

Wernicke encephalopathy

Wernicke encephalopathy is a severe neurologic disorder resulting from dietary vitamin B₁ deficiency

WE presents with the classic triad of ocular findings, cerebellar dysfunction, and confusion

Signs and symptoms

- Acute confusion
- Delirium
- Ataxia
- Ophthalmoplegia
- Memory disturbance
- Hypothermia with hypotension
- Delirium tremens

Etiology

Alcohol abuse, AIDS, malignancy, hyperemesis gravidarum, prolonged total parenteral nutrition, iatrogenic glucose loading in a thiamine deficient patient

Bariatric surgery, of which there are more than 100,000 weight-loss procedures performed annually in the United States, has been associated with both malnutrition and WE

Emergency Department Care

Thiamine (Vit B1) Amp IV 200 mg thrice daily





Neurobion Amp IM 3cc= 100mg B1



B complex Amp IM/IV 2cc= 10mg B1

Note

Patients with WE are likely **hypomagnesemic** and should be treated empirically with parenteral magnesium sulfate, as they may be unresponsive to parenteral thiamine in the presence of hypomagnesemia

Note

Administering dextrose to an individual in a thiamine-deficient state exacerbates the process of cell death

Radiographic features

In acute stages, hemorrhage, necrosis, and edema may be present

In chronic stages, atrophic changes may be present especially involving the mamillary bodies

CT scan is Usually normal

MRI

T2/FLAIR: symmetrically increased signal intensity in the

- Mammillary bodies
- Dorsomedial thalami
- Tectal plate
- Periaqueductal grey matter
- Around the third ventricle

DWI/ADC: restricted diffusion can also be seen in the same regions

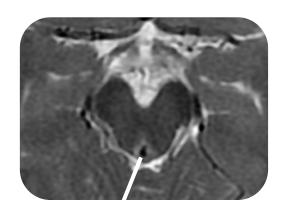
MRI with contrast

Symmetric hypointensity or no abnormalities on T1weighted images

T1 C+ (Gd): Contrast enhancement can also be seen in the same regions, most commonly of the mammillary bodies

