Alcoholic Cerebellar Degeneration
Clinical Syndrome

The clinical syndrome of alcoholic cerebellar degeneration is remarkably stereotyped. The usual presentation, as in this patient, is a progressive unsteadiness in walking evolving over months and years.
Clinical Syndrome

The cerebellar syndrome predominantly affects stance, eye movements, and gait, sometimes with trunkal ataxia and titubation. Dysarthria and upper limb ataxia are rare.
Pathophysiology

Ataxia may develop during periods of abstinence.

Identical cerebellar degeneration has been observed in non-alcoholic patients with severe malnutrition.
Eye Movements

Square Wave Jerks
Horizontal Saccadic Hypermetria
Horizontal Gaze Evoked Nystagmus
Saccadic Pursuit
Deficits Caused by Lesions of Dorsal Vermis, Fastigial Nucleus, and Uncinate Fasciculus

Dorsal Vermis Lesion
- Ipsilateral hypometria and mild contralateral hypermetria of saccades
- Gaze is tonically deviated away from the side of the lesion
- Smooth pursuit is impaired for targets moving towards the side of the lesion

Unilateral Fastigial Nucleus Lesion†
- Ipsilateral hypermetria and contralateral hypometria of saccades – “ipsipulsion”
- Gaze is tonically deviated towards the side of the lesion
- Smooth pursuit is impaired for targets moving away from the side of the lesion
- Similar defects are features of Wallenberg’s syndrome

Uncinate Fasciculus (Which Runs In Superior Cerebellar Peduncle)
- Ipsilateral hypometria and contralateral hypermetria of saccades – “contrapulsion”

Pathological Changes

Selective atrophy of the anterior and superior parts of the cerebellar vermis
Involvement of the cerebellar hemispheres less extensive
Loss of neurons in the cerebellum involves all types but Purkinje’s cells are the most seriously affected
Figure 1: Sagittal T1WI shows striking atrophy of the superior vermis.
MRI Findings

Figure 2: Axial T2WI through the upper midbrain and vermis shows the cerebellar folia are thinned and the CSF spaces increased.

Courtesy Anne Osborn, M.D