Syringomyelia as a Complication of Tuberculous Meningitis

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Abstract

Tuberculous meningitis (TBM) is a common manifestation of extrapulmonary tuberculosis. Syringomyelia is a rare complication of TBM. We report a case of syringomyelia due to TBM.

A 25 year old Thai male was admitted with a history of progressive paraparesis and loss of body sensation. He had a history of TBM in the previous year, and was treated with antituberculous drugs. Physical examination revealed a temperature of 37°C. Motor power was grade 3/5 with generalized hyperreflexia. He had bilateral loss of pain, temperature and vibratory sensation below the T7 level. A magnetic resonance imaging of the spine demonstrated a long segment of syrinx from C4 to the conus medullaris region. A T12-L1 laminectomy and syringosubarachnoid shunt were done. His clinical symptoms improved after surgery.

Key word : Tuberculous Meningitis, Complication, Syringomyelia

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Mycobacterium tuberculosis (TB) infection is a major public health problem in Thailand. Tuberculous meningitis (TBM) is a common manifestation of extrapulmonary TB. Its complications include a vegetative state, hemiparesis, cerebellar dysfunction and syringomyelia[1]. Syringomyelia may result from obstruction and anomalies of the craniocervical junction, tumor, trauma, and arachnoiditis. Syringomyelia is usually a late complication of TBM, and may not occur until 1 to 22

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years after the acute infection[2-7]. However, there have been a few reports of the development of syringomyelia as an acute complication[8,9]. We report here a case of syringomyelia due to TBM.

CASE REPORT

A 25 year old Thai male was admitted with a 3-week history of high grade fever, generalized throbbing headache and vomiting. Two weeks after the onset of these symptoms he noticed pain and difficulty in turning his neck. Four days before admission, he developed paraparesis. He had no significant past medical history. Physical examination revealed a temperature of 39°C, full consciousness, generalized cervical lymphadenopathy and nuchal rigidity. Motor power was grade 3/5 in the lower extremities. He had generalized hyperreflexia with negative Babinski's sign. He had bilateral loss of pain sensation below T7 level. No other abnormal neurological signs were detected.

Lumbar puncture showed clear cerebrospinal fluid (CSF) under high pressure, white blood cells 152/mm^3, a protein level of 6028 mg/dl, CSF glucose 23 mg/dl, while plasma glucose 99 mg/dl, and negative results of gram, Indian-ink, and acid fast bacilli (AFB) stains. CSF cultures were negative. A cervical lymph node biopsy was positive for AFB stain, but the culture was negative. Enzyme-linked immunosorbant assays (ELISA) of the CSF were negative for cryptococcal antigen. A magnetic resonance imaging (MRI) scan of the spine showed lumbosacral arachnoiditis. TBM with spinal arach-noiditis was diagnosed.

He was treated with anti-tuberculous drugs (isoniazid, rifampicin, pyrazinamide and ethambutol) with clinical improvement. Six months later, complete recovery was observed. His motor power improved to grade 5/5. However, one year later, he developed progressive paraparesis. He was fully conscious with a temperature of 37°C. His motor power in the legs was grade 3/5. He had generalized hyperreflexia and a positive Hoffman's sign but Babinski's sign was negative. Sensory testing revealed bilateral loss of sensation to pain, temperature and vibration below the T7 level. The other neurological signs were normal. An MRI scan of the spine demonstrated a long segment of syrinx from C4 to the conus medullaris region (Fig. 1A, B and 2).

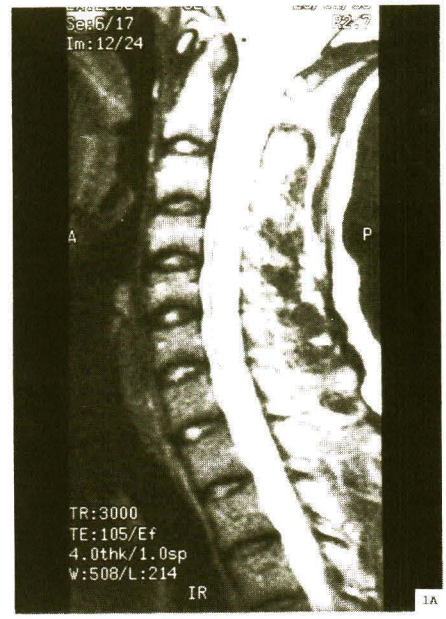

Fig. 1A. Long segment syrinx. The sagittal T2 - weighted image. The cervicothoracic spine shows mild expansion of lower cervical cord with large central syrinx cavity.

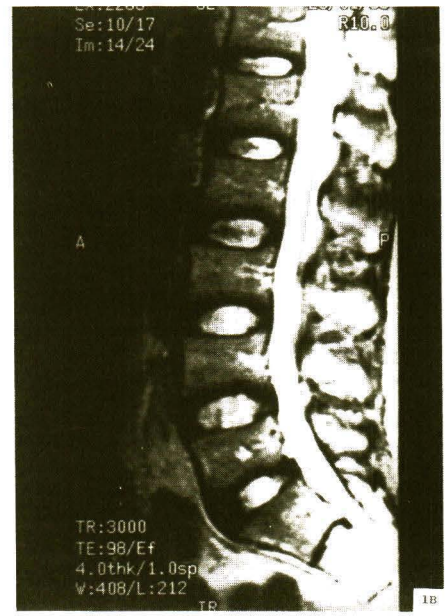

Fig. 1B. Long segment syrinx. The sagittal T2 – weighted image. The lumbar spine reveals syrinx extend to conus medullaris level.

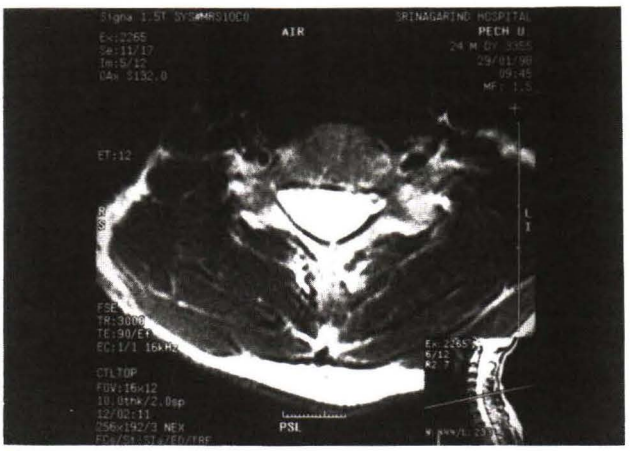

Fig. 2. Axial T2 - weighted at upper C6 lever shows high signal intensity area within the cord which indicates the presence of syrinx.

Table 1. Reported cases of syringomyelia due to tuberculous meningitis.

Reference		Case No.	Duration after tuberculous meningitis
Appleby et al.	1969[2]	1	11 years
		2	1 year
Barnett	1973[3]	3	22 years
Giminez et al.	1974[4]	4	20 years
		5	4 years
		6	6 years
		7	8 years
Savoiardo	1976[5]	8	15 years
Suzuki et al.	1985[6]	9, 10	No data
Tsuchiya et al.	1988[7]	11, 12 , 13	No data
Caplan et al.	1990[8]	14	7 years
Schon and Bowler	1990[9]	15	1 year
Fehlings and Bernstein	1991[10]	16	5 months
Schapira et al.	1992[11]	17	6 months
Daif et al.	1997[12]	18	2 weeks
		19	1 week

A T12-L1 laminectomy and syringosubarachnoid shunt were performed. The spinal cord was tense and there was surrounding adhesive fibrous tissue of the cauda equina and nerve roots. Histopathological examination of the arachnoid membrane revealed fibrosis and calcification, but the AFB stain was negative. Cultures of the biopsy specimens were also negative. The patient was transfered to a rehabilitation ward for one week postoperatively. He showed only minimal improvement. Four months after the surgery, he noticed a burning sensation in the legs. Motor power was grade 4/5, and he was able to walk with crutches.

DISCUSSION

Tuberculous vertebral osteomyelitis is a relatively common cause of myelopathy in coun-

tries where TB is prevalent. Other causes of myelopathy due to TB are less common. They include intradural or extradural solitary granuloma of the spinal cord, necrotizing granulomatous arachnoiditis with compression and inflammatory thrombosis of spinal cord vessels and syringomyelia. Medline literature review for the period 1966 to 1998, revealed 19 cases of syringomyelia due to TBM. All of the reported cases are summarized in Table 1. Because the diagnosis of syringomyelia has been facilitated by MRI and, prior to its use, physicians may have overlooked the condition, there have been only a few reported cases. The mechanisms of syrinx formation following inflammatory arachnoiditis include : a) inflammatory occlusion of spinal cord vessels leading to myelomalacia, and b) focal scar tissue causing a block in the CSF pathway, thus forcing CSF into the central canal of the spinal cord. In our case, the mechanism of syrinx formation may have been from fibrosis and calcification of the arachnoid membrane causing a block in the circulation of CSF. From the literature review, 19 cases of syringomyelia due to TBM were found. The condition occured 1 to 22 years after the acute infection in 10 cases. In the other 4 cases syringomyelia developed within one year. Our patient developed syringomyelia about 1.5 years after the acute infection. The mechanism of the syrinx formation in our case was an obstruction of the CSF pathway due to adhesive fibrous tissue around the spinal cord. The adhesive fibrous tissue was removed and a syringosubarachnoid shunt was performed. The patient improved clinically following surgery.

SUMMARY

The authors reported a case of syringomyelia due to TBM, which occurred 1.5 years after the acute infection. The mechanism of the complication may have blocked the circulation of CSF. Adhesive fibrous tissue surrounding the cauda equina was removed and a subarachnoid shunt was performed. The neurological deficit improved. Physicians should be aware of syringomyelia as a complication of TBM, because it has a good prognosis if it is treated early.

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ภาวะแทรกซ้อนโพรงน้ำในไขสันหลังเนื่องจากการติดเชื้อวัณโรคเยื่อหุ้มสมอง

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วัณโรคเยื่อหุ้มสมองเป็นวัณโรคนอกปอดที่พบบ่อย ภาวะโพรงน้ำในไขสันหลัง (syringomyelia) เป็นภาวะแทรกซ้อนของการติดเชื้อวัณโรคเยื่อหุ้มสมองที่พบได้น้อย จึงเสนอรายงานผู้ป่วยโพรงน้ำในไขสันหลังซึ่งเกิดจากวัณโรคเยื่อหุ้มสมอง 1 ราย

ผู้ป่วยชายไทยอายุ 25 ปี รับการรักษาในโรงพยาบาลด้วยอาการชาและอ่อนแรงของขา 2 ข้าง ผู้ป่วยมีประวัติเคยติดเชื้อวัณโรคเยื่อหุ้มสมองเมื่อ 1 ปีก่อน และรับการรักษาด้วยยาต้านวัณโรคครบ ตรวจร่างกายพบอุณหภูมิ 37 องศาเซลเซียส กำลังกล้ามเนื้อเกรด 3/5 รีเฟล็กซ์ไวทั่วตัว มีการสูญเสียความรู้สึกเจ็บปวด อุณหภูมิ และการสั่นสะเทือนตั้งแต่ใต้ระดับประสาทส่วนอกระดับที่ 7 ลงมา ตรวจ magnetic resonance imaging ของไขสันหลังพบโพรงน้ำไขสันหลังตั้งแต่ระดับส่วนคอระดับที่ 4 ยาวถึงส่วน conus medullaris การรักษาด้วยการผ่าตัด กระดูกบริเวณส่วนอกที่ 12 และ ส่วนเอวที่ 1 (laminectomy) ร่วมกับการทำ syringosubarachnoid shunt อาการผู้ป่วยดีขึ้นหลังจากให้การรักษา

คำสำคัญ : วัณโรคเยื่อหุ้มสมอง, ภาวะแทรกซ้อน, โพรงน้ำไขสันหลัง

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