Spinal obstruction-related versus craniocervical junction related syringomyelia: a comparative study of spinal cord injury

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Research Article

Keywords: Syringomyelia, Decompression, Spinal cord injury, Chiari malformation, CSF

Posted Date: November 18th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-1073541/v1>

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Abstract

Background

No prior reports have focused on spinal cord injury (SCI) characteristics or inflammation after destruction of the blood-spinal cord barrier by syringomyelia. To compare the difference of syringomyelia-related central SCI between craniocervical junction (CCJ) and post-traumatic syringomyelia (PTS) before and after decompression.

Methods

Between 2015 and 2019, 106 CCJ, 26 CCJ revision and 15 PTS patients (mean history of symptoms 71.5 ± 94.3 , 88.9 ± 85.5 and 32.3 ± 48.9 months). The symptom courses were analysed with the ASIA, Klekamp and Samii scoring systems and Kaplan-Meier statistics for neurological changes. The mean follow-up was 20.7 ± 6.2 , 21.7 ± 8.8 and 34.8 ± 19.4 months.

Results

Compared with the other group, the interval time after PTS was longer, but the natural history of syringomyelia was shorter ($P=0.0004$, 0.0173 , respectively). The initial symptoms were usually paraesthesia ($P=0.258$), and the symptoms were mainly hypoesthesia ($P=0.006$), abnormal muscle strength ($P=0.004$), gait ($P<0.0001$) and abnormal urination ($P<0.0001$). SCI associated with PTS was more severe than that CCJ related ($P=0.003$). The cavities in the PTS group were primarily located at the thoracolumbar level, which was different from those in the cervical-thoracic segment at the CCJ. The rate of syrinx/cord was more than 75% ($P=0.009$), and the intradural adhesions tended to be more severe ($P<0.0001$). However, there were no significant differences in peripheral blood inflammation markers (PBIM) or long-term clinical efficacy except for the RBC ($P=0.042$).

Conclusion

The natural history of PTS tends to progress faster and is more severe than CCJ related. PBIM had no distinguishing effect on the difference in inflammation of syringomyelia except for the RBC. The predictive value of NLR for syringomyelia-related inflammation except in the acute phase was negative.

Introduction

The most common clinical manifestation of syringomyelia is dilatation of the central canal of the spinal cord. It is often associated with Chiari malformation (CM), basilar invagination or atlantoaxial dislocation, arachnoid adhesion and other cerebrospinal fluid circulation disorders¹⁻⁵ and is often

associated with other spinal deformities. Thus, it is a type of chronic central spinal cord injury (SCI)⁶. In patients with SCI caused by trauma, 50% of them have syringomyelia^{7,8}. In some cases, the cavity can be reduced by intradural decompression⁹. However, SCI-related symptoms, such as dissociative sensory disturbances, muscle atrophy, and joint deformity, are often unimproved and can even worsen¹. Therefore, how to repair SCI caused by the cavity is a bottleneck in clinical treatment. Ependymal cells surrounding the central canal are a source of endogenous stem cells, indicating a potential method of endogenous SCI repair^{10,11}.

To date, there is no feasible animal model of CM except compression^{12,13}. SCI is accompanied not only by damage to the nerve tissue-cerebrospinal fluid barrier but also by damage to the nerve tissue-blood barrier¹⁴. Therefore, the pro-oxidation and antioxidation processes that occur in the CNS may be reflected in the components of the CSF and blood. A better understanding of the potential molecular pathways associated with syringomyelia formation will reveal targets for the treatment and prevention of syringomyelia.

However, no previous reports have been published comparing syringomyelia associated with CM, Revision and PTS.

Methods

The study was reviewed and approved by the local ethics committee with waiver of informed consent from patients given its retrospective nature.

Between January 2015 and December 2019, 146 consecutive patients with intradural decompression for syringomyelia associated with CM, revision or PTS were treated at our institution (Table 1). In this study, PTS was defined as local arachnoid obstruction. The detailed inclusion and exclusion criteria are shown in Figure 1.

