text{Min}[x]}{\text{Max}\left|\frac{a+b}{2}\right|}\right] \times 100;$$

a and b represent the maximum diameters above and below the site of injury, x the minimum diameter at the site of injury (Figure 1). ΔS represents the percentage of stenosis in the axial plane and is obtained by the formula:

$$\Delta S\% = \left[1 - \frac{\text{Min}\,|Sx|}{\text{Max}\left|\frac{Sa+Sb}{2}\right|}\right] \times 100;$$

Sa and Sb represent respectively the areas above and below the site of injury, Sx on the site of stenosis (areas measured by planimetry) (Figure 2). The MRI was carried out with T1 and T2 weighted images including sagittal entire cord images in addition to sagittal and axial slices centred on the site of injury. Four types of spinal cord pathologies were sought: contusion, myelomalacia, cyst and syrinx. The presence of atrophy, cord transection, and arachnoiditis was noted. For this study, the different pathologies are defined as follows (Wang,[7] Silberstein[15]): *contusion* as a heterogeneous signal of the cord on T1 and T2 weighted images with a focal area of high signal on T1 images, surrounded by a much

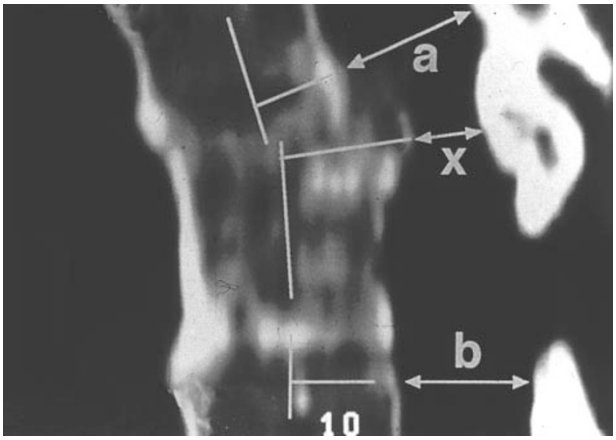

Figure 1 CT scan, measurement of percentage of spinal canal stenosis in the sagittal plane (ΔD)

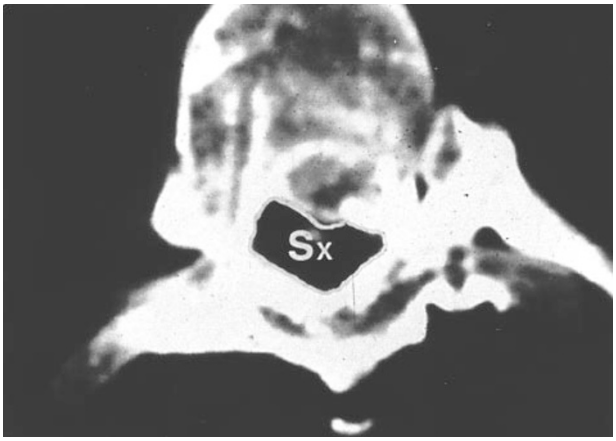

Figure 2 Ct scan, measurement of spinal canal stenosis in the axial plane (ΔS)

larger area of high signal on T2 images. *Malacia* is an area of signal intensity between that of CSF and normal spinal cord, decreased on T1, increased on T2, with an ill defined contour. *The cyst and the syrinx* are homogeneous areas with the same signal intensity as CSF (ie low T1, high T2), with a well defined contour. *The syrinx* extends beyond the limits of the vertebral injury, the cyst is round or oval and confined to the site of injury. *Cord atrophy* is an abnormal narrowing of the spinal cord in the sagittal plane, extending two segments or more beyond the site of injury, and finally *cord transsection* is a complete absence of spinal cord signal (complete discontinuity on sagittal images). *Arachnoiditis* is a thickening of the meninges but is difficult to diagnose on MRI.