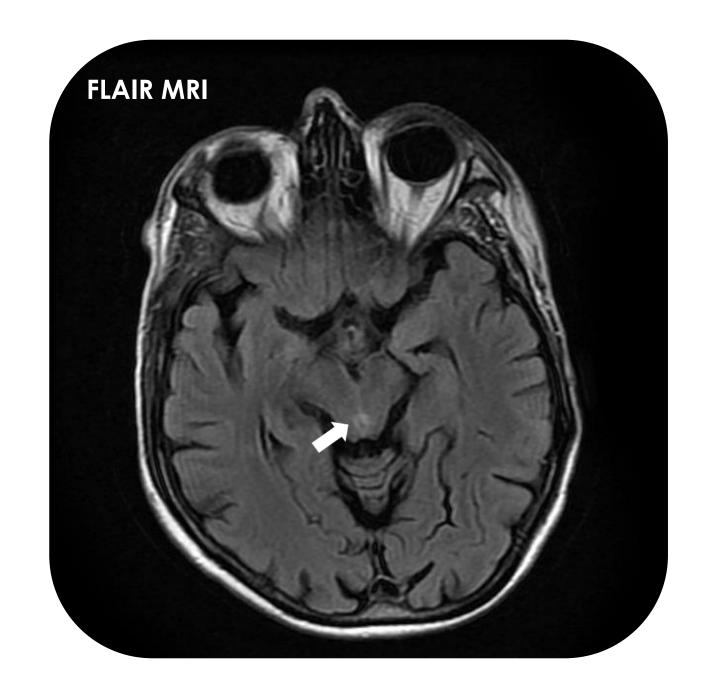
Atypical Findings on MRI

Symmetric hyperintensity of CNN, cerebellum, and supratentorial brain cortex



Aqueduct

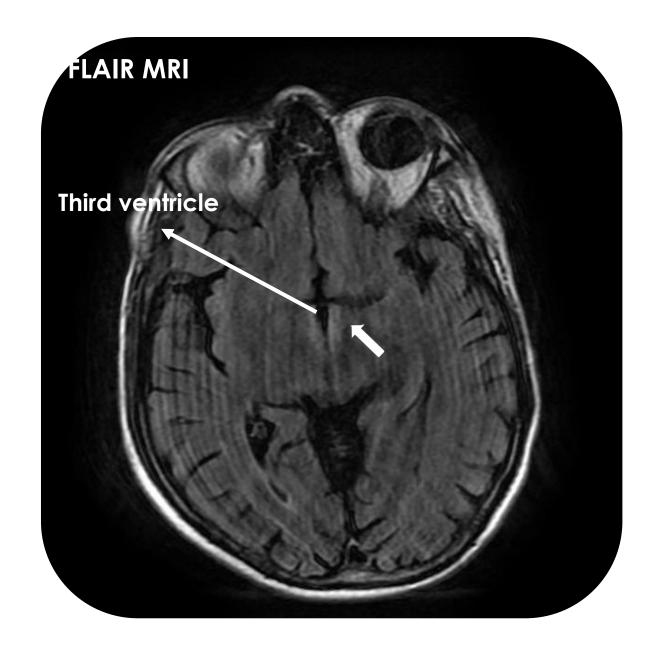
Signal intensity in Periaqueductal grey matter



Signal intensity in Periaqueductal grey matter

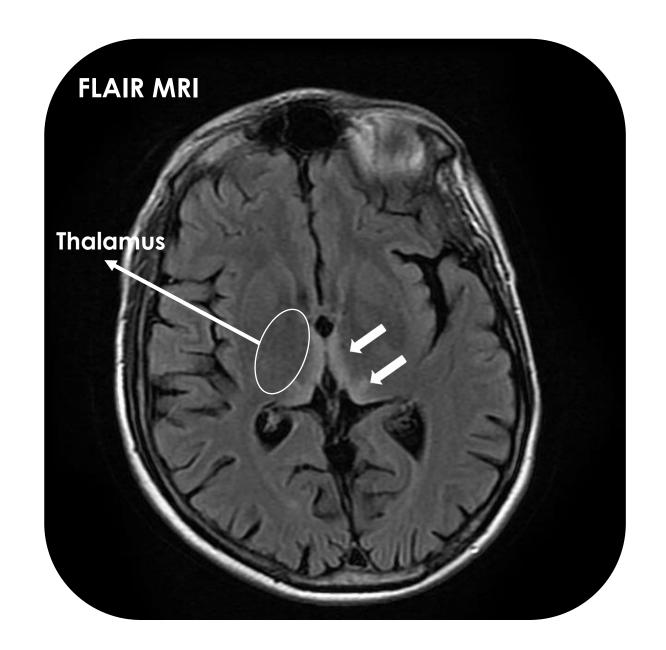


Signal intensity **around** third ventricle



Signal intensity in

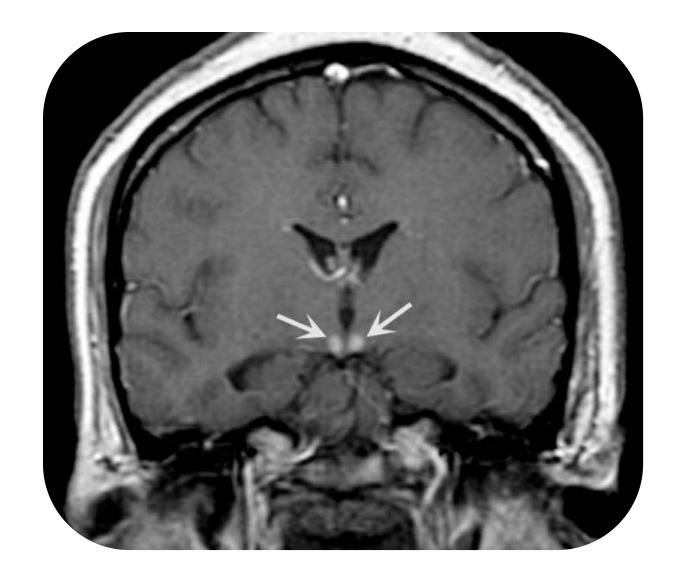
Dorsomedial thalami and around third ventricle



