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Neuroimaging Highlight

Headache due to Spontaneous Intracranial Hypotension in a Patient with Vertebral Bone Metastasis

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A 70-year-old man presented with a 7-day history of progressively worsening headache, rendering him bedbound. He has a history of prostate cancer with local bone metastasis at the L2 vertebral body, treated with focal radiation and chemotherapy. His headache was bi-occipital and pulsatile, radiating down the neck. It worsened with upright posture and Valsalva maneuvers, with immediate resolution upon lying down. Associated symptoms included photophobia, phonophobia, nausea and tinnitus. He had no history of prior trauma, back pain or spinal procedures. His headache was unresponsive to hydration, analgesics and caffeine. His neurological examination was unremarkable.

MRI of the brain showed subdural collections, pachymeningeal enhancement, brainstem sagging and flattening of the pons (Figure 1A–C), suggestive of spontaneous intracranial hypotension (SIH). Spine MRI demonstrated progression of bone metastases from L2 to the entire spine, confirmed by F-18 prostate-specific membrane antigen positron emission tomography (Figure 1D–E). Several dynamic digital subtraction myelograms were unrevealing. Ultimately, a CT myelogram in the left decubitus position showed CSF leakage into a T2–3 paraspinal vein, confirming CSF-venous fistula (CVF) (Figure 2A–B). Subsequent endovascular embolization failed due to difficult venous access, and a left T2–3 epidural fibrin glue injection provided temporary relief (Figure 2C). Finally, minimally invasive decompression and embolization of the CVF resulted in complete headache resolution within 2 days.

SIH is frequently associated with CSF leaks, with CVFs being the least common type.[1] CVFs are characterized by abnormal connections between the spinal subarachnoid space and epidural veins, allowing direct CSF-venous drainage without creating epidural collections visible on standard imaging.[2] Consequently, diagnosing CVFs is difficult and requires digital subtraction myelography or dynamic CT myelography in the lateral decubitus position.[3,4]

The relationship between CVF formation and vertebral bone metastases is not fully understood. Pathophysiology may be multifactorial secondary to tumor erosion, pressure gradient formation and local mass effect. Direct erosion and local mass effect could contribute to rupture of spinal arachnoid granulations into adjacent veins, which has been proposed to cause CVF.[5] Dural integrity may be altered via osteolysis from bone metastases or mechanisms associated with spinal degenerative disease.[6] Venous congestion from tumors may additionally create a pressure gradient that facilitates abnormal CSF diversion into the epidural plexus.

SIH is rarely reported in cancer patients and even less so in those with bone metastases. We found one case describing SIH in a breast cancer patient with vertebral bone metastases,[7] where, similar to our case, no CSF leak site was identified on initial imaging.

Several treatment strategies have been proposed for CVF, including surgical ligation, nerve root skeletonization and blood or fibrin patching.[8] More recently, transvenous embolization has shown high safety and efficacy.[9,10] Of these, surgical ligation remains the most effective.[11] In our patient, access to the left T2–3 foraminal vein was not possible, so we proceeded with surgical ligation of the venous outflow pathway.

In summary, vertebral bone metastases may represent a potential risk factor for CVFs, requiring thorough evaluation in patients with orthostatic headache and spinal pathology. While identifying CVFs can be challenging, advanced imaging techniques enable early detection and guide appropriate intervention.