Results

Spinal cord pathologies findings on MRI

The contusion was most often visualised (27 cases ie 36%), followed by syrinxes (21 cases ie 28%) (Figures 3 and 4), cysts (14 cases ie 18.6%), malacia (two ie 2.6%). Cord transsection was observed in six cases (8%), atrophy in 16 (21.3%), arachnoiditis in eight (10.6%). In 11 patients (14.6%) no major pathology was found. A syrinx was diagnosed in 21 patients (18 males, three females), mean age 42. The delay between injury and the first MRI ranged from 1 month to 29 years and 6 months (mean 8 years). The analysis of the influence of the neurological level showed a higher risk in those who were paraplegic (18 cases ie 33.3%) than for those who were tetraplegic (three cases ie 14.2%), but not in a significant manner ($P=0.10$). The risk was also not significantly increased in grade A (18 cases ie 33.3%) compared to grade B, C, D, E (three cases ie 14.2%) ($P=0.10$). Comparative analysis between flaccid (2/6 cases ie 33.3%) and spastic lesions (19/69 cases ie 27.5%) showed a non statistical significance. The occurrence of a syrinx had no correlation with the initial extent of the lesion-syndrome, this being on average 3.6 levels in the absence of a syrinx as opposed to 4.5 levels with a syrinx. The existence of venous hyperpressure factors increased significantly the risk of a syrinx, with 52.3% of the patients with a syrinx displaying hyperpressure factors, as opposed to 37.3% patients of our overall population ($0.05<P<0.10$).

The average longitudinal extent of the syrinx was 6.5 levels (2–18). The direction of extension was rostral in ten cases, caudal in five, and both in six. The average rostal extension was 4.5 levels that of caudal was two. The upper limit of the syrinx reached the bulbar area in two patients (Figures 3 and 4), without communication with the fourth ventricule, and C1 in two patients. The starting point of the cavity was always situated at the level of the initial cord injury, with one exception. The analysis of the rostral extremities of the syrinx revealed tapered extremities

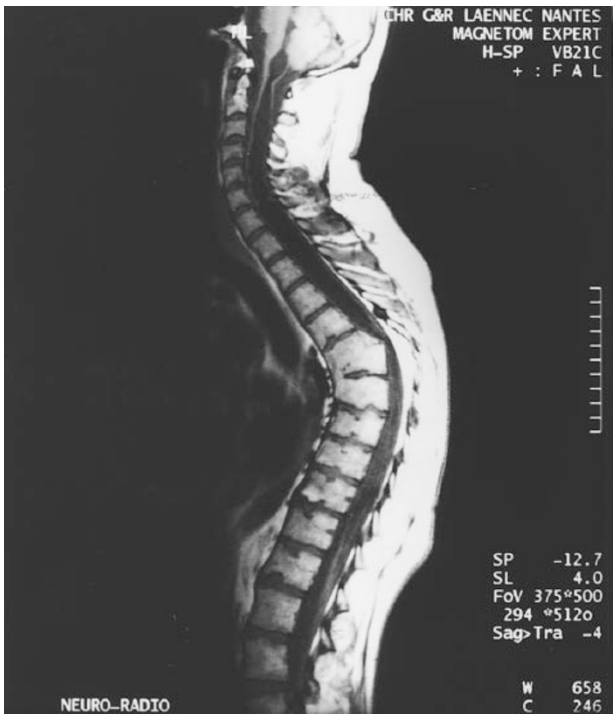

Figure 3 T1 weighted MRI; a 45-year-old patient with complete paraplegia T4 since 1970. Diagnosis of the syrinx in 1988. Clinical signs: right sensory disturbances and right modification of deep tendon reflexes. No neurological deterioration since 1988

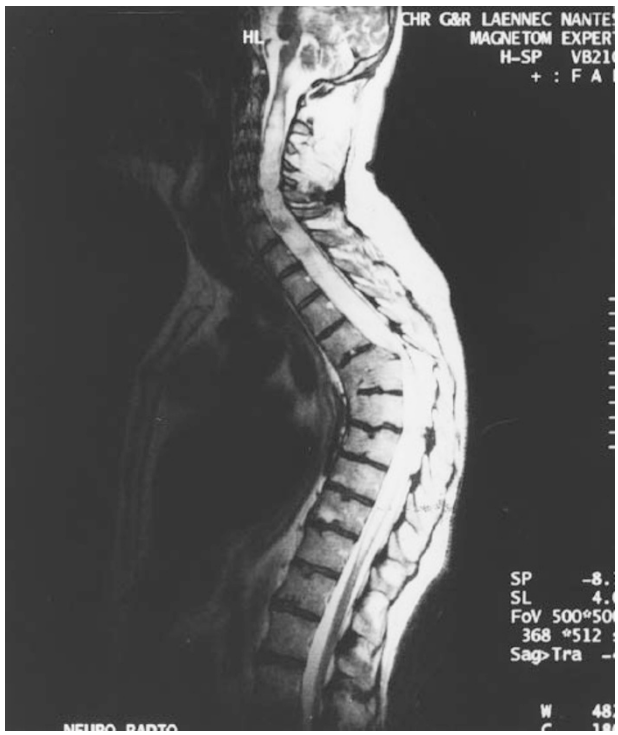

Figure 4 Same case in T2 Weighted MRI

in five cases, rounded in nine, and tense extremities in seven cases. The presence of septa was noted five times, exclusively in tense cavities. A correlation between the clinical and the MRI findings showed that 22 patients had neurological deterioration, the MRI confirmed the diagnosis of syringomyelia for 11. For the other 11 patients, the MRI showed a isolated contusion in two cases, contusion associated with atrophy in three, the association contusion-atrophy-cord transsection in one, a cyst in two, and no pathological images in three. Among the 21 patients displaying an MRI diagnosed syrinx, 11 showed neurological deterioration. The average extent of syrinx, in these patients, was 9.5 levels as opposed to three levels for patients without neurological change. This difference is statistically significant ($P<0.01$). In the same way the appearance of the rostral extremities is correlated with the neurological deterioration. Among the 14 patients displaying syrinxes with tapered or rounded extremities, five had a neurological deterioration as opposed to six of the seven patients who had syrinxes with their rostral extremities under tension ($0.02<P<0.05$).

The analysis of the initial bony injury

In conformity with our classification, a luxation in 73.3% of the cases (55), showed a burst fracture in 16% (12 cases), a fracture in 9.3% (seven cases). One patient had no vertebral injury. The initial treatment was surgical (reduction and internal fixation) in 85.4% of the cases (64), conservative in 14.6% (11 cases). Among the 64 patients who underwent surgery, the approach was posterior in 81.25% (52 cases), anterior in 10.9% (seven cases), both in 7.8% (five cases). A laminectomy was performed in 68.7% (44 cases) of operated patients. This laminectomy was isolated 36 times, associated with dura opening eight times. A closure of the dura is always performed, with an expansile dura plasty, if necessary. In five cases a traumatic dura opening was noted.

Radiological analysis of the lesion site

The review of standard X-rays, taken at the time of this study, showed kyphosis in 60% of the cases (45), with a mean angle of 18.2° (5–80°), equal or greater to 30° in 36% of the cases (27).

The percentage of spinal canal stenosis measured by CT scan reflects the quality of the reduction (Figure 5). The mean sagittal stenosis (ΔD) is 25% (extreme −20%, +100%). In 51 cases (68%), ΔD is between −20% and +30%, 24 patients (32%) have a sagittal stenosis greater than 30%. The mean axial stenosis (ΔS) is 20.5% (extreme −80%, +100%). In 44 cases (58.6%), ΔS is between −80% and +20%. Thirty-one patients (41.33%) have an axial stenosis greater than 20%. Spinal canal stenosis is the result of posterior wall prolapse in 69.3% of the cases, fixed intracanalar fragments in 26.6% of the cases or free 4%, and/or of kyphosis for which the seriousness increases in line with the importance of the stenosis. Indeed 63% of the patients presenting a kyphosis greater or equal to 30°, had a ΔD greater than 30%. If we correlate ΔD with the initial treatment, all the patients for whom ΔD is greater than 30%, had undergone surgery by posterior approach or received conservative treatment, this ΔD being always less than 30% for anterior or double approaches.

Post-traumatic canal stenosis and occurrence of syrinx

ΔD is significantly higher in the group of patients with a syrinx (44.9% *versus* 17.3%; $P<0.001$). This is also true for ΔS (33.9% *versus* 15.2%; $0.02<P<0.05$). Consequently, the occurrence of a syrinx is significantly correlated with the spinal canal stenosis (Figure 6). Two threshold values stand out in this present study: that of 30% of stenosis for ΔD, 20% for ΔS. Indeed when ΔD is less than 30%, the incidence of syrinx is

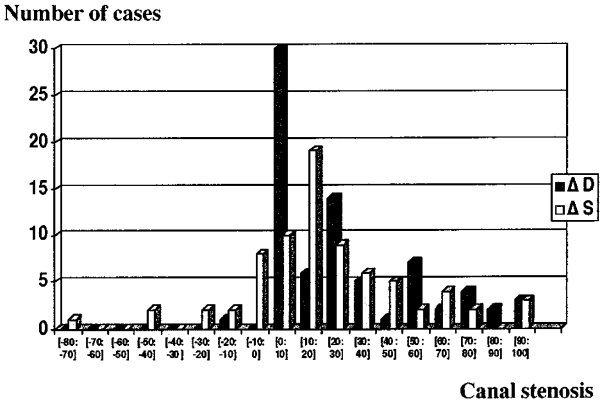

Figure 5 Distribution of the canal stenosis in the sagittal plane (ΔD) and in the axial plane (ΔS). The Y axis indicates the number of cases in each range of canal stenosis

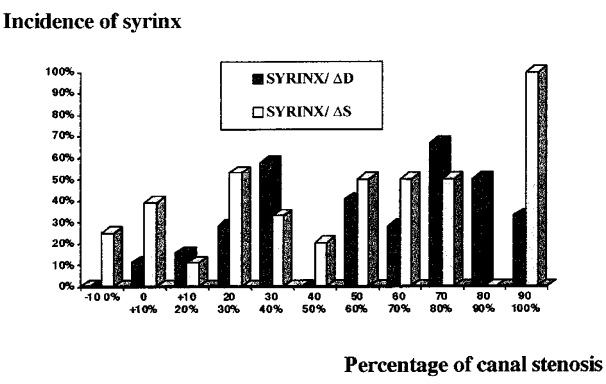

Figure 6 Relationship between occurrence of syrinx and spinal canal stenosis. The Y axis indicates the incidence of syrinx (in percentage) in each range of canal stenosis in the sagittal plane (ΔD) and in the axial plane (ΔS)

15.6% (8/51 cases) as opposed to 54.1% (13/24 cases), when ΔD is equal or greater than 30% ($P<0.001$). In the axial plane when ΔS is less than 20% the occurrence of syrinx is 18.1% (8/44 cases) as opposed to 41.9% (13/31 cases), when ΔS is equal or greater than 20% ($0.02<P<0.05$) (Figure 7). The incidence of a syrinx is significantly decreased in patients who have had a laminectomy (12.9% as opposed to 66.6% without), only when ΔD is less than 30% ($0.001<P<0.01$). In the same way, the effectiveness of surgical opening of the dura seems to decrease the incidence of a syrinx developing, whatever the degree of stenosis (no syrinx in our eight cases of dura opening).

Comments

(a) The first part of our comments concern the MRI findings and particularly the incidence, definition, and correlation with clinical symptoms and the different pathological images on MRI. We found an incidence of 28% of syrinxes on MRI. These results differ in some way from those in the literature, in part because there is no generally agreed definition of a syrinx. The majority of the authors are in agreement concerning the type of signal (same than CSF) and the well defined character of the margins. The discrepancies concern the diameter,[16] and the required extent.[10,17–19] Our definition relies on the definition of Wang[7] and we postulate that a syrinx must extend beyond the lesional site, the minimal extent required being two levels. The comparison of the incidence of syrinxes in this present study and that of Wang[7] shows higher incidence in our study (28% *versus* 20.9%), despite a lower mean delay post injury (8 years *versus* 29 years). This finding does not corroborate the fact that the risk of syrinx increases with the delay post injury. The length of syrinx is, in our study, from 2 to 18 segments (average 6.5), in accordance with the findings of Wang[7] (average six). All syrinxes but one started at the level of the initial lesion site, which underlines the major role of the initial core in the genesis of syrinx.[5,7–10] Finally, the existence of neurological deterioration in patients presenting syrinxes, is statistically correlated with the extent of syrinx, and with the aspect of the extremities of the cavity. As Wang,[7] we believe that the longer the syrinx, the greater the probability that it is symptomatic. In this study 50% of patients presenting neurological deterioration have a syrinx. The study of the images in the other half demonstrates the predominance of atrophy (36% of the cases). The distinction between post traumatic cystic myelopathy and post traumatic non cystic myelopathy is reported in the literature and for some authors, is correlated with myelomalacia,[20,21] with atrophy[22] and in all cases,[20,21] with the presence of a tethered cord a the lesion site. This post traumatic non cystic myelopathy, the clinical expression of which is similar to that of PTS, requires further study.

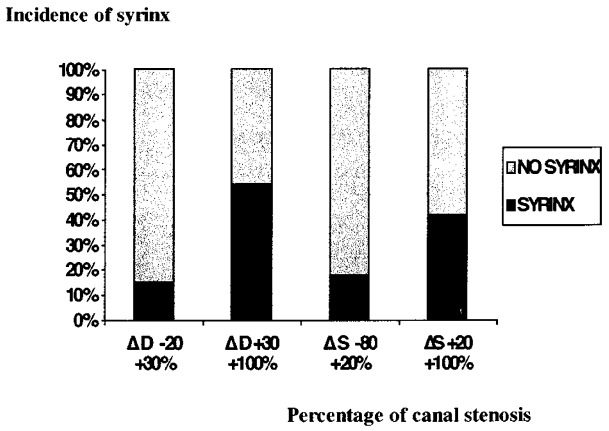

Figure 7 Relationship between occurrence of syrinx and spinal canal stenosis. The X axis indicates four ranges of canal stenosis; ΔD < 30%, ΔD > 30%, ΔS < 20%, ΔS > 20%. The Y axis indicates the incidence of syrinx in each range. ΔD < 30%: incidence of syrinx = 15.6%; ΔD > 30%: incidence of syrinx = 54.1%. ΔS < 20%: incidence of syrinx = 18.1%; ΔS > 20%: incidence of syrinx = 41.9%. Threshold values: ΔD = 30% ($P<0.001$); ΔS = 20% ($0.02<P<0.05$)

(b) The second part of our comments concern the relationship between the canal stenosis and the genesis of the syrinx It has recently been underlined,[6,23] that the occurrence of syrinx is reported in the literature in patients undergoing either conservative or surgical treatment, but that there are no reported cohort studies comparing the incidence of PTS in these two groups of patients. To our mind, the question is not the type of treatment but its result, particularly the quality of the reduction of the vertebral fracture. This present study shows that there is a significant correlation between post traumatic canal stenosis (ie bony compression) and the occurrence of syrinx, with objectivation of critical thresholds of impingement, above which the incidence of syrinx increases significantly. The apparent discordance between our findings and those of Wang,[7] who found no correlation between the spinal canal stenosis, the degree of kyphosis and the occurrence of sryinx is linked to a difference of evaluation of the spinal canal stenosis. In fact, Wang defined the spinal canal compromise on MRI images, and considered as stenosis a reduction of the anterior posterior diameter superior to 1/3 of the average adjacent diameters. In our study, the evaluation of spinal canal stenosis by CT scans allows a precise measurement of the midsagittal diameter, and of the narrowest cross sectional area, and the calculation of a percentage of impingement. The measurement of this stenosis in the two planes of the space (ie sagittal and axial), gives a better evaluation of the bony compression, the measurement of ΔS being sometimes altered by the acquired axial enlargement of some burst fractures.

This correlation between the spinal canal stenosis and the occurrence of a syrinx supports the pathogenic theory of Williams[8,9] who recognised two steps in the formation of PTS: the initial formation of the cyst, either by liquefaction of hematomyelia, myelomalacic necrosis, release of lysosomial enzymes, or by ischemia and phenomena of tethered cord at the origin of traction-distension produced by arachnoiditis; the secondary extension of the cavity which is the consequence of an increase in epidural venous pressure, at the origin of intra cordal fluid movements. The 'slosh', leading to the rostral extension and breaking down the zones of structural weakness, and the 'suck', consequence of the pressure gradient being at the origin of the caudal extension and the filling of the cavity. These two phenomena are increased when there is a blockage in the subarachnoid space. The significant correlation with factors of venous hyperpressure found in this present study, the worsening reported in the literature after straining,[8,24] weightlifting exercises,[25] forced expiratory through a closed glottis, coughing or Vasalva manoeuver,[24] lithotripsy,[26] comfort this theory.

In his 1981 publication,[8] Williams emphasized the narrowing of the spinal canal, as well as the presence of scars of the dura and arachnoid at the site of injury, as critical factors for the development of syrinx. Other authors pointed to the major role of phenomenon of tethered cord in the genesis of syrinx, considering it as an essential prerequisite, and underlining the constance of arachnoiditis.[21] The experimental study of Cho[27] on rabbits confirms the major role of subarachnoid blockage in the pathogenesis of syrinx.

The widely evoked role of persistent bony compression[8–10] has been demonstrated in a recent study[5] which correlates the occurrence of syrinx in 20 cases with kyphosis. In the latter,[5] two patients with PTS underwent vertebral realignment, which was followed by collapse of the syrinxes. Similarly, Sgouros and Williams[28] showed the effectiveness in all cases of a laminectomy associated with opening of the subarachnoid space on the treatment of the syrinx. Indeed the treatment of the persistent bony compression allows collapse of syrinx by normalisation of CSF pathways in the subarachnoid space. This data corroborates our findings that the insufficiency of reduction of the verbal lesion, the post traumatic canal stenosis, can be a risk factor in the enlargement of syrinxes by blockage of CSF flow.

The treatment of syrinx, besides techniques of drainage, implies curing of the major mechanism of the filling of the cavity, and should consider the treatment of the spinal canal stenosis, possibly associated with the treatment of the arachnoiditis.

The prevention of PTS, requires the absolute reduction of the initial vertebral injury, whatever the mode of treatment. Williams[9] underlined the necessity of a laminectomy at the acute phase, coupled with a dura opening and the evacuation of hematoma to prevent the constitution of the syrinx. But our results show that the laminectomy is insufficient when the canal stenosis is too great (ie more than 30%). In our experience a limited laminectomy is only performed in cases of posterior approach for the open reduction and stabilisation. This laminectomy is sometimes associated with a dura opening to allow the evacuation of hematoma. In our opinion, such surgical procedures decrease the risk of arachnoiditis. In fact, the choice of the surgical approach, at the acute phase, must be determined by the type of the vertebral injury. Our results show that all the patients for whom the canal stenosis is greater than 30% had undergone a posterior approach, the canal stenosis being always less than 30% for anterior or double approaches. Indeed anterior or anterolateral approaches allow a better canal restoration, particularly in burst fractures which are difficult to treat by a posterior approach. Whatever the surgical approach is, the aim being the canal restoration, the effectiveness of the reduction must be evaluated by CT Scan.

To conclude: this present study seems to demonstrate the role played by the insufficiency of reduction of the vertebral lesion in the genesis of syrinx. The quality of the initial treatment of the vertebral injury could be the first step in the prevention of syrinx.

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