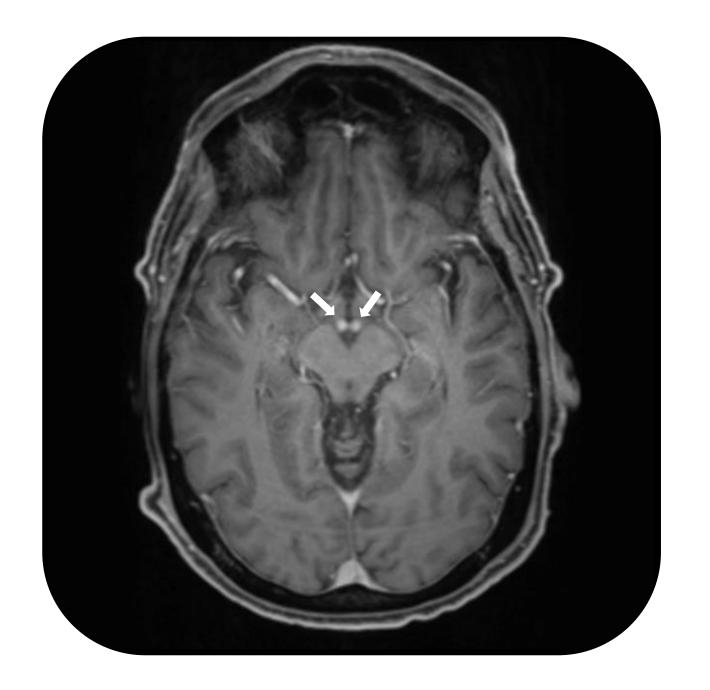
Signal-intensity alterations of the medial thalami and periventricular region of the third ventricle



Bilateral mammillary body signal abnormality with contrast enhancement

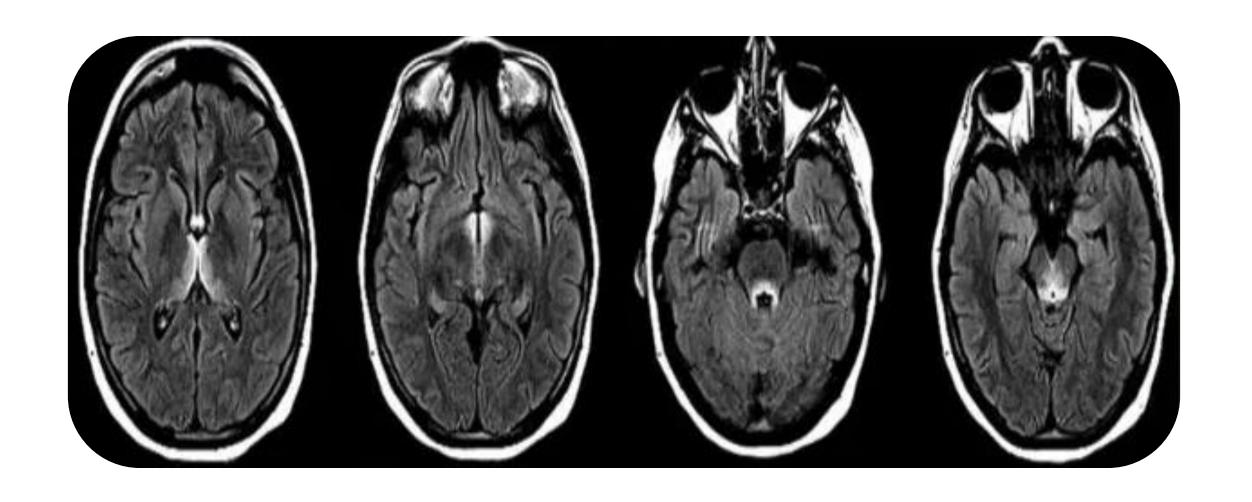


Bilateral mammillary body signal abnormality with contrast enhancement

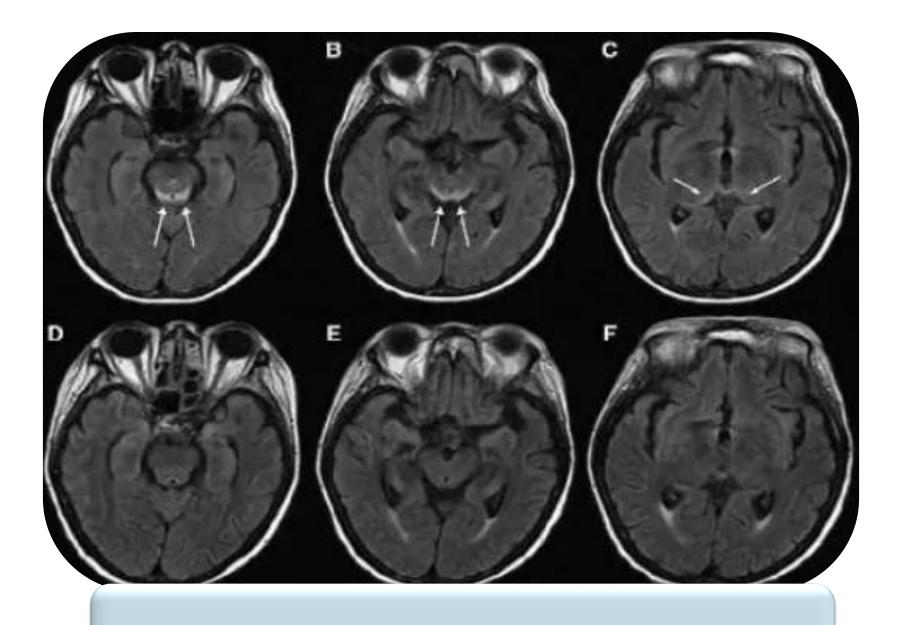


Signal intensity in medial thalami and around Third Ventricle





MRI of 16 week pregnant women with recent Mental Status Change, repeated vomiting and weight loss

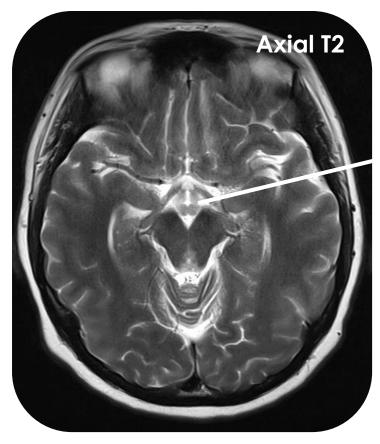


MRI before and after Thiamine administration

Case Review

The patient presented with symptoms of ataxia, nystagmus and vertical gaze palsy. The patient was awake, NIHSS = 0, although disorientated to time and space

The patient underwent bariatric surgery 2 months before presentation (sleeve gastrectomy) which led to the neurological deficits in this case of thiamine deficiency-induced Wernicke encephalopathy



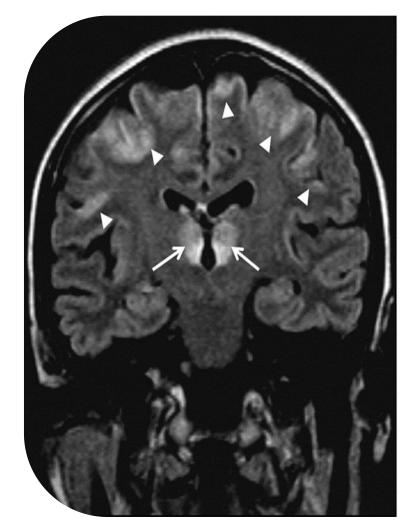
Mammillary bodies



A, Signal-intensity alterations with different intensity patterns in the thalami (arrows). Diffuse signal-intensity alterations of the frontal cortex (arrowheads)

