

ORIGINAL ARTICLE

Factors associated with early onset post-traumatic syringomyelia

H-Y Ko^{1,2}, W Kim¹, SY Kim^{1,2}, MJ Shin¹, YS Cha¹, JH Chang¹ and YB Shin¹

Study design: Retrospective study.

Objectives: To identify factors associated with the development of early onset post-traumatic syringomyelia within 5 years of spinal cord injury.

Setting: Department of Rehabilitation Medicine, Pusan National University School of Medicine, Korea.

Methods: We retrospectively examined the records of 502 patients with traumatic cervical or thoracic spinal cord injury who underwent follow-up magnetic resonance imaging (MRI) examinations more than once a year for at least 5 years. Patients were assessed in terms of the neurological level of injury, the severity of initial spinal cord injury, the use of surgery and the extent of spinal canal involvement. The latter was evaluated by calculating the shortest antero-posterior diameter of the injured vertebral canal and the spinal reserve capacity as shown on MRI at the time of trauma onset and at the time of diagnosis of syringomyelia.

Results: Syringomyelia developed within 5 years in 37 (7.3%) of the 502 patients. The mean age of these 37 patients was 44.6 years (range, 17–67 years) and the mean interval from spinal cord injury to onset of syringomyelia was 38.8 months (range, 2–54 months). The development of post-traumatic syringomyelia within 5 years was not significantly related to the severity or level of injury, the use of spinal surgery or the extent of spinal canal encroachment ($P \geq 0.05$ for each comparison).

Conclusion: Early onset syringomyelia occurring within 5 years after spinal cord injury was not associated with neurological injury level, severity of injury, the use of spinal surgery or canal encroachment.

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Keywords: post-traumatic syringomyelia; neurological level of injury; canal encroachment; spinal cord injury

INTRODUCTION

Post-traumatic syringomyelia can occur after traumatic spinal cord injury, and is a major cause of delayed aggravated neurological symptoms. Post-traumatic syringomyelia is characterized by the formation of a cavity filled with cerebrospinal fluid (CSF), which causes symptoms including severe pain, motor weakness and changes in spasticity. Syringomyelia was first described by Bastian in 1867,¹ and, in 1880, Struempell *et al.*² reported instances of syringomyelia following spinal cord injury.³

The clinical incidence of post-traumatic syringomyelia in patients with spinal cord injury has been estimated at 0.3–3.2%, although the incidence recorded radiologically and at autopsy appears to be higher than the clinical incidence.^{4,5} The increased use of magnetic resonance imaging (MRI) for diagnosis has caused post-traumatic syringomyelia to be reported in 12–22% of patients with spinal cord injury.⁶

The mechanisms underlying the development of post-traumatic syringomyelia have yet to be established. Processes postulated to be involved include: 'slosh and suck' at the site of injury, liquefaction, development of hematoma, a malacic change in the spinal cord, necrosis, enzymatic lysis, tissue loss and arachnoiditis.^{7,8}

Several studies have been sought to identify clinical and radiological factors associated with post-traumatic syringomyelia. Factors examined include: age at onset, gender, cause of injury, site of injury, severity of injury, type of vertebral fracture, treatment method and the

degree of encroachment into the vertebral canal.^{6,9} Complete spinal cord injury and arachnoiditis have been associated with an increased incidence of post-traumatic syringomyelia, and increasing age, damage at the cervical and thoracic levels, dislocated spinal fracture and the use of spinal instrumentation without decompression have all been identified as risk factors for the early development of post-traumatic syringomyelia.⁶ To date, however, none of those factors have been established as being definitively causative.

The present study have been sought to identify factors associated with the development of early-onset syringomyelia within 5 years following traumatic spinal cord injury. Patients were followed up for at least 5 years post-injury. Factors investigated included the site of neurological injury (that is, cervical vs thoracic), the severity of the spinal cord injury, the type of treatment (that is, surgery vs non-surgery), and the degree of spinal canal involvement. Data were obtained using MRI.

MATERIALS AND METHODS

This retrospective study examined the records of 502 patients with traumatic spinal cord injury who had undergone follow-up MRI examinations more than once a year for at least 5 years following the injury. Patients with spinal cord injuries received clinical follow-up, in the interval since 1993. Patients with a concurrent brain injury at the time of trauma onset, with lumbar or cauda equina injury, or who were pediatric, were excluded. The present study used

¹Department of Rehabilitation Medicine, Pusan National University School of Medicine, Beomeo, Mulgeum, Yangsan, Gyeongnam, Korea and ²Research Institute for Convergence of Biomedical Science and Technology, Pusan National University Yangsan Hospital, Beomeo, Mulgeum, Yangsan, Gyeongnam, Korea
Correspondence: Dr H-Y Ko, Department of Rehabilitation Medicine, Pusan National University School of Medicine, Beomeo, Mulgeum, Yangsan, Gyeongnam 626-770, Korea.
E-mail: drkohy@gmail.com

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the criteria for post-traumatic syringomyelia as a cystic cavity to extend at least two spinal segments.

Each patient was assessed in terms of the cervical or thoracic spinal cord injury, and the extent of injury was measured using the ASIA Impairment Scale (AIS), whereby 225 patients were of grade AIS A (complete injury), 28 of AIS B, 159 of AIS C and 90 of AIS D (the latter three classes had incomplete injuries). Finally, each patient was assessed in terms of whether the treatment involved surgery (decompression laminectomy and vertebral fixation; the surgery group) or not (the non-surgery group).

The extent of spinal canal involvement was evaluated by calculating the shortest anterior–posterior (A–P) diameter of the vertebral canal and by estimating the spinal reserve capacity on MRI scans taken at the time of trauma onset and at the time of diagnosis of syringomyelia. The percentage value of the shortest A–P diameter of the vertebral canal (A–P%) was calculated using the following formula proposed by Hashimoto *et al.*¹⁰ and Keene *et al.*¹¹

$A-P\% \text{ (at the level of skeletal injury)} = (1 - X/Y) \times 100$, where X represents the shortest A–P diameter on the mid-sagittal plane at the level of the vertebral injury, and Y represents the mean value of the longest A–P diameter measured in the mid-sagittal planes of the regions of the spinal canal proximal and distal to the injured vertebra (Figure 1).

The spinal reserve capacity was defined as the ratio of the area of spinal canal surrounded by the dura mater to the overall area measured on the horizontal plane of the damaged region of spinal cord, and was calculated using a formula proposed by Weisz *et al.*¹²

$\text{Spinal reserve capacity (\%)} = C2/C1 \times 100$, where $C1$ represents the area of the spinal canal and $C2$ represent the area of the neural content (the spinal cord) (Figure 2).

An independent samples' t -test was used to analyze associations between the development of syringomyelia and the factors investigated. Statistical analysis was performed using SPSS for Windows (Version 10.1; SPSS Inc., Chicago, IL, USA).

RESULTS

The 502 study patients were of mean age 46.2 years, and were composed of 407 men and 95 women. In terms of injury location, 237 patients had cervical injuries and 265 had thoracic injuries. With regard to the severity of injury, 225 patients had complete injuries and 277 had incomplete injuries (Table 1).

Syringomyelia developed within 5 years after injury in 37 (7.3%) of the 502 patients. Only eight patients of 37 had symptomatic clinical presentations suggesting syringomyelia including pain of the hands, alteration of sensation or motor strength. Twenty (8.4%) of the 237 patients with cervical injury and 17 (6.4%) of the 265 patients with thoracic injury developed syringomyelia within 5 years after injury ($P \geq 0.05$). Eighteen (8.0%) complete injury patients and 19 (6.9%) incomplete injury patients developed syringomyelia ($P \geq 0.05$). The mean age of syringomyelia patients was 44.6 years (range, 17–67 years). The mean time of onset of syringomyelia following occurrence of the spinal cord injury was 38.8 months (range, 2–54 months) (Table 2). Fifteen (6.7%) of the 223 surgery patients and 22 (7.9%) of the 279 non-surgery patients developed syringomyelia ($P \geq 0.05$).

In syringomyelia patients, the percentage value of the shortest A–P diameter of the spinal canal at the time of trauma onset (A–P %) was 38%, and the spinal reserve capacity was 71%. In non-syringomyelia patients, these values were 40 and 65%, respectively. The two groups did not differ in terms of these measures of spinal canal involvement ($P \geq 0.05$).

The mean interval between spinal cord injury and diagnosis of syringomyelia was 38.8 months. A–P% and spinal reserve capacity were compared between syringomyelia and non-syringomyelia patients at that timepoint. At the time of injury and at the time of

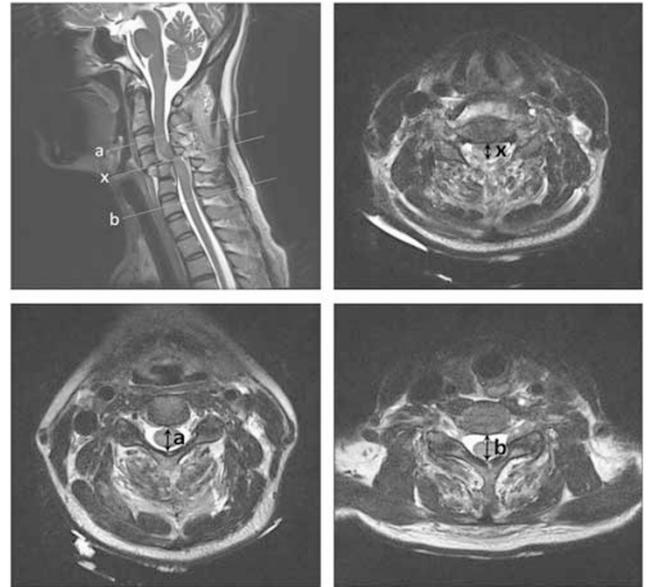


Figure 1 Magnetic resonance image, used to measure the ratio of the narrowest antero-posterior diameter in the spinal canal ($A-P\% = [1 - x/(a + b)/2] \times 100$), where x is the minimum diameter at the site of injury; a is the maximum diameter above the site of injury; and b is the maximum diameter below the site of injury.

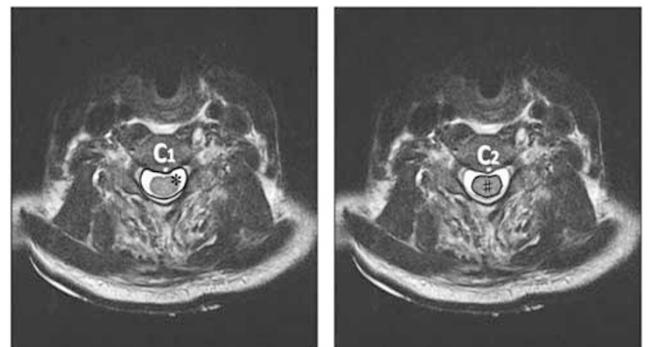


Figure 2 Magnetic resonance image, used to measure spinal reserve capacity, calculated as ($SRC\% = C2/C1 \times 100$), where $C1$ (*) is the area of the spinal canal and $C2$ (#) is the area of the neural contents.

Table 1 Demographic and clinical characteristics of the 502 patients with spinal cord injury

Demographic and clinical data	Values
Sex (male/female)	407/95
Age (years)	46.2 ± 12.3
Neurological level of injury (cervical/thoracic)	237/265
Severity of injury (complete/incomplete)	225/277
Treatment method (surgical/non-surgical)	223/279

diagnosis of syringomyelia, mean A–P% and spinal reserve capacity were 25% and 62%, respectively, in patients with syringomyelia, and 24% and 58%, respectively, in patients without syringomyelia; the figures were thus similar in the two groups ($P \geq 0.05$) (Figure 3).

Table 2 Demographic and clinical characteristics of the 37 patients with post-traumatic syringomyelia

Demographic and clinical data	Values
Sex (male/female)	31/6
Age (years)	44.6 ± 10.5
Average duration of onset (months)	38.8
Neurological level of injury (cervical/thoracic)	20/17
Severity of injury (complete/incomplete)	18/19
Treatment method (Surgical/non-surgical)	15/22

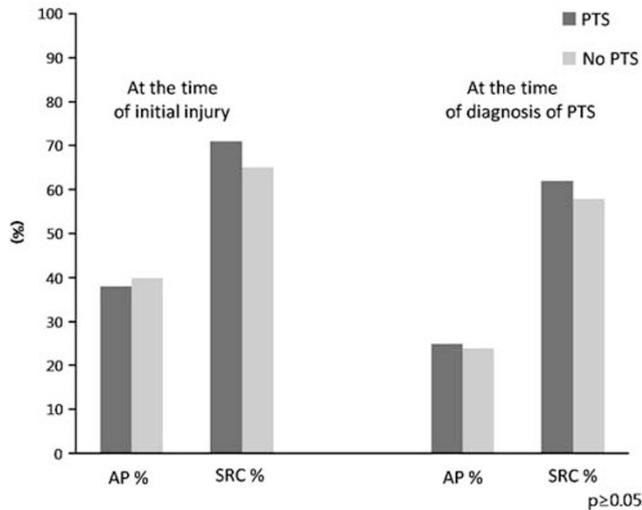


Figure 3 Extent of spinal canal encroachment at the time of injury and the diagnosis of post-traumatic syringomyelia in patients with and without syringomyelia. A–P%, ratio of the narrowest antero-posterior diameter in the spinal canal, SRC, spinal reserve capacity.

DISCUSSION

Although diagnosis of post-traumatic syringomyelia has been suggested to require the presence of a cystic cavity extending to more than two spinal segments, other authors have indicated that syringomyelia should be diagnosed on the basis of clinical and radiological findings, with no attention paid to cavity size.³ MRI has been found to be the most sensitive diagnostic imaging technique, and also yields information on the morphology of spinal cord cysts. On all imaging sequences, a spinal cord cyst should have a signal intensity equivalent to that of CSF. If necessary, gadolinium administration may help differentiate a cyst from a scar or tumor, because a post-traumatic cyst should not show nodular or mass-like enhancement.¹³

The time from spinal cord injury to the development of post-traumatic syringomyelia ranged widely in previous reports.^{5,6} We assessed only those patients who developed early post-traumatic syringomyelia, thus within 5 years after spinal cord injury, because of difficulties in conducting long-term follow-up including regular MRI examinations in a general clinical setting. Post-traumatic syringomyelia has been reported to occur anywhere between 3 months and 34 years following spinal cord injury.^{3,14} In the present study, we found that syringomyelia was diagnosed as early as within 2 months following spinal cord injury, and that the mean time for diagnosis was 38.8 months.

We found that the incidence of syringomyelia developing within 5 years after spinal cord injury was 7.3%. The incidence of clinical post-

traumatic syringomyelia has been reported to range from 1.1–3.4%,^{3–5,14} whereas the incidence of symptomatic syringomyelia has ranged from 1–9%.^{5,13,14} In contrast, autopsy and MRI studies have reported an incidence of approximately 22%.⁶

The present study examined whether the site of neurological injury was predictive of syringomyelia development. We found that the incidence of syringomyelia was similar in both cervical and thoracic spinal cord injury patients. Abel *et al.*¹⁵ and Klekamp *et al.*¹⁶ reported similar findings. However, other studies suggest that syringomyelia development may be associated with the injury site. Sgouros and Williams¹⁷ found that 78% of syringomyelia patients had a thoracolumbar spinal injury, whereas El Masry *et al.*⁵ and Perrouin-Verbe *et al.*⁹ concluded that cavities form more commonly in thoracic compared with cervical spinal cord injury patients. In contrast, Rossier *et al.*,¹⁴ Lyons *et al.*,¹⁸ and Vannemreddy *et al.*⁶ found that syringomyelia was more commonly associated with cervical spinal cord injury. Such observations are consistent with theories suggesting that the development of syringomyelia is associated with an increase in CSF pressure, because the mid-cervical spinal canal and the subarachnoid space are both relatively narrower than the thoracolumbar canal, as shown by Yarkony *et al.*¹⁹

We also investigated whether syringomyelia development correlated with the severity of neurological injury. We found that syringomyelia rates were similar in the complete and incomplete injury groups. However, other studies have concluded that the prevalence of syringomyelia in complete injury patients was approximately double of that found in incomplete injury patients.^{6,14}

The present study examined whether syringomyelia development differed when conservative or surgical treatment was given. We found that the incidence of syringomyelia was similar in both the surgery and non-surgery groups. In a study of post-traumatic spinal cord injury patients, Vannemreddy *et al.*⁶ reported that syringomyelia occurred earlier in patients who underwent fixation surgery or spinal instrumentation compared with non-surgery patients. In addition, syringomyelia occurred later in patients who underwent posterior laminectomy compared with those who underwent open reduction and internal fixation rather than laminectomy. Thus, local narrowing of the spinal canal may occur in patients who undergo open reduction and internal fixation without decompression surgery. Moreover, post-traumatic inflammatory responses in the separated soft tissue and bone may cause changes in the kinetics and dynamics of the CSF, accompanying fibrosis of the pia mater and arachnoiditis, in turn leading to the development of syringomyelia.⁸

The current study explored whether a link existed between the severity of spinal canal encroachment and the occurrence of post-traumatic syringomyelia. We found that the severity of encroachment at the time of spinal cord injury did not differ between syringomyelia and non-syringomyelia patients. Consistent with these findings, Wang *et al.*²⁰ found no correlation between the severity of spinal stenosis and the occurrence of post-traumatic syringomyelia. However, others have suggested that an association may exist between the severity of spinal stenosis and syringomyelia. A study in 20 patients found a correlation between the development of post-traumatic syringomyelia and spinal cord compression accompanied by kyphosis and syringomyelia.³ Moreover, spinal canal compromise and formation of a scar in the dura mater and subarachnoid membrane at the site of injury were reported to be the probable key factors in the formation of a spinal cord cavity.⁷ Using an animal model, Cho *et al.*⁸ showed that trauma-induced adhesive arachnoiditis caused blockage of the subarachnoid space; this interfered with normal CSF circulation. In the test animals, syringomyelia was caused by CSF influx into the

dorsal root entry point at the site of the injury, in turn attributable to an increased pressure within the subarachnoid space. Perrouin-Verbe *et al.*⁹ reported that development of post-traumatic spinal stenosis correlated with syringomyelia, and that syringomyelia was less frequent in patients with severe spinal stenosis who underwent laminectomy. It should be noted that a number of factors may affect correlations between spinal stenosis severity and syringomyelia; these include (for example) the type of surgery employed and the resultant anatomical changes.

The current study involved patients who were followed up for at least 5 years post-injury. However, other studies have shown that post-traumatic syringomyelia can develop as late as 34 years after spinal cord injury.⁵ This may explain why some of our present findings differ from those of long-term studies. In the present study, patients were classified simply into surgery and non-surgery groups, with no attention being paid to the surgical methods used. Also, neither patient age/gender nor MRI cord findings after injury was evaluated as a potential risk factor; this may be considered a limitation of our study. In addition, the validity of measurements of spinal canal compromise should be further evaluated. A future study should consider measurement bias in this regard.

CONCLUSIONS

The present study investigated whether a range of factors were predictive of syringomyelia development within 5 years following post-traumatic spinal cord injury. We found that 7.3% of patients developed syringomyelia within 5 years of injury. We also found that syringomyelia development was not associated with the site of neurological injury, the severity of the injury, a history of spinal surgery or the extent of spinal canal involvement. Therefore, it appears that such variables are of no predictive value in terms of syringomyelia development. Although the present findings are consistent with the reports of others, not all authors who work in the field are in agreement. Therefore, further meticulous long-term studies are required before the present findings may be considered clinically applicable.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

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