Regression of pituitary macroadenoma after endovascular embolization (of thoracic CSF-venous fistula for symptomatic intracranial hypotension



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Introduction

Pituitary hyperemia and gland enlargement can be cardinal features of intracranial hypotension secondary to cavernous sinus and epidural venous plexus distention.¹ This phenomenon can therefore complicate radiographic interpretation of sellar lesions when both diagnoses co-exist. We report a unique case of a rapidly enlarging pituitary macroadenoma in the setting of a thoracic CSF-venous fistula causing symptomatic intracranial hypotension.

Methods and Materials

• Serial MRIs obtained at 4 and 7 months showed unusually rapid growth of a pituitary macroadenoma and cardinal features of



Figure 3. Endovascular obliteration of right T6-7 foraminal CSF-venous fistula. Dynamic CT myelogram demonstrates serpentine structures

suspicious for CSF-venous fistula near the right posterior T6-7 neural foramen (left). 3D reconstruction demonstrates selective Onyx cast in the right T5-6 and T6-7 internal and external vertebral venous plexus after endovascular embolization (right).

- intracranial hypotension.
- CT-guided lumbar puncture for dynamic myelogram in the decubitus position revealed a CSF-venous fistula in the inferior and posterior right T6-7 neural foramen.
- Patient underwent endovascular obliteration with Onyx of the right epidural venous plexus at T6-7 via selective micro-catheterization of the right T6-7 intervertebral vein.







Figure 4. CSF-Venous Fistula embolization reverses intracranial hypotension and induces tumor regression. MRI imaging prior to and 4 weeks following endovascular obliteration of CSF-venous fistula shows **(A)** resolution of pachymeningeal enhancement, diencephalon sag, and foramen magnum crowding and **(B)** pituitary tumor regression and reduced mass effect on the optic apparatus.

Figure 1. Pituitary macroadenoma and intracranial hypotension. A) T2 weighted (left), T1 pre-contrast (middle) and T1 post-contrast sequences show an enhancing solid and cystic mass with extension to the left cavernous sinus and suprasellar extension to contact the optic chiasm. B) T1 post-contrast axial (left), T2 coronal (middle) and T1 sagittal sequences show diffuse leptomeningeal enhancement, reduced mamillo-pontine distance, low-hanging cerebellar tonsils, and foramen magnum crowding.



Figure 2. Sellar lesion expands rapidly in short interval. Baseline (left), 4 months (middle) and 7 months (right) after presentation.

Results

Repeat MRI obtained 4 weeks post procedurally showed near complete resolution of pachymeningeal enhancement, diencephalon sag, and foramen magnum crowding with concurrent improvement in clinical symptoms. Importantly, imaging also showed an interval decrease in the size of the patient's pituitary macroadenoma from 1.7 x 1.6 x 1.2 cm to $1.4 \times 1.3 \times 1.0$ cm with no further mass effect on the optic apparatus. This lesion remained stable in size on follow-up MRI obtained 4 months after treatment with durable symptom relief.

Conclusions

Pituitary enlargement has been reported as a feature of intracranial hypotension and has even been misdiagnosed as tumor in this context.² This report summarizes the joint presentation of a macroadenoma and intracranial hypotension that nearly prompted surgical intervention given progressive tumor enlargement in relatively short time. Endovascular treatment of a thoracic CSF-venous fistula induced durable tumor regression, suggesting that pressure disequilibrium can potentiate intratumoral venous congestion and complicate pituitary tumor management in this setting.

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