One-and-a-Half Syndrome : An Unusual Cause of Dizziness

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One-and-a-Half Syndrome: An Unusual Cause of Dizziness

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Introduction and Learning Objectives

Introduction: This Case Report describes a rare cause of dizziness: One-and-a half syndrome

Learning Objectives:

- Describe the clinical findings in a patient with one-and –a-half syndrome
- Outline the neurological basis for this syndrome
- Describe the likely clinical outcome for such patients

Case Description

Chief Complaint: Dizziness, double vision and imbalance

History of Prsent Illness: Patient is an 80 year old woman who reported an onset of imbalance on the night prior to presentation. She noted that while lying still in bed she felt fine, but that on turning her eyes she saw double. She attempted to walk but was extremely unbalanced to the point where she had to hold onto things. Her husband, who is a retired physician, checked her blood pressure and found it to be normal. The patient went back to bed and slept. The following morning when she awoke, she still had similar symptoms and decided to go to the Emergency Room.

On evaluation in the Emergency Room, she was still complaining of double vision on eye movement, dizziness on turning her head and feeling extremely unbalanced on attempting ambulation. She had no headache, weakness, numbness, other sensory disturbance or fever.

Past Medical History

- Hypertension
- Degenerative Joint Disease
- Osteopenia
- Hemochromatosis
- Lumbar fadiculopathy

Past Surgical History

- Tonsillectomy
- Appendectomy
- Hysterectomy
- Cystocele repair
- R total knee replacement

Medications: Amlodipine 5 mg every other day

Allergies: NKDA

Review of Systems: A 12 point review of systems was negative except for diplopia, dizziness and gait imbalance.

Family History: Mother died of cancer in her 70s but also had a history of stroke

Social History: Married with 7 children. Retired homemaker. Completely independent in ADLs and IADLs prior to hospitalization. Recently moved to independent living in a CCRC. Non-smoker for many years. Occasional alcohol use.

Case Description (continued)

Physical Examination: Temperature 36.7 °C, Pulse 84, Blood pressure 144/75, respiratory rate 16, pulse ox 96% on 2 liters of oxygen. No carotid bruit, no cyanosis, clubbing or edema. Heart regular rate and rhythm no murmur. Lungs clear. Abdomen soft, non-tender, normal bowel sounds.

Neuro Examination: Alert and oriented to person, place and time, excellent historian. Trifocal glasses. Pupils equal and reactive to light. Complete paralysis of horizontal gaze in the right eye, only able to abduct left eye. Face symmetric with no facial muscle weakness. Facial sensation is symmetric and intact. Uvula midline and gag reflex intact. Tongue protrudes midline. Motor examination shows full strength and no drift or fix. Deep tendon reflexes 1+ throughout except for R knee related to prior surgery. Babinski downgoing bilaterally. Sensory examination intact. Testing of coordination revealed no dysmetria. No ataxia. Gait was not assessed.

Diagnostic Studies: CBC, BMP, lipid profile, coagulation studies and troponin were all within the normal range.

CT head showed chronic micro-vascular ischemia with no acute intracranial pathology. Carotid ultrasound showed no hemodynamically significant stenosis.

MRI was initially reported as showing only chronic microvascular changes. However after further history was given it was reviewed and reported as:

"Reviewing diffusion weighted imaging there is an extremely small vague focus of questionable minimally bright diffusion signal in the right posterior pontine region immediately ventral to the slightly right-sided margin of the fourth ventricle. While this extremely subtle and questionable focus of slightly increased diffusion weighted signal could certainly reflect artifact, there does appear to be some preferential punctate T2 signal abnormality as well on image 6 of series 801 which slightly stands out above extensive background of chronic pontine microvascular ischemic change. It is further noted that the area of abnormality may either involve the area of the right medial longitudinal fasciculus (MLF) and/or potentially the right nerve VI nucleus region, and therefore this would seem to correlate with the patient's symptoms."

Hospital Course: Patient was admitted and seen in consultation by neurology. The neurologist diagnosed one-and-a- half syndrome consistent with complete paralysis of horizontal gaze in one eye, and preservation of abduction in the contralateral eye.