B. signal-intensity alterations in the mamillary bodies (arrows), periventricular region of the third ventricle (empty arrows), and brain cortex (arrowheads)

A 54-year-old woman with a history of food refusal had changes in consciousness

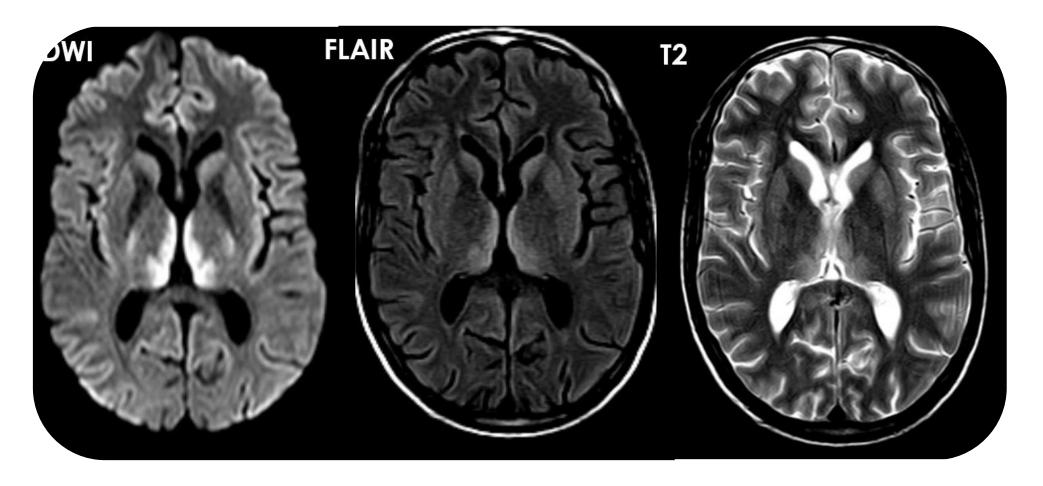




Alterations in the tectal plate (white arrows) and mamillary bodies (white arrowheads) are seen

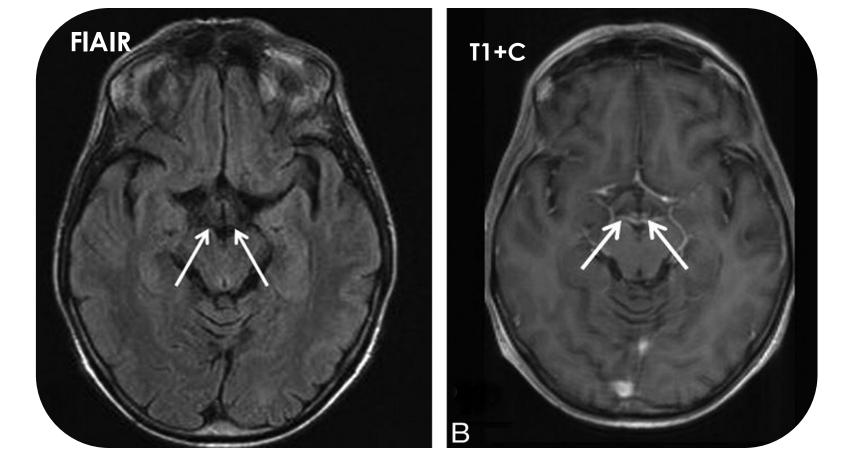
A 21-year-old woman presented with hyperemesis gravidarum, changes in consciousness, and ocular abnormalities





Abnormal signal bilateral and symmetrical involving the **medial** and posterior aspect of both thalami displaying bright signal in DWI, T2 and FLAIR

Deterioration of conscious level and agitation. Patient had gastric surgery 2 months ago for peptic ulcer



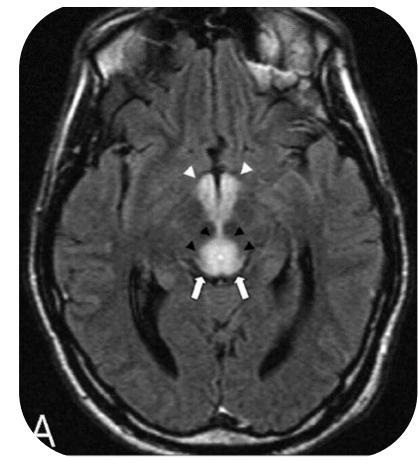
A 47-year-old woman with a history of alcohol abuse presented with ataxia, changes in consciousness, and ocular abnormalities

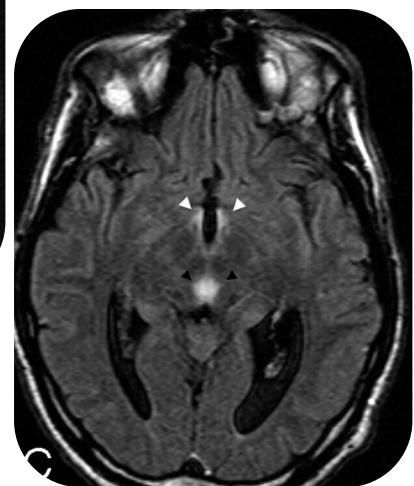
- A, No signal-intensity alteration are seen at the mamillary body
- B, Contrast enhancement is seen in the mamillary bodies

A 33-year-old man presented with sudden and progressive changes in consciousness after prolonged voluntary food starvation

A, hyperintensity of tectal region (white arrows), periaqueductal area (black arrowheads), and mamillary bodies (white arrowheads)

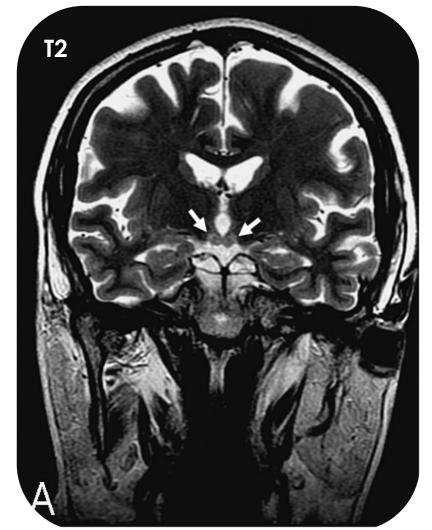
C, 10 days after thiamine replacement, a partial regression of the lesions is easily seen

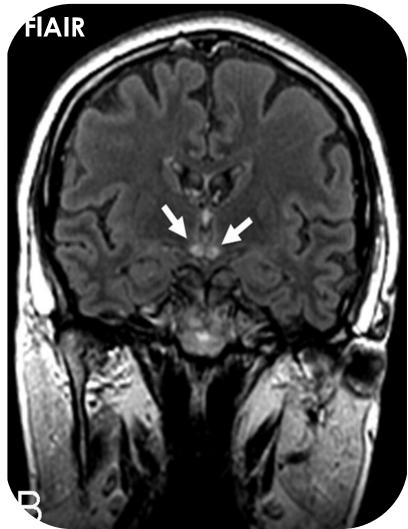




A 53-year-old woman with an history of chronic alcohol abuse presented with the classic neurologic triad of Wernicke encephalopathy

