Brain Injury due to Ventricular Shunt Placement Delineated by Diffusion Tensor Imaging (DTI) Tractography

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Objective: Infection and hemorrhage are well-known complications from insertion of intracranial shunts. However, permanent injury to the brain caused by catheterization of the cerebral ventricles has rarely been reported.

Methods: We report a patient who presented at age 14 years for evaluation of a severe behavioral disorder. The patient had sustained direct injury to the corticospinal tract and limbic system during revision of a ventriculoperitoneal shunt at the age of 9 years.

Results: Despite persistent evidence of severe disruption of the corticospinal tract on diffusion tensor imaging at age 14 years, the patient had regained complete motor function.

Conclusion: Recovery of motor function after serious injury to motor cortex during childhood is a dramatic example of the plasticity of the child's brain to injury. In addition, we suggest that the behavioral disorder that emerged in this patient may be related to limbic system injury suffered during the shunt revision.

Key Words: ventriculoperitoneal shunt, brain injury, diffusion tensor imaging

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Infection and hemorrhage are well-known complications from insertion of intracranial shunts.[1–3] However, permanent injury to the brain caused by catheterization of the cerebral ventricles has rarely been reported.[4] We report a patient who sustained direct injury to the corticospinal tract and limbic system during revision of a ventriculoperitoneal shunt during early childhood. Despite persistent evidence of severe disruption of the corticospinal tract on diffusion tensor imaging (DTI) at age 14 years, the patient had no residual motor deficit, attesting to the dramatic plasticity of the developing brain to injury.

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CLINICAL REPORT

A 14-year-old male patient was referred for neurologic and behavioral evaluation after sexually assaulting younger children at school. The patient's medical history included uneventful placement of a ventricular catheter at the age of 5 years for hydrocephalus secondary to congenital aqueductal stenosis. At the age of 9 years, after revision of the shunt, the patient developed acute left hemiparesis, which subsequently resolved over several weeks. At age 14 years, the patient became withdrawn, but also exhibited intermittent episodes of aggression including sexually assaulting children at school. There was no family history of psychiatric disorders.

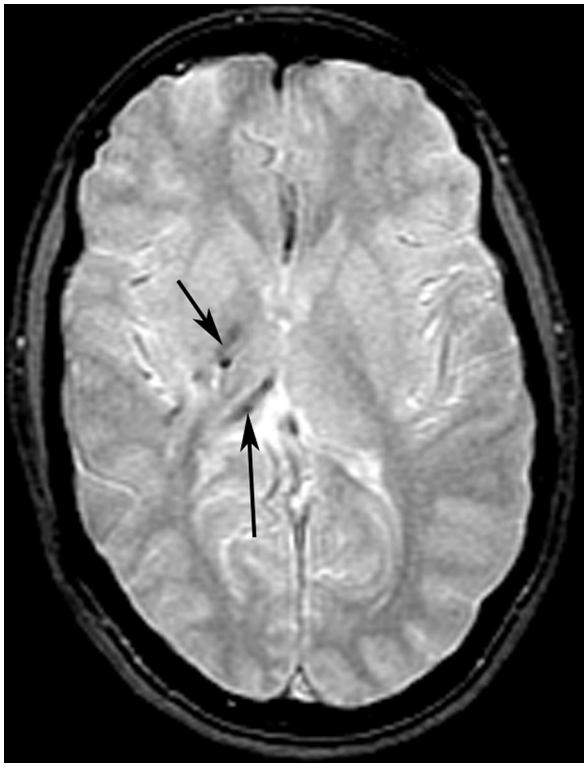

FIGURE 1. Axial GRE MRI (TR/TE, 700/15 milliseconds) demonstrates magnetic susceptibility-related signal loss in the posterior limb of the right internal capsule (short arrow) and the right thalamus (long arrow).

Complete neurologic examination at the time of presentation demonstrated 5/5 motor strength in upper and lower extremities, including intrinsic hand muscle strength. The patient had normal muscle bulk and tone. Deep tendon reflexes were normal bilaterally. There was no pronator drift and gait was tandem with normal heel and toe walking. Fine finger movements, rapidly alternating movements, and coordination were intact. Cranial nerves 2 through 12 were intact bilaterally. The patient had normal vibration and position sensation. The patient exhibited significant deficits in judgment, working memory, and insight. The patient's behavioral abnormalities were consistent with childhood-onset bipolar disorder.