Given the acute onset of her symptoms, it was considered most likely a cerebrovascular accident. Patient was started on Aspirin 325mg daily. Her LDL was normal at 113. She was given an eye patch to be worn over her right eye as this was the eye with no horizontal movement.

Post Hospital Course: Patient was admitted from the medical floor to the inpatient rehabilitation unit. She did well with physical therapy. She was able to ambulate with a rolling walker without which she tended to list to the left, and had difficulty with changes in direction. She experienced difficulties with high level balance activities and required use of the eye patch to reduce her dizziness.

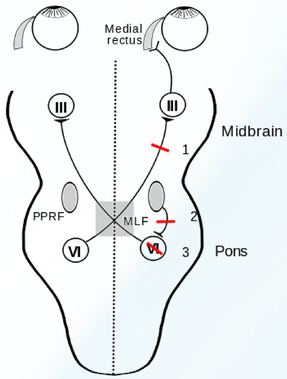
The patient was discharged back to independent living at the CCRC after a week stay in in-patient rehab. She was felt to be safe at home with the assistance of her husband.

Discussion

The "one –and-a-half syndrome" was first described by Dr. C. Miller Fisher[1] in a 1967 in a paper describing little known neuro-opthalmological observations made on the Stroke Service of the Massachusetts General Hospital. He described it as "a conjugate lateral gaze palsy in one direction, plus one half of a gaze palsy in the other."

It presents as complete (bilateral) gaze palsy in one direction (looking to the side of the lesion), and one half (unilateral) gaze palsy in the other direction (looking away from the lesion).

The syndrome is usually caused by a single unilateral lesion of the paramedian pontine reticular formation, or the abducens nucleus on one side (causing the conjugate gaze paresis to the side of the lesion), with an interruption of the internuclear fibers of the ipsilateral medial longitudinal fasciculus after it has crossed the midline from its site of origin in the contralateral abducens nucleus (causing failure of abduction of the ipsilateral eye.)



Source: http://en.wikipedia.org/wiki/File: One and a half syndrome.svg

In a study of causes of internuclear opthalmoplegia (INO) published by Bolanos et al in 2004[2], INO was described as unilateral in 55.4%, bilateral in 33.8% and one –and-a-half syndrome in 10.8%. Causes for the one and a half syndrome were most frequently vascular (infarct and hemorrhage), demyelinating and neoplastic. For patients with unilateral or bilateral INO other causes such as traumatic and infectious were found, with neurocysticercosis and brainstem encephalitis being the most common infectious causes. The study showed that INO has a favorable outcome with complete recovery in 49.2% of cases. Most of the complete recoveries occurred during the first 6 months after presentation. Infectious and demyelinating causes had a better outcome.

The MRI in this case was initially described as normal but after review by the radiologist with a clinical history of one and a half syndrome, the interpretation was revised to reflect a subacute punctate pontine infarct potentially involving the region of the right medial longitudinal fasciculus and/or the right nerve VI nucleus region.

Etiology of Internuclear Opthalmoplegia[2]

	Unilateral N = 36	Bilateral N = 22	One and Half Syndrome N = 7
Vascular	14	6	4
Demyelinating	10	9	2
Infectious	6	3	0
Traumatic	3	2	0
Neopalstic	2	1	1
Other	1	1	0

Prognosis: A review of INO as an isolated or predominant symptom of brain stem infarction by Kim in 2004[3] found good outcomes for patients with isolated INO as in our case. Diplopia and dizziness were the most common presenting features, with sensory symptoms reported in a few patients. In this study, there was 100% resolution of symptoms within one month.

References:

1. C. Miller Fisher. Some Neuro-opthalmological Observations. *J.Neurol. Neurosurg.Psychiatr.*, 1967, 30, 383-394.
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3. Kim JS Internuclear opthalmoplegia as an isolated or predominant symptom of brainstem infarction. *Neurology* 2004: 62: 1491-1496.

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