Klekamp and Samii scores (KS scores)¹⁵ and ASIA (evaluated by YCH) were used to evaluate the clinical course of the different groups before and after surgery. The long-term results were summarized with Kaplan-Meier statistics (Figure 2). The SC tension group was defined as > 75%, 50-75%, 25%-50%, 10%-25% and < 10% by the ratio of syrinx/cord (Figure 3). Peripheral blood inflammation markers (PBIM) were often tested one day before surgery (Figure 4).

Patients in the CM and revision groups will suffer from former magnum and foramen of Magendie dredging (FMMD), as has been reported previously (Figure 5)⁹. Finally, for PTS patients, we adopted anterior or posterior decompression. In addition, some authors have suggested an anatomy-based comprehensive classification of spinal osteotomies or arachnoid lysis (Figure 5) for compression fractures⁸.

Follow-up data were obtained during outpatient visits or by telephone interviews. Treatment success was defined as a sustained improvement of preoperative symptoms or stabilization of previously progressing symptoms. Treatment failure was defined as postoperative neurological deterioration. Patients were assessed at 3 months and 12 months postoperatively for neurological function using KS scores (Table 2). Long-term results were summarized with Kaplan-Meier statistics in the three groups. The patients also underwent postoperative MRI to determine the tension of the syrinx.

Statistics

For statistical tests of significance, the chi-square test, Kruskal-Wallis test, Mann-Whitney test, one-way ANOVA test and Fisher tests were used. Long-term follow-up was analysed with the Kaplan-Meier method by RStudio Version 1.3 to determine the rates of patients with and without postoperative clinical recurrences. For statistical analyses, the software packages Prism version 7.0 and SPSS version 25.0 were used.

Results

The clinical characteristics of the cases are presented in Table 1. None of the patients in the CM or revision group had an atlantoaxial dislocation history. Two patients in the CM group suffered from dorsal kyphosis. In one case in the CM group, syringomyelia progressed to the medulla oblongata acutely, and the syringomyelia was partially relieved after FMMD⁷. Interestingly, the preoperative NLR of the patient was as high as 6.5. Most patients suffered a history of trauma in the subarachnoid compression group, among which one had local subarachnoid adhesion.

The CM group had 106 patients (with a mean age of 48.0 ± 12.7). The revision group (47.0 ± 11.3) was similar to the CM group. Most of patients in all three groups were concentrated in the range of 40-60 years old, but in our centre, paediatric patients are frequently treated, so the number of patients in the CM group aged 1-20 years was lower in this study. PTS patients were mostly male ($P < 0.0001$), and there was no significant difference in age compared with the other groups ($P = 0.8018$). Nearly half of the PTS group had experienced a complete SCI. Compared with the revision group, the interval time after PTS was longer ($P = 0.0004$) but the natural history of syringomyelia was shorter ($P = 0.0173$). The initial symptoms of syringomyelia were usually paraesthesia ($P = 0.258$) and neuropathic pain (13.33%), but these symptoms were rare in the PTS group. The symptoms in the PTS group were mainly hypoesthesia ($P = 0.006$), abnormal muscle strength ($P = 0.004$), abnormal gait ($P < 0.0001$) and abnormal urination ($P < 0.0001$). Compared to the other groups, the revision group had a higher rate of occipital pain ($P = 0.099$) and swallowing dysfunction ($P = 0.01$), while differences in neuropathic pain ($P = 0.178$) and dysesthesia ($P = 0.303$) showed no significance.

The cavities in the PTS group were primarily located at the thoracolumbar level, which was different from those in the cervical-thoracic segment at the craniocervical junction (CCJ). The tension in the revision

group was more than 75% ($P=0.009$).

SCI associated with PTS was more severe than that associated with CCJ. Compared with the PTS group, the SCI caused by syringomyelia associated with the CCJ was more distributed in grade D ($P=0.003$). Moreover, the decrease in pinprick and light touch sensation was higher in the PTS group ($P=0.0005$, $P<0.0001$, respectively). However, the SCI history in the PTS group often caused irreversible damage to SC function. Although the history of the revision group was longer, there was no significant difference in ASIA compared with the CM group. There was no significant difference in UE muscle strength among the three groups ($P=0.1012$). It should be noted that previous SCI in the PTS group usually does not affect UE muscle strength. The subdural adhesions were often worse ($P<0.0001$) in the PTS group. However, there was no significant difference among the groups for PBIM except the RBC, which showed marginal statistical significance ($P=0.0421$), presumably because the blood-SC barrier limited the reflection of the difference of chronic inflammation or the sample size is too small.

Compared with the CM group, the revision group had a higher proportion of cerebellar tonsil manipulation, but there was no significant difference ($P=0.276$). The PTS group had the highest rate of adhesion lysis, followed by fusion. After FMMD with or without revision, complications within 7 days were observed in 23.07% and 9.43%, respectively, without a significant difference for patients with PTS ($P=0.133$). Syringomyelia declined to 58.8% of the CM group and remained stable at 39.95%. The rate of postoperative increase was meagre, at 1.25%. Due to the higher tension of the syrinx in patients in the revision group and PTS group, the rate of syrinx cavities decrease was higher in this group, without reaching statistical significance ($P=0.123$). The analysis of long-term outcomes suggested no significant differences among the CM group, revision group and PTS group ($P=0.257$) (Figure 2).

Due to the influence of intradural manipulation, the relief rate of headache was low in the short term, but the rate of improvement was higher in the CM group after 3 months (70% vs. 41.6%). In terms of neurogenic pain, the improvement rate of the CCJ group was higher than that of the PTS group (55.2%, 52.6% vs. 42.9%). In terms of paraesthesia, the improvement rate of the CM group and the PTS group was higher than that of the revision group (67.5%, 58.3% vs. 45.4%). For hypoesthesia, the improvement rate of the PTS group was higher than that of the CCJ group (53.8% vs. 33.3%, 45.4%). Furthermore, because of the longer history in the revision group, their symptoms were often more severe. Although the lower limb symptoms in the PTS group were more severe in terms of MS, the improvement rate of MS related to cavitation was slightly lower than that in the CCJ group (38.5% vs. 40%, 38.9%). In terms of gait, the improvement rate of the CM group was higher than that of the revision group and the PTS group (54.8% vs. 28.5%, 23.1%), but the PTS group's past trauma history can easily lead to residual gait disorder, which makes confounding factors unable to be ruled out. In terms of urination, the improvement rate of the revision group and the PTS group was higher than that of the CM group (66.7%, 40% vs. 33.3%), but the proportion of urination disorders in the CM group and the revision group was relatively low. The CCJ group had a higher remission rate of cranial nerve symptoms in the posterior group (72.7%, 66.6%), while in the PTS group, there were 2 cases of bulbar cavity causing related symptoms, and 1 case

was relieved after surgery. In terms of sweating symptoms, the remission rate in the CCJ area was lower (14.3%, 0%), while there were 2 patients in the PTS group, of which 1 case was relieved after surgery.

Discussion

With the ageing of society, an increasing number of cervical degenerative diseases patients suffered central SCI, many of whom have symptoms that are more severe in the upper limbs than in the lower limbs¹⁶. Syringomyelia is an expansion of the central canal of the SC, which is the simplest form of central SCI. With the help of a syringomyelia model, we can better study central SCI caused by various conditions. CM is the most common clinical cause of syringomyelia. Spinal obstruction-related syringomyelia is similar to the compression syringomyelia model¹³. The long-term natural history of syringomyelia remains unclear^{7,17}. The history of syringomyelia related to the craniocervical junction, especially in the revision group, was significantly longer than that in the PTS group. We suspect that the duration of the natural history may be related to the extent of SCI. In addition, most patients in these three groups suffered intradural decompression. Therefore, we made relevant clinical comparisons among the three to compare their similarities and differences to improve our understanding of central SCI. In future studies, we will explore its molecular mechanism, deepen the understanding of ependymal cells involved in the repair of SCI, and provide a theoretical basis for endogenous SC repair.

In our study, PTS were mostly male. This might be because men are more likely to be injured. Studies have pointed out that the fluid in the cavity mainly comes from the subarachnoid space¹⁸. In a case of abnormal pulsation of cerebrospinal fluid, it can enter the central canal of the SC through the perivascular space. In our data, the proportion of high-tension cavitation in the PTS group was higher than that in the CM group, but the data of the revision group may be influenced by outpatient selection bias. The potential cause is that the mechanisms of the formation and the postoperative changes of the cavity in the PTS and CM groups are different, including more serious subdural adhesion and BBB destruction in PTS¹⁹. However, some authors suggest that there is no correlation between tension and injury²⁰. With the expansion of the central canal, SCI and dysfunction gradually became aggravated, which is also in line with the clinical cavities in the CM group. In the CM group, the history of related symptoms lasted longer than that in the PTS group, and the progression of SCI was slower. Generally, pain-temperature cross-fibres immediately in front of the central canal are the first to be involved, and the typical clinical manifestations of segmental pain-temperature sensation and tactile separation appear. Our data showed that there was no significant difference in upper limb muscle strength among the three groups, while the proportion of hypoesthesia in the revision and PTS groups was higher. In addition, we noticed that both pain and light sensation often declined in the PTS group, while a higher proportion of dissociated sensory loss was observed in the CM group, which suggests that PTS is associated with more severe trauma. However, neurogenic pain was rare in the PTS group, which suggests that central canal dilatation was more likely to be accompanied by SC parenchyma damage in the PTS group. The PTS group may be related to the faster progression of abnormal CSF circulation dysfunction; further enlargement of the central canal involves the anterior horn neurons and manifestations such as muscle atrophy. In clinical

practice, we also noticed that in the craniocervical junction group, the first and second interosseous muscles of the upper limb or small interosseous muscle atrophy and ulnar finger extension difficulty were common. However, due to a short medical history, the PTS group seldom showed anterior motor horn injury of the upper limb or muscle atrophy. Longitudinal conduction tracts farther away from the central canal, such as the corticospinal tract and spinothalamic tract, always show signs of damage in the later stages of the disease. The revision group had a higher proportion of impaired motor function and gait than the CM group, which also supports this view. However, many studies have found that the size of syringomyelia is not related to the severity of clinical symptoms^{20,21}. Our previous basic research found that SCI and changes occurred in the early stage of cavity formation. The occurrence of SCI caused by cavities may not be solely due to central canal expansion, but it is likely that central canal expansion and SCI coexist^{13,19}. Therefore, it is necessary to further clarify the pathological damage and the mechanism of SCI caused by syrinx.

Generally, it has been suggested that immunity and inflammation play major roles in the initiation and development of pancreatic cancer, hepatocellular carcinoma, glioma and other tumours^{22–24}. It has been shown that inflammation is related to changes in peripheral blood leukocytes that are related to the NLR²⁵. The degree of preoperative PBIM, for example, neutrophils, lymphocytes, monocytes, or their ratios, has been suggested to be related to the prognosis and immunity therapy outcome of cancers^{26,27,28}. Ependymal cells are activated after SCI, and then cell proliferation, differentiation and migration play a repair role²⁹. In previous studies, we found that the number of ependymal cells increases significantly after the formation of syringomyelia. In addition, syringomyelia was found to be significantly related to the inflammatory pathway through previous syringomyelia animal model SC tissue transcriptome and metabolomic³⁰. Here, our study confirmed the negative predictive value of the leukocyte count and NLR for inflammation. We noticed that there was a patient with acute syringomyelia progression in the CM group with a lumbar compression fracture, and his NLR was as high as 6.5, which may indicate that the inflammatory reaction for the acute phase was more severe⁷.

Some researchers have suggested that oxidative stress plays important roles in the pathophysiology of not only acute SCI but also chronic SCI^{31,32}. Erythrocytes have been shown to be potential markers for the diagnosis of some diseases³³. Some authors have suggested that erythrocytes lose all of their organelles when they mature, causing a reduction in their potential to replace proteins that have lost their functions, which makes them prone to any aberrations and very sensitive to oxidative stress³⁴. Woźniak suggested that higher lipid peroxidation will increase the concentrations of thiobarbituric acid reactive substances in the RBCs of cervical SCI patients³⁵. Recent studies have suggested erythrocytes as a potential biomarker in the treatment of oxidative stress-associated diseases, such as chronic obstructive pulmonary disease, cardiorespiratory fitness in chronic SCI individuals³⁶ and primary open-angle glaucoma³⁷. Some authors have already shown that mild anaemia or low RBC levels can be found after SCI³⁸. However, other authors suggested abnormally low levels of RBCs in early chronic SCI patients and augmentations over

time, and the levels of RBCs, Hb and Ht returned to near normal levels in late chronic SCI patients³⁹. The molecular mechanism of syringomyelia needs further research to elucidate.

Conclusions

The natural history of PTS tends to progress faster. The first symptom is usually paraesthesia, and SCI is more serious than syringomyelia associated with the craniocervical junction. The difference in inflammation of syringomyelia caused by different aetiologies cannot be found through PBIM except for the RBC.

Declarations

Acknowledgments

We would like to thank AJE for the English language editing.

Funding (information that explains whether and by whom the research was supported): Not applicable

Conflicts of interest/Competing interests (include appropriate disclosures) : Not applicable

Availability of data and material (data transparency) : Not applicable

Code availability (software application or custom code) : Not applicable

Ethics approval (include appropriate approvals or waivers) : The study was reviewed and approved by the local ethics committee with waiver of informed consent from patients given its retrospective nature.

Consent to participate (include appropriate statements) : Not applicable

Consent for publication (include appropriate statements) : Not applicable

Authors' contributions (All authors must be mentioned) : Chenghua Yuan: Writing - Original, Draft Data Curation, Jian Guan: Writing- Reviewing and Editing, Visualization, Yueqi Du: Software, Draft Data Curation, Zeyu Fang: Software, Draft Data Curation, Xinyu Wang: Software, Draft Data Curation, Qingyu Yao: Software, Draft Data Curation Can Zhang: Software Shanhang Jia: Resources Kai Wang: Resources Zhenlei Liu: Methodology, Resources, Wanru Duan: Resources Xingwen Wang: Resources, Visualization Zuwei Wang: Resources, Visualization Hao Wu: Resources Zan Chen: Resources Fengzeng Jian: Writing- Reviewing and Editing, Project administration.

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Tables

Table 1: Perioperative clinical data of syringomyelia of different aetiologies.

	Chiari I malformation (n=106)	Revision (n=26)	PTS (n=15)	P
Male	25(23.58%)	7(26.92%)	13(86.67%)	< 0.0001^a
Age, years	48.0 ± 12.7	47.0 ± 11.3	50.5±8.4	0.8018 ^b
1-20	5(4.71%)	0	0	
20-29	3(2.83%)	3(11.54%)	0	
30-39	14(13.21%)	4(15.38%)	1(6.67%)	
40-49	29(27.36%)	5(19.23%)	6(40%)	
50-59	36(33.96%)	11(42.31%)	5(33.33%)	
60+	19(17.92%)	3(11.54%)	3(20%)	
Previous ASIA				0.001^c
Complete	NA	0	6(40%)	
Incomplete		26(100%)	9(60%)	
Previous surgery				
Yes	NA	26(100%)	9(60%)	
Conservative		0	6(40%)	
Interval	NA	61.7 ± 60.4	203.0±136.4	0.0004^d
SM Symptom duration, months	71.5±94.3	88.9±85.5	32.3±48.9	0.0173^b
< 1	4(4.08%)	0	1(6.67%)	
1- 6	22(22.45%)	1(3.85%)	4(26.67%)	
7 -24	25(25.51%)	6(23.08%)	7(46.66%)	
> 24	47(47.96%)	19(73.07%)	3(20%)	
First SM Sign				0.258 ^c
Neuropathic pain	32(32.65%)	11(42.31%)	2(13.33%)	
Dysesthesia	37(37.75%)	11(42.31%)	9(60%)	
Sensory deficit	9(9.18%)	0	0	
Motor	20(20.42%)	4(15.38%)	4(26.67%)	
Symptoms				
Occipital pain	31(29.24%)	12 (46.15%)	0	0.099 ^a
Neuropathic pain	59(55.66%)	19 (73.08%)	7(46.67%)	0.178 ^a
Dysesthesia	75(70.75%)	22(84.62%)	12(80%)	0.303 ^a
Hypesthesia	61(57.55%)	22(84.62%)	13(86.67%)	0.006^a
Motor power	50(47.17%)	18(69.23%)	13(86.67%)	0.004^a
Gait	31(29.24%)	14(53.85%)	13(86.67%)	<0.0001^a
Sphincter function	9(8.49%)	3(11.54%)	10(60%)	<0.0001^c

Swallowing function	11(10.38%)	9(34.62%)	2(13.33%)	0.01^c
Sweating	14(13.21%)	2(7.69%)	2(13.33%)	0.844 ^c
ASIA				
UE	48.2±5.4	47.7±2.8	47.7±3.5	0.1012 ^b
LE	48.8±5.4	48.3±3.3	39.9±14.2	<0.0001^b
PP	104.5±12.2	105.3±7.9	91.5±11.5	0.0005^b
LT	106.1±11.4	105.3±7.8	94.0±10.1	<0.0001^b
A	0	0	0	0.003^b
B	1(0.94%)	0	1(6.67%)	
C	1(0.94%)	0	4(26.67%)	
D	90(84.91%)	25(96.15%)	9(60%)	
E	14(13.21%)	1(3.85%)	1(6.67%)	
Radiological data				
Ventilation dilation	7(6.60%)	1(3.85%)	0	0.945 ^{a1}
Scoliosis	22(20.75%)	5 (19.23%)	3(13.33%)	0.984 ^a
Occipitalization of atlas	8(7.55%)	3(11.54%)	0	0.792 ^{a1}
Basilar invagination	12(11.32%)	5(19.23%)	0	0.452 ^{a1}
Klippel-Feil syndrome	3(2.83%)	0	0	1.00 ^c
Syringomyelia	106(100%)	26(100%)	15(100%)	
Lesion location				
Medulla oblongata	2(1.89%)	0	2(13.33%)	
Cervical	14(13.21%)	3 (11.54%)	0	
Cervicothoracic	90(84.91%)	23(88.46%)	8(46.67%)	
Thoracic	1(0.94%)	0	2(13.33%)	
Thoracolumbar	0	0	1(6.67%)	
CTL	1(0.94%)	0	4(20%)	
Whole	0	0	0	
Cord/canal				0.009^b
A	51(48.11%)	19(73.08%)	11(73.34%)	
B	24(22.64%)	6(23.08%)	2(13.33%)	
C	26(24.53%)	1(3.84%)	2(13.33%)	
D	5(4.72%)	0	0	
E	0	0	0	
Management				
Arachnoid opened	106(100%)	26(100%)		
Tonsil manipulated	61(57.55%)	18(69.23%)		0.276 ^a
Fusion	NA	NA	4(26.67%)	
Arachnoid lysis			4(26.67%)	
Laminectomy			1(6.67%)	
Arachnoid			1(6.67%)	

lysis+Syringostomy				
Arachnoid lysis+Cord			3(20%)	
transection				
Cord transection			1(6.67%)	
SSS			1(6.67%)	
Intradural findings				<0.0001^b
0	14(13.21%)	2(7.69%)	2(16.67%)	
1	89(83.96%)	19(73.08%)	0	
2	3(2.83%)	5(19.23%)	10(83.33%)	
Complication				0.133^c
Isolated fever	2(1.89%)	0	1(6.67%)	
Aseptic meningitis	5(4.71%)	5(19.23%)	0	
CSF fistula	1(0.94%)	1(3.84%)	0	
Hydrocephalus	0	0	0	
Cerebral infarction	2(1.89%)	0	0	
Swallowing dys	0	0	0	
Haemorrhage	0	0	0	
Wound infection	0	0	0	
Urinary tract infection	0	0	0	
Pneumonia	0	0	1(6.67%)	
Total	10(9.43%)	6(23.07%)	2(13.33%)	
Length of follow-up (mos)	20.7 ± 6.2	21.7 ± 8.8	34.8±19.4	

* Significant difference (p < 0.05) between groups.

^aChi square test. ^{a1} Correction of the Chi square test.

^bKruskal-Wallis test.

^cFisher exact test.

^dMann-Whitney test.

^eOne-way ANOVA test.

Table 2: Changes in related symptoms of syringomyelia caused by different aetiologies

Symptom	Chiari I malformation (n = 106)	Revision (n = 26)	Craniocervical (n = 132)	PTS (n = 15)
Occipital pain				
Preop	4.6 ± 0.7	4.4 ± 0.7	4.6 ± 0.7	0
Postop	4.7 ± 0.7	4.4 ± 0.7	4.6 ± 0.7	0
3 mos	4.7 ± 0.6	4.7 ± 0.6	4.7 ± 0.6	0
1 yr	4.8 ± 0.4	4.7 ± 0.5	4.8 ± 0.4	0
Neuropathic pain				
Preop	4.1 ± 0.9	3.7 ± 1.0	4.0 ± 0.9	3.9 ± 1.2
Postop	4.2 ± 0.9	3.8 ± 1.0	4.2 ± 0.9	4.1 ± 1.0
3 mos	4.4 ± 0.8	4.1 ± 0.8	4.4 ± 0.8	4.1 ± 1.0
1 yr	4.4 ± 0.8	4.0 ± 0.9	4.3 ± 0.8	4.1 ± 1.0
Dysesthesia				
Preop	3.5 ± 1.1	3.1 ± 1.0	3.4 ± 1.1	3.3 ± 1.1
Postop	3.9 ± 1.0	3.3 ± 1.1	3.8 ± 1.0	3.7 ± 1.0
3 mos	4.1 ± 1.0	3.4 ± 1.1	3.9 ± 1.0	3.7 ± 1.0
1 yr	4.1 ± 1.0	3.4 ± 1.1	3.9 ± 1.1	3.7 ± 1.0
Hypesthesia				
Preop	3.6 ± 1.3	2.7 ± 1.0	3.4 ± 1.3	3.0 ± 0.9
Postop	3.6 ± 1.3	2.8 ± 1.0	3.5 ± 1.3	3.1 ± 0.7
3 mos	3.8 ± 1.2	2.9 ± 1.1	3.6 ± 1.2	3.3 ± 0.9
1 yr	3.8 ± 1.2	3.0 ± 1.1	3.6 ± 1.3	3.3 ± 0.9
Motor weakness[#]				
Preop	4.3 ± 0.9	4.1 ± 0.8	4.2 ± 0.9	3.5 ± 1.1
Postop	4.4 ± 0.8	4.2 ± 0.7	4.4 ± 0.8	3.6 ± 1.2
3 mos	4.5 ± 0.7	4.3 ± 0.7	4.5 ± 0.7	3.9 ± 1.1
1 yr	4.5 ± 0.8	4.3 ± 0.7	4.4 ± 0.8	4.0 ± 1.2
Gait ataxia				
Preop	4.5 ± 1.0	3.9 ± 1.1	4.4 ± 1.0	2.7 ± 1.5
Postop	4.5 ± 0.9	4.0 ± 1.0	4.4 ± 1.0	2.5 ± 1.4
3 mos	4.6 ± 0.8	4.0 ± 1.1	4.5 ± 0.9	2.7 ± 1.4
1 yr	4.6 ± 0.8	4.2 ± 0.9	4.5 ± 0.8	2.7 ± 1.4
Bladder function				
Preop	4.8 ± 0.6	4.8 ± 0.6	4.8 ± 0.6	3.3 ± 1.4
Postop	4.8 ± 0.6	4.8 ± 0.6	4.8 ± 0.6	3.3 ± 1.4
3 mos	4.8 ± 0.5	4.9 ± 0.4	4.9 ± 0.5	3.6 ± 1.3
1 yr	4.8 ± 0.5	4.9 ± 0.4	4.8 ± 0.5	3.6 ± 1.3
Swallowing				
Preop	4.8 ± 0.6	4.5 ± 0.7	4.8 ± 0.6	4.9 ± 0.3
Postop	4.9 ± 0.3	4.8 ± 0.4	4.9 ± 0.4	4.9 ± 0.3

3 mos	4.9 ± 0.3	4.8 ± 0.5	4.9 ± 0.4	4.9 ± 0.2
1 yr	4.9 ± 0.3	4.7 ± 0.7	4.9 ± 0.4	4.9 ± 0.2
Overall				
Better	80(75.5%)	20(77.0%)	100(75.7%)	9(60.0%)
Unchanged	13(12.3%)	2(7.6%)	15(11.4%)	1(6.7%)
Worsen	13(12.2%)	4(15.4%)	17(12.9%)	5(33.3%)

* Unless otherwise specified, all values are expressed as the mean ± SD.

Paralysis of the lower extremities indicates the muscle strength of the upper extremities.

Figures

CM-I, Chiari Malformation-I; PTS, post-traumatic syringomyelia; PFD, posterior fossa decompression; PFDD, posterior fossa decompression and duraplasty; CNS, central nervous system;

Figure 1

Flow chart of 147 consecutive syringomyelia patients between 2015 and 2019.

Figure 2

Left chart: Changes in related symptoms of syringomyelia caused by different aetiologies. Right chart: Survival curve of syringomyelia

Figure 3

Changes in the size of syringomyelia caused by different aetiologies before and after surgery

Figure 4

Peripheral blood inflammatory markers of syringomyelia caused by different aetiologies

Figure 5

Left chart: Case presentation of the CM group. A patient was found to have syringomyelia due to facial paralysis. (A) Schematic drawings of the foramen magnum region. Midsagittal T2-weighted MRI scans of the craniocervical region and CT scans suggested that there was no other instability or basilar invagination (B). Three (C) months after the initial surgery and 30 (D) months after the initial surgery showing the ratio of the syrinx/canal from A to B. (E, F) A hypertrophic tonsil obstructing the foramen of Magendie. (G, H) The right PICA (asterisk) obstructing the foramen of Magendie was lysed, and the tonsil was coagulated. Lt = Left tonsil; Rt = Right tonsil; M = Medulla oblongata Middle chart: Case presentation of the revision group. (A) Preoperative sagittal T2-weighted MRI shows a large syringomyelia. (B) Three months, (C) 9 months, and (D) 3 years after the first surgery. (E) CT shows that the partial bone defects of the occipital bone and syringomyelia persisted. (F) Postoperative MRI 2 years after the second surgery showing the syringomyelia was obviously reduced. (G, H) A hypertrophic tonsil obstructing the foramen of Magendie. (I, J) PICA (asterisk) obstructing the foramen of Magendie was lysed, and the tonsil was coagulated. Lt = Left tonsil; Rt = Right tonsil; M = Medulla oblongata Right chart: Case presentation of the PTS group. (A) A postoperative sagittal T2-weighted MRI scan shows some oedema and internal fixation. (B, C and D): Sagittal T2-weighted MRI and CT after 2 years shows a large syringomyelia (up to C4 and down to the L1 vertebra) and an L1 compression fracture. (E, F and G): Myelography showing that the circulation of cerebrospinal fluid was blocked at L1. (H and I): Postoperative sagittal T2-weighted MRI data showing that the syrinx was obviously reduced. (J and K): Intraoperative images showing that the obvious adhesion around the spinal cord at L1 was removed.

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