Magnetic resonance imaging with DTI was performed as a component of the clinical evaluation. A shunt catheter was seen to transverse the right corona radiata, posterior limb of the internal capsule, and thalamus. Susceptibility-related signal loss along the course of the catheter was suggestive of old hemorrhage (Fig. 1). Asymmetric volume loss of the right hippocampus, amygdala, mamillary body, and fornix were present (Figs. 2 and 3). DTI-based fiber tracking showed dramatically decreased caliber of the right corticospinal tract and fornix (Fig. 4).

DISCUSSION

We describe a case of significant brain injury, affecting the corticospinal tract and limbic system, during ventriculoperitoneal shunt revision. Despite persistent evidence of severe disruption of the corticospinal tract, the patient recovered completely, with no evidence of motor deficit. Development of a severe behavioral disorder in this patient after the shunt revision suggests that it may have been instigated by injury to the limbic system.

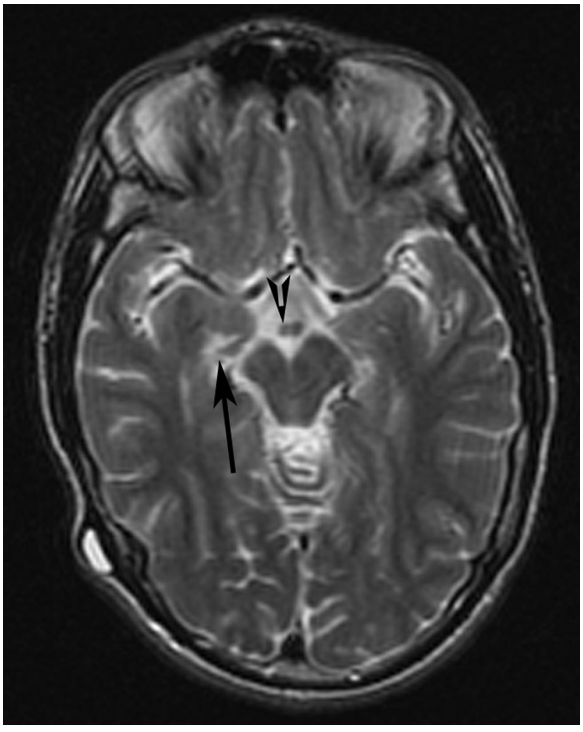

FIGURE 2. Axial T2-weighted MRI (4500/118) demonstrates asymmetric atrophy of the right mamillary body (arrowhead) and right amygdala (arrow) with compensatory dilatation of the temporal horn of the right lateral ventricle.

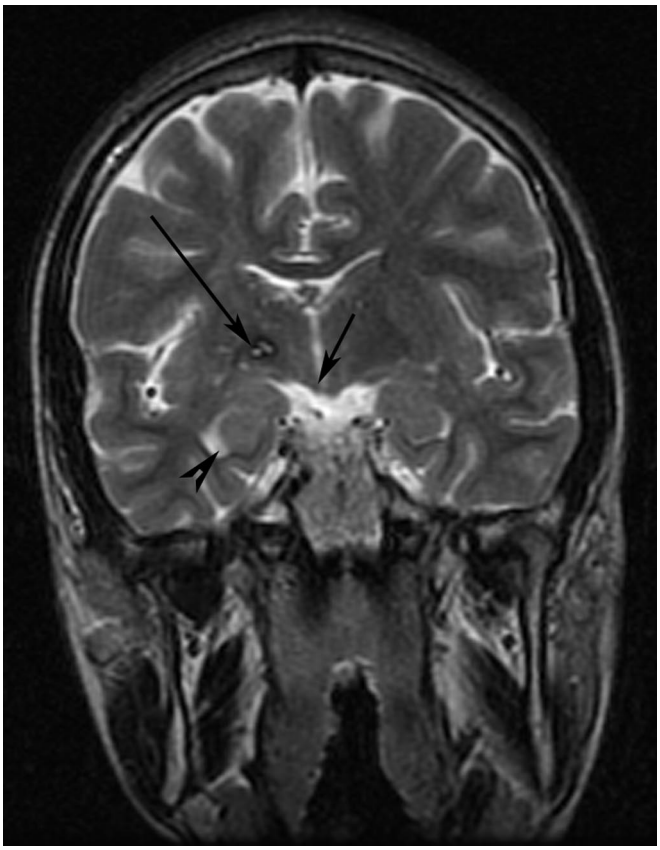

FIGURE 3. Coronal T2-weighted MRI (4000/124) demonstrates atrophy of the right mamillary body (short arrow) and hippocampus (arrowhead) as well as hemosiderin deposition in the region of the right internal capsule (long arrow).

Recovery of motor function after serious injury to motor cortex during childhood is a dramatic example of the plasticity of the child's brain to injury. The mechanism by which such patients regain motor function is incompletely understood, but 2 plasticity mechanisms are known. The first is the recