

CASE PRESENTATION

History of present illness:

48-year-old female with gait ataxia and dizziness after left cerebellar stroke resulting from vertebral artery occlusion.

Background:

- 48-year-old female with no past medical history presented acute headache, nausea, and intractable vomiting for 3 days.
- CT showed acute/subacute left cerebellar infarct
- CTA showed occlusion of vertebral artery close to origin, reconstitution distally.
- Neurology hypercoagulable workup including prothrombin mutation, protein S, protein C, AT III, Silica clotting time, Jak-2 mutation, Factor V Leiden, Homocysteine, Lipoprotein A, Cardiolipin Abs, DRVVT, COVID PCR and COVID Abs was negative except positive b2 glycoprotein and negative Beta-2-glycoprotein (IgA, IgG, IgM)

Past Medical History: none

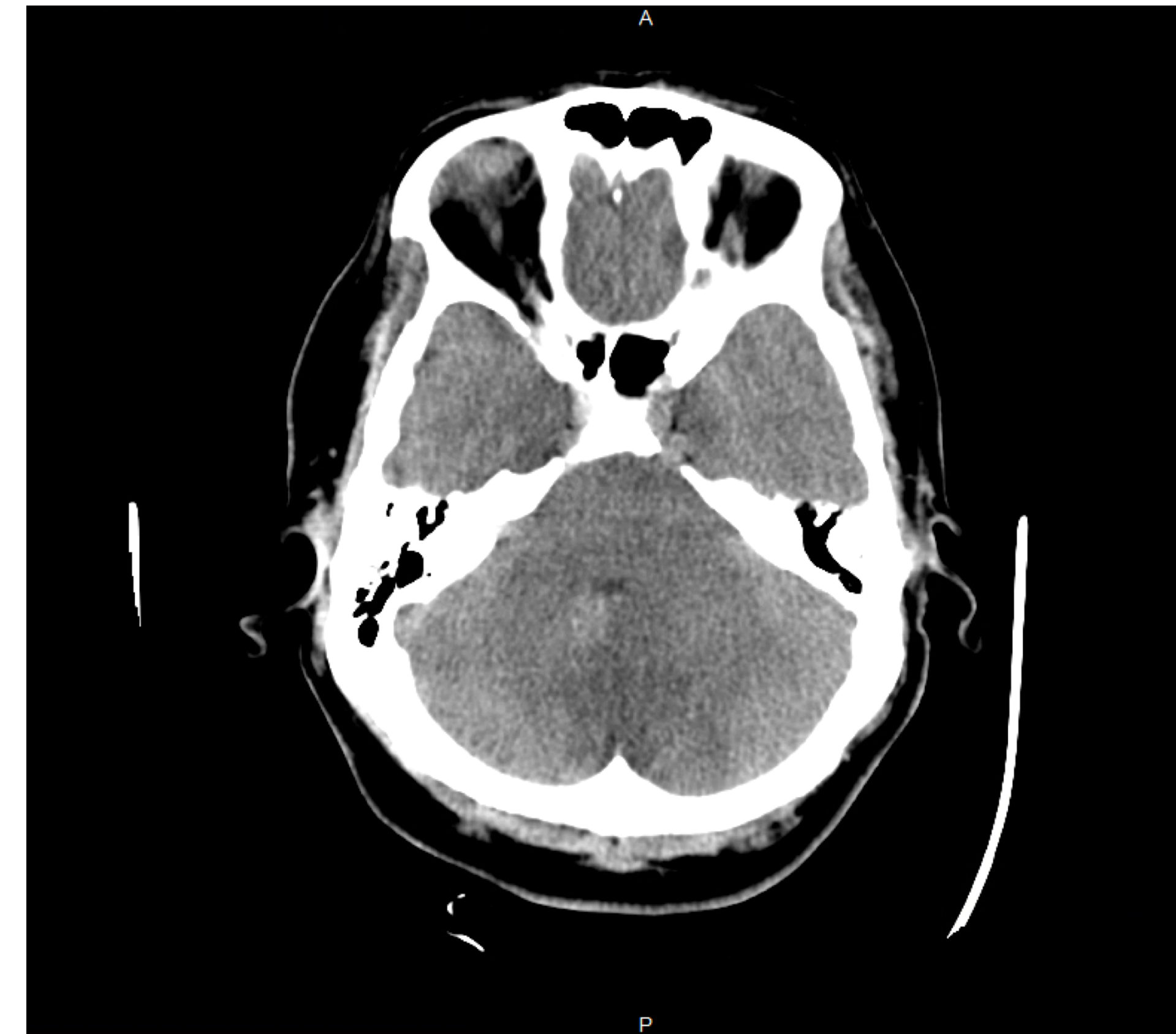
Physical exam:

- Full strength, normal sensation and proprioception
- Intact cranial nerves
- Mild dysdiadokinesia on the left
- Gait: decreased step length, frequent pauses
- Negative for dysmetria

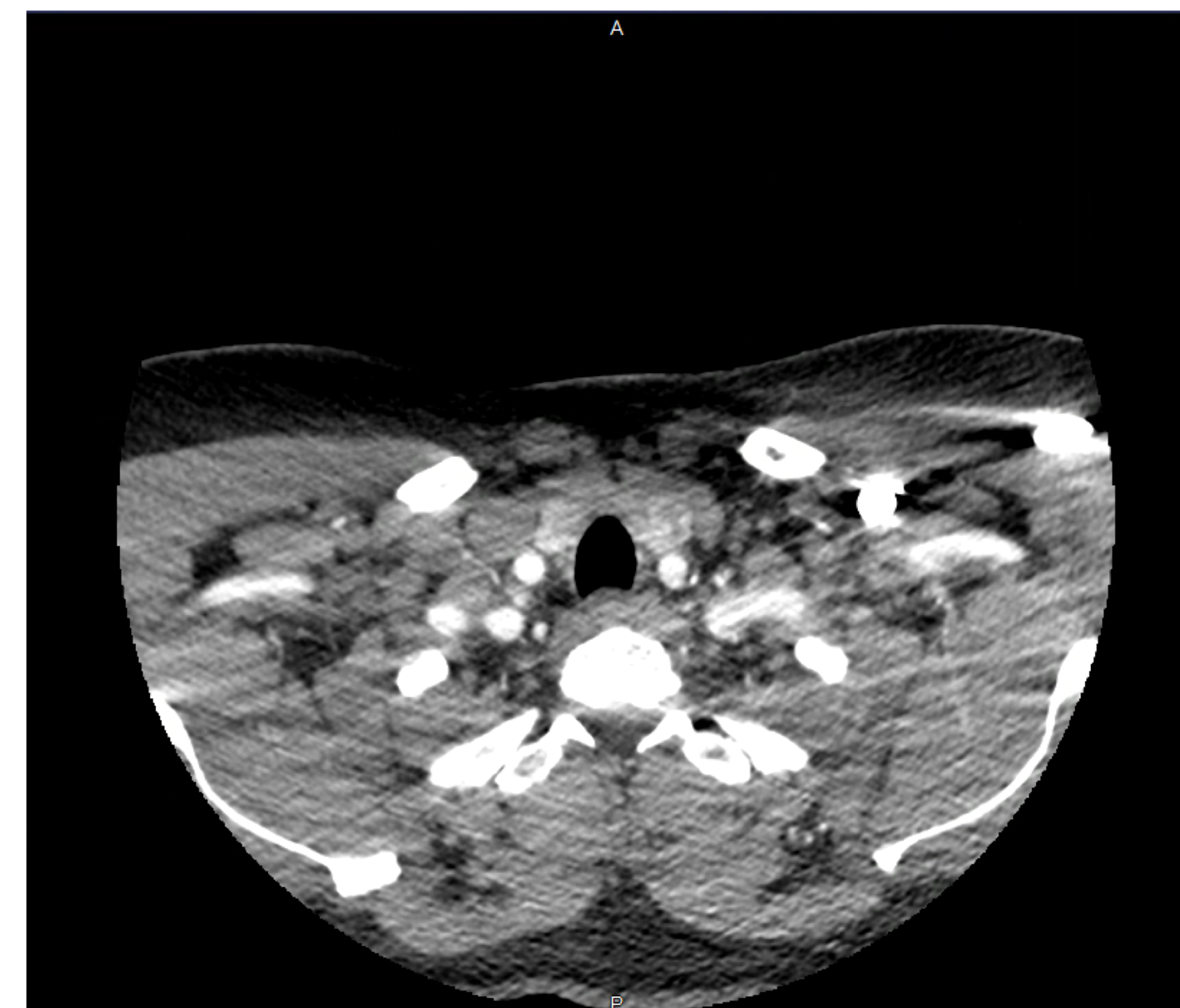
Plan:

- Admitted to acute inpatient rehab on day 10.
- Daily physical therapy and occupational therapy sessions for 1 week.

IMAGES



CT Head



CTA Neck



DISCUSSION

Beta-2-glycoprotein I (B2GI) inhibits the intrinsic coagulation pathway and is involved in the regulation of blood coagulation. Antibodies are commonly found in lupus and may play a role in formation of atherosclerosis in ischemic stroke. We present a case of a young individual with risk factors for hypercoagulability with a cerebellar infarct localized to the vermis of the posterior lobe. Her lack of cranial nerve involvement, sparing of the sympathetic nervous system, and lack of sensory or motor symptoms reflect noninvolvement of the brain stem, despite the proximal location of the lesion.

CONCLUSIONS

In young patients with cerebellar stroke without typical inciting factors, presentation may be atypical. Workup should include serum factors that may contribute to hypercoagulation.

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