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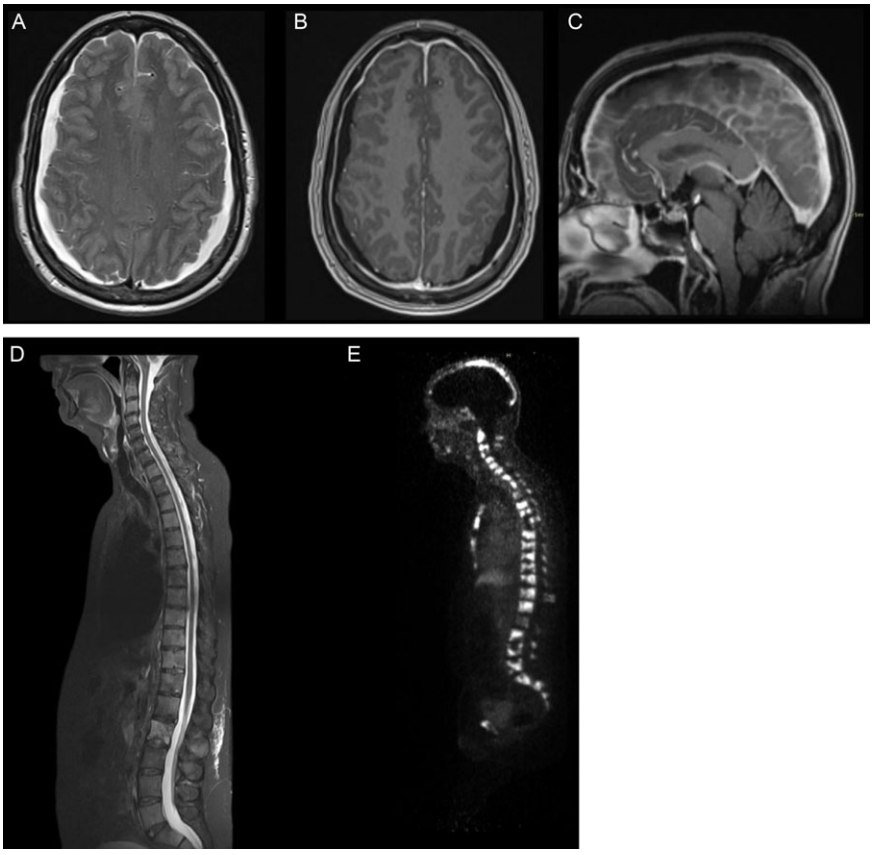

Figure 1. MRI of the brain and spine, along with a positron emission tomography (PET) scan of the patient. **(A)** Axial T2-weighted MRI of the brain showing bilateral fluid collections in a subdural distribution. **(B)** Axial T1-weighted post-contrast MRI demonstrating diffuse dural thickening and enhancement. **(C)** Sagittal T1-weighted post-contrast MRI of the head showing sagging of the upper brainstem and flattening of the ventral pons along the clivus. **(D)** Sagittal T2-weighted short tau inversion recovery MRI sequence redemonstrating diffusely abnormal signal throughout the spine consistent with diffuse metastatic disease. **(E)** F-18 prostate-specific membrane antigen PET imaging demonstrating diffuse avid skeletal metastases affecting the skull and almost the entire skeleton (SUV max 40.8).

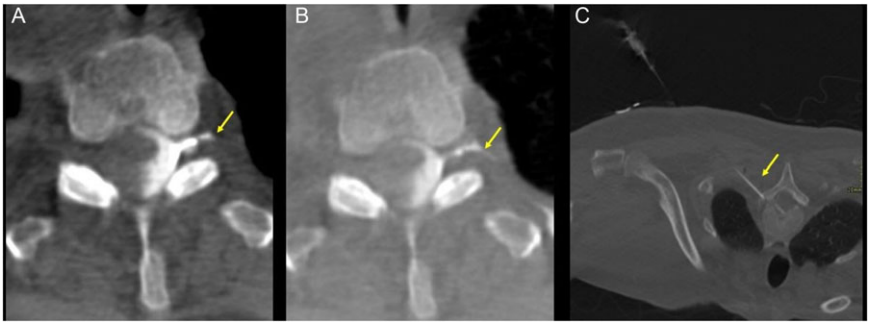

Figure 2. CT myelogram of the patient. CT myelogram demonstrating **(A)** early contrast leakage and **(B)** delayed (5 minutes) contrast extension from the nerve root sleeve into an adjacent paraspinal vein at the left T2–3 foramen (yellow arrows). **(C)** Transforaminal epidural injection of fibrin glue at the left T2–3 level via a left transforaminal approach (yellow arrow).

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