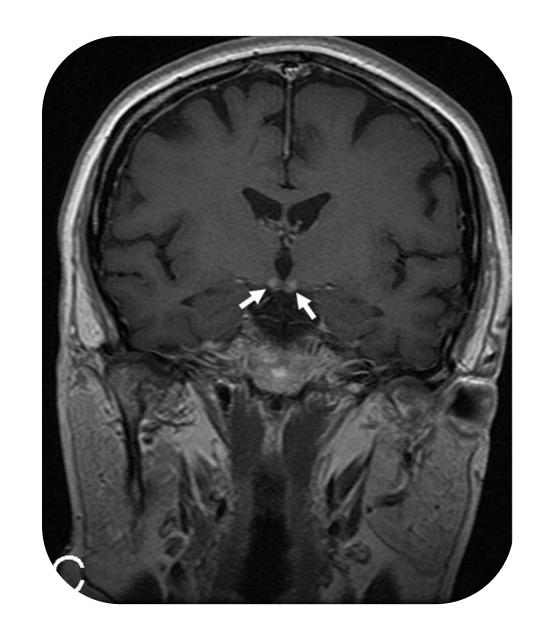
A, B. high signal intensity mamillary bodies





Central enhancement of both mamillary bodies is seen on coronal T1+C

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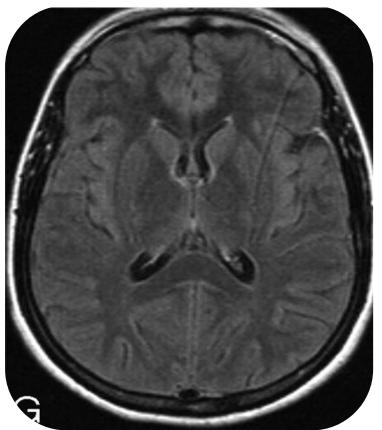


of the basal ganglia shows alterations of the medial thalamic nuclei

12 days after the start of thiamine replacement therapy, regression of neurologic symptoms and FLAIR abnormalities is seen

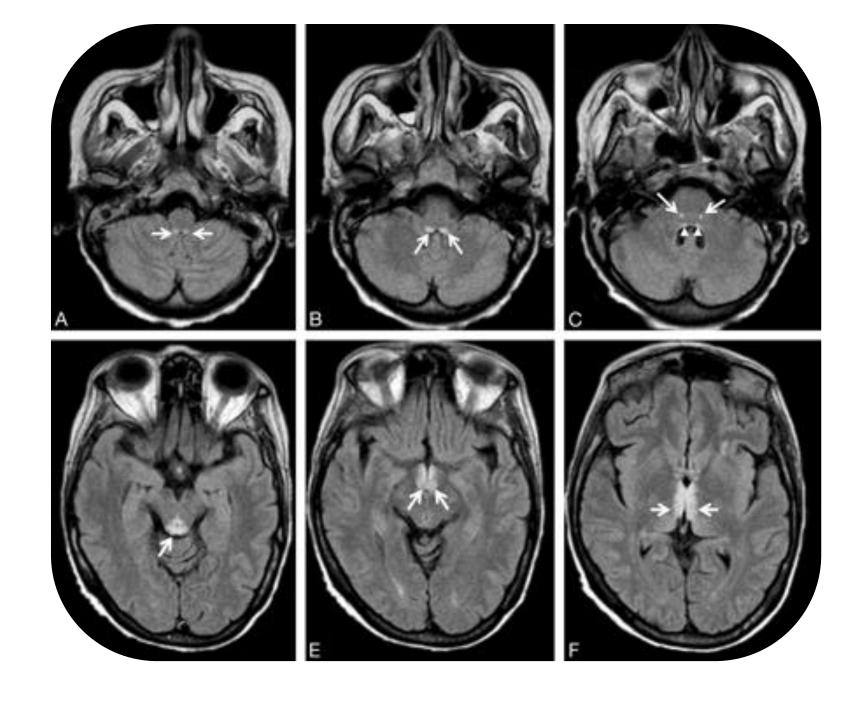
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Symmetric
hyperintense lesions
in hypoglossal
nuclei, medial
vestibular nuclei,
facial nuclei

A 54-year-old woman with leukemia, changes in consciousness, and ataxia



Thank You For Your Attention