

CASE PRESENTATION

HPI: 46 Y/O RHAAF with past medical history significant for drug abuse, lower back sprain in 2002 was seen in a local hospital ER for lower back pain x 2 days, she was treated with pain meds and ? Epidural injection with steroids and discharged to home as pain improved. Next morning she woke up with no movement in bilateral lower extremities and also no sensations, she was also incontinent of bowel and bladder she came back to the hospital and was admitted for further work up.

ALLERGIES: NKDA.

ACTIVE MEDS: ZYRTEC, MOTRIN
PRN FOR PAIN.

MARITAL STATUS: MARRIED, LIVES
WITH MOTHER.

FAMILY HISTORY: SISTER DIED OF AIDS
SISTER LIVING WITH
AIDS.

- SOCIAL HISTORY

- SMOKES : CIG 1 PPD.

- MARIJUANA: 4 JOINTS DAILY.

- ALCOHOL: BEER/LIQUOR 12
PACK/DAY.

- HAVE USED COCAINE 4-5 TIMES ONLY

- **ROS:**

- + FOR BILATERAL LOWER EXTREMITY WEAKNESS AND NUMBNESS.

- + FOR BOWEL AND BLADDER INCONTINENCE.

■ EXAMINATION:

■ GENERAL:

- UNREMARKABLE, EXCEPT BILATERAL LOWER EXT EDEMA. EXTREMITIES WARM AND PULSES PALPABLE.

■ NEUROLOGICAL:

- MENTAL STATUS: NORMAL

- CN II – XII: INTACT

- Motor:

RUE/LUE:	D	T	B	WE	WF	FE	G	I
	5	5	5	5	5	5	5	5

RLE	:	HF	KE	KF	DF	PF
		3	3	3	4	4

LLE	:	0	0	0	0	0
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Reflexes

UPPER RT & LT

Biceps	Triceps	Brachioradialis
+1	+1	+1

Lower extremity Rt and Left.

Patellar	Achilles	Toes
2+	2	mute

Coordination:

Finger-Nose : nml

Hell/knee/shin: unable to test secondary to severe weakness

Rapid-alternating: nml

Gait: unable to walk.

Data reviewed:

BMP: Na 135 K 4.3 BUN 21 Cr. 0.4

CBC :WBC 12.1 HB 12 PLt. 36 ESR 48

LFT:Albumin 3.3 TP 6.3 AST 58 ALT 96 ALP 176

HCV +ve , HC viral load 334,838

LP: MBP 4.1 , -ve OCB, elevated IgG index

Imaging:

MRI brain-nml

MRI c-spine- c5-c6 mild left foramina stenosis secondary to disc herniation.

MRI L spine mild disc bulging at level of L4-L5 and L5-S1

MRI T spine Intramedullary lesion from upper end plate of T10-T11.



Assessment And Plan

46 y/o RHAAF with Pm hx significant for IV drug abuse, recently found to be HCV +ve with mild to moderate increase in liver enzymes and severe thrombocytopenia. LP results were significant for elevated wbc mostly lymphocytes, nml glucose and protien, -ve OCB and elevated IgG index. Relevant neuro-exam → absent sensation to all modalities below T11.

Differential Diagnosis

- Transverse myelitis
- Intramedullary tumor
- Epidural hematoma secondary to epidural block given on initial ER visit.

Introduction of Discussion

Hepatitis C virus (HCV), a single stranded RNA virus of the flaviviridae family is a major cause of chronic liver disease, affecting approximately 2.7 million people in the United States. The association of HCV with demyelinating myelitis is not well established. In this case other infectious and autoimmune etiologies were excluded.

Diagnostic Criteria for TM

- Sensory, motor or autonomic dysfunction attributable to the spinal cord. Bilateral signs and / or symptoms clearly defined sensory level inflammation defined by CSF pleocytosis or elevated IgG index or gadolinium enhancement. Progression to nadir between 4 hours and 21 days.

DISCUSSION

- My impression in this case is myelitis secondary to chronic HCV. Evaluation for other infectious or autoimmune causes of myelitis was unrevealing. Multiple Sclerosis is unlikely given the absence of typical brain lesions on MRI and negative oligoclonal bands in CSF. These observations suggest that myelitis in this patient is etiologically related to HCV infection, possibly via immune-mediated mechanism. Both direct viral invasion and immune – mediated mechanics have been implicated in the pathogenesis of HCV associated myelitis and HCV-associated .Case have been reported in which HCV RNA in the CSF suggesting the possibility of direct viral invasion (unfortunately in this case we were unable to check HCV RNA in csf).

TREATMENT OPTIONS

- Intravenous Steroids.

Intravenous steroids treatment is often instituted for patients with acute TM. Corticosteroids have multiple mechanisms of action including anti-inflammatory activity, immunosuppressive properties and antiproliferative actions.

- In the steroid-treated group, the median time to walking was 23 days versus 97 days, full recovery occurred in 80 % vs 10 % and full motor recovery at 1 year was present in 100 % vs 20 %.
- Plasma Exchange (PLEX)

TREATMENT OPTIONS

- PLEX is often initiated if a patient has moderate to severe TM (i.e, inability to walk, markedly impaired autonomic function and sensory loss in the lower extremities) and exhibits little clinical improvement after instituting 5-7 days of intravenous steroids. PLEX is believed to work in auto-immune CNS diseases through the removal of specific or non specific soluble factors likely to mediate, be responsible for or contribute to inflammatory- mediated target organ damage. Predictors of good response to PLEX include early treatment (less than 20 days from symptoms onset) male sex, and a clinically incomplete lesion (some motor function in the lower extremities , intact or brisk reflexes). PLEX may significantly improve outcomes of patients with severe (though incomplete) TM and who have not significantly improved on IV steroids.

TREATMENT OPTIONS

- Other immunomodulatory treatment.
 - No controlled information currently exists regarding the use of other treatment strategies in patients with acute TM. Some clinicians consider pulse dose intravenous cyclo-phosphamide (500-1000 mg/m²) for patients with TM that continues to progress despite IV steroids therapy.
 - CSF filtration is a new therapy, in which spinal fluid is filtered for inflammatory factors including cells, compliment, cytokines, and antibodies prior to being re infused into the patient.
 - chronic immunomodulatory therapy should be considered for the small sub group of patients with recurrent TM.
- Long term management
- Many patients with TM will require rehabilitative care to prevent secondary complications of immobility and to improve their functional skills.

QUESTIONS

1. 40 Y/O OTHERWISE HEALTHY FEMALE CAME TO ER WITH ACUTE ONSET OF BILATERAL LOWER EXTREMITY WEAKNESS, SEVERE SENSORY DEFICIT RT > LT, WITH BOWEL AND BLADDER INCONTINENCE.
 - WHAT OTHER HISTORY IS IMPORTANT IN THIS CASE.
 - 1. MALIGNANCY.
 - 2. MULTIPLE SCLEROSIS.
 - 3. RECENT TRAUMA OR FALL.
 - 4. AUTOIMMUNE DISORDERS.
 - All of the above

2. WHAT WILL BE YOUR CHOICE OF IMAGING IN THE ABOVE MENTIONED CASE.

1. X-RAY L/S SPINE.
2. CT SPINE WITH AND WITH OUT CONTRAST.
3. MRI OF THORACIC AND L/S SPINE.
4. MRI / MRA OF HEAD AND NECK.

3. YOU RECENTLY DIAGNOSED ONE OF YOUR PATIENTS WITH TRANSEVERSE MYELITIS OF UNCLEAR ETIOLOGY, TREATED WITH HIGH DOSE STEROIDS . THERE WAS INITIAL RECOVERY PHASE FOR ABOUT 1 WEEK IN WHICH PATIENT STRATED WALKING WITH ASSISTANCE,NOW SHE SUDDENLY HAS DECLINE IN HER CONDITION. WHAT WOULD BE YOUR NEXT STEP.

1. REPEAT IMAGING ASAP TO R/O EPIDURAL HEMATOMA.
2. PERFORM ANOTHER L.P
3. INCREASE THE DOSE OF STEROIDS.
4. PLEX TREATMENT FOR RECURRENT TM
5. PSYCH CONSULT FOR POSSIBLE PSYCHOLOGICAL SYMPTOMS.

4. WHEN WOULD YOU CONSIDER PLEX AS A TREATMENT OPTION FOR YOUR PATIENT, WHO IS JUST DIAGNOSED WITH TM.

1. AS SOON AS YOU MADE THE DIAGGNOSIS OF TM.
2. IF PATIENT HAS MILD TM, i.e. ABLE TO WALK WITH ASSISTANCE AND GOOD BOWEL AND BLADDER CONTROL.
3. IF PATIENT EXHIBITS LITTLE OR NO IMPROVEMENT AFTER INSTITUTING 5-7 DAYS OF IV STEROIDS.
4. IF PATIENT HAS MODERATE TO SEVERE TM, i.e. BOWEL AND BLADDER INCONTINENECE AND UNABLE TO WALK.
5. IF PATIENT BECAME FEBRILE.