

ORIGINAL RESEARCH

An Interesting Case of Bilateral Compressive Myelopathy In pregnancy

Dr. Renu Kumari Gupta¹, Dr. Rehana Najam², Dr. Astha Lalwani³, Dr. Shubhra Shrivastava⁴, Dr. Swati Jain⁵

¹P.G, 3rd Year, Department of Obstetrics & Gynaecology, Teerthanker Mahaveer Medical College & Research Center, Uttar Pradesh, India.

²Consultant at Sunshine Hospital Gandhi Nagar, Moradabad, Uttar Pradesh, India.

³Professor, Department of Obstetrics & Gynaecology, Teerthanker Mahaveer Medical College & Research Center, Uttar Pradesh, India.

⁴Assistant Professor, Department of Obstetrics & Gynaecology, Teerthanker Mahaveer Medical College & Research Center, Moradabad, Uttar Pradesh, India.

⁵Assistant Professor, Department of Obstetrics & Gynaecology, Teerthanker Mahaveer Medical College & Research Center, Moradabad, Uttar Pradesh, India.

Corresponding Author: Dr. Renu Kumari Gupta,
Email: ren1995gupta@gmail.com

ABSTRACT

Acute inflammatory myelopathies often known as acute myelitis are uncommon and etiologically diverse. A 26-year-old primigravida at 35+2 weeks gestation presented to casualty with complaints of inability to move lower limbs since 5 days. A diagnosis of bilateral compressive myelopathy was made for which neurosurgery opinion was taken and was advised decompressive spinal surgery for which pregnancy had to be terminated. After explaining all high risks and guarded fetomaternal prognosis patient was taken up for planned lower segment caesarean section at 36 weeks.

Keywords: Acute inflammatory myelopathies, Acute myelitis, Primigravida, Neurosurgery Decompressive spinal surgery.

INTRODUCTION

Prevalence of myelopathy during pregnancy ranges from 0.4 to 1.5 per thousand.^[2,3] making it a rare event. Acute inflammatory myelopathies, often known as acute myelitis (AM), are a nosological category that is uncommon and etiologically diverse.^[4] We differentiate between transverse myelitis and partial myelitis based on the extent of the lesion in the axial plane.^[5]

Pregnancy-related cancer is uncommon, accounting for 0.02 to 0.1% of all pregnancies.^[6]

The incidence of malignancy during pregnancy is anticipated to continue rising due to rising gestational age, which is now peaking in industrialised nations in the late 30s and early 40s.^[7]

For the patient, family, and medical personnel, this incident poses a serious difficulty. Numerous moral and professional concerns arise while attempting to strike a balance between managing a pregnancy, giving birth, and coordinating oncological and surgical therapy procedures. At the same time, striking this equilibrium places a psychological strain on the patient, her caregivers, and all engaged medical staff. These complex problems support the need for an all-encompassing multidisciplinary strategy; nevertheless, the dearth of information in the literature restricts the ability of such a management strategy to deliver evidence-based care.

Any neurologic defect connected to the spinal cord itself is referred to as a myelopathy.^[8]

The most frequent cause of myelopathy is a degenerative spinal condition. This may be caused by an osteophyte or extruded disc material in the cervical spine or, less frequently, in the thoracic spine compressing the spinal cord.^[9]

The next most frequent etiologies after degenerative are spinal cord compression brought on by extradural masses brought on by bone metastasizing disease or physical trauma. Myelopathy can also be caused by primary neoplastic, viral, inflammatory, neurodegenerative, vascular, nutritional, and idiopathic conditions.^[9]

Only approximately 15% of tumours in central nervous system malignancies develop intraspinally. 60% of these tumours are extradurally located (ED), roughly 30% are intradural extramedullarily located (ID-EM), and only 10% are real intramedullary spinal cord cancers (IMSCT). The level at which the tumour arises also has a significant impact on the clinical presentation and treatment plan, in addition to its placement in respect to the dura and spinal cord.^[10]

Meningiomas, nerve sheath tumours, as well as a wide range of other cancers and tumour imitators, are included in the well-defined category of intradural extramedullary lesions.^[11]

In areas where this modality is sufficiently accessible, using magnetic resonance imaging (MRI) to evaluate spinal lesions has become the standard of care.^[12]

Case report

A 26 years old female, presented to Casualty- Primigravida at 35+2 weeks came to casualty with chief complaints of inability to move lower limbs since 5days. It started initially with the weakness in the lower limbs since one month which progressively increased ascendingly resulting in total inability to move. Patient also complained of edema in both lower limbs which was gradual and progressive. She also gave history of loss of sensation. Patient was received with bladder catheter in situ due to urine retention and constipation with no passage of stools since 2 days. There was history of fever since 10days not associated with rigors/chills and back pain since 5days. There was no H/O weight loss, cough, trauma, haemoptysis, malaena, upper body weakness or cranial nerves deficit.

Patient also complaint of decrease fetal movements since 5days, with no significant complaints of pain, bleeding or leaking PV. She wasn't under routine ANC care.

On Examination, patient was moderately built and nourished. Vitals were stable, mild pallor was present, pedal edema upto knees, pitting type was present. Cardiovascular and Respiratory system was normal. Multiple hypo and hyperpigmented patches in reticulate and lacy pattern present on all over the body since birth, sparing the face associated with mild scaling on lower abdomen not associated with itching, pain and burning. Per abdomen, mild gaseous distention was present with uterus about 34 weeks with singleton live pregnancy in cephalic position.

Digital rectal examination: Sentinal tag + at 2 o clock position, no fissure / fistula sinus. Anal tone is normal. No ballooning. Finger soiled with fecal mater.

- On neurological examination, patient was alert and oriented.
- Tenderness present over T8- T9 level.
- No spinal deformity.
- Sensory intact throughout all dermatomes of lower limbs
- Reflexes were exaggerated in LL B/L.
- Ankle clonus present B/L
- Babinski Positive B/L.

Motor	Right	Left
Knee	increased	increased
Ankle	increased	increased
Plantar	Exaggerated	Exaggerated
Biceps	Increased	Increased
Triceps	Increased	Increased

Sensory	Right	Left
Pain	Preserved	Preserved
Dorsal		
Column	Normal	Normal

Muscle mass/Bulk Normal

Power in B/L lower limb - 1/5

	Left	Right
Hip	1/5	1/5
Knee	1/5	1/5
Ankle	1/5	1/5
Toe	1/5	1/5

Tone normal

Reflexes Left Right

Knee	++	++
Ankle	++	++
Plantar	-	-

Special test Left Right

SLR	45-50	45-50
Lasegue's	+	+
FABER	+	+

No Neurological deficit at cranial nerves was found.

Investigations

BG	A positive
Hb	9.5
TLC	12920
Platelet	3.89
KFT:	
Urea	25
Creatinine	0.56
Uric acid	5.1
LFT:	
Total bilirubin	0.9
Direct bil	0.7
Indirect bil	0.2
SGOT	62
SGPT	23
ALP	397

Urine protein 24 hour	246mg/dl
Sr TSH	1.84
Fever profile	Typhi dot positive
	Widal's ratio: 1:3

Electrolytes:	
Na	140
K	3.8
Cl	100
PT/APTT/INR	14/28/1

B/L lower limb colour doppler:
No abnormality detected



Course of stay

Orthopedic and Neurosurgeon's reference was sought. Upper motor neuron disease with Extramedullary compression myelopathy, Infective (tubercular) / neoplastic etiology was suspected hence Magnetic resonance imaging (MRI) with contrast and limb physiotherapy was advised. Meanwhile Antitubercular therapy was also started.

MRI (Dorsolateral spine): Altered signal intensity at T9 extending to Right Paravertebral space, epidural and spinal cord with associated destruction of costochondral junction and mass effect over spinal canal(right>left)? Infective tubercular osteitis/ osteoid lesion

Neurosurgeon advised decompressive spinal surgery for which pregnancy had to be terminated. After explaining all high risks and guarded fetomaternal prognosis patient was taken up for planned lower segment caesarean section at 36 weeks. Healthy male child weighing 2.66kg was delivered. LMWH was given postoperatively. Patient regained little motor function on post op day 2. Post LSCS patient was hence transferred to neurosurgeon side for the decompressive surgery.

Patient was reluctant for surgical intervention at that time hence was managed conservatively. Patient regained some motor functions after 1month and continued ATT.

DISCUSSION

Between 40 to 80 cases/million are most likely to experience spinal cord injury (SCI), both traumatic and non-traumatic. When the medulla lesion causing paraplegia is bigger than or equivalent to T6, it is thought that a reflex autonomic hyperactivity (HRA) condition may be triggered, which is related to the severity of paraplegia-related pregnancy.^[15]

The likelihood of a quadriplegic or paraplegic mother giving birth to a child is estimated to be 1.5 births per thousand.

This syndrome is characterised by an overactive sympathetic nervous system brought on by cutaneous or visceral stimulation below the damage threshold. It may be the cause of thrust hypertension, which may lead to mortality or serious cardiovascular and neurological damage.^[16]

Patients with damage levels above T10–T11 do not experience uterine contraction discomfort and are not at risk for developing severe dysautonomia. Patients with injuries that are more severe than T10–T11 do not experience pain associated with uterine contractions and are not at risk for severe dysautonomia.

The danger of disregarding the threat of a preterm birth is potentially greater in this situation. Acute myelitis is diagnosed using clinical criteria, which may include be paraneoplastic, inflammatory, or infectious. When an onset occurs within a short period of time, the qualifier "acute" is added. The length of "acute myelopathy "is severe spinal cord injury, without assuming the nature of the harm. Between nine and ten million new cases of tuberculosis are recorded each year, and approximately 1.8 billion individuals are affected, according to WHO estimates. People from emerging and developing nations in particular are affected by this.^[19,20,21]

The arterial system is most frequently used to access the spine; as a result, pathogens enter through the terminal arterioles and appear in the vertebral body more anteriorly and typically with early paradiscal symptoms.^[22]

Spread across the venous plexus is less common, and in this scenario, the infection foci are more in the middle of the vertebra.^[23,24,25]

Clinical symptoms, such as those connected to nonspecific pyogenic spondylodiscitis.^[26] indicate spinal involvement. Back pain is one of them; it tends to be local but can also be radicular or pseudo radicular.^[27,28] Many people describe the pain as being deeply seated and unrelated to load. Muscle tightness and restricted mobility are frequently noted.^[29,30]

Poverty, overcrowding, illiteracy, malnutrition, alcoholism, drug abuse, diabetes mellitus, and immunosuppressive drugs are risk factors for tuberculosis.

Conventional radiographs provide a decent overall picture, whereas computed tomography (CT) can show disco-vertebral lesions and paravertebral abscesses, and magnetic resonance imaging (MRI) can show whether the disease has spread to soft tissues and how much of the spinal cord is affected.^[32]

The World Health Organization (WHO) suggests treating tuberculosis according to a category. The WHO treatment category for spinal TB is category 1. Two phases make up the category-1 antituberculosis treatment plan: an intensive (initial) phase and a continuing phase. Antituberculous therapy for tuberculosis includes a combination of four first-line medications during the two-month intense phase: isoniazid, rifampicin, streptomycin, and pyrazinamide. Two medications (isoniazid and rifampicin) are administered for 4 months during the continuation phase. The WHO advises 9 months of treatment for tuberculosis of the bones or joints due to the significant risk of disability and mortality, as well as the challenges in evaluating treatment response.^[33]

Urinary tract infections, which are made more likely by routine self-catheterization, thrombophlebitis risk, and bedsores are the most common pregnancy complications.^[34,35]

CONCLUSION

Paraplegia is rare during pregnancy but poses diagnostic, therapeutic and monitoring issues. Any compressive myelopathy due to any cause ought to be decompressed surgically and timely to prevent future damage in the surrounding vital structures. In our patient, she improved conservatively by ATT and terminating the pregnancy, but sometimes surgery is the mainstay of treatment. Multidisciplinary care with early diagnosis and timely intervention is the key to treatment in such cases. Tuberculosis should always be kept in differentials especially in developing country like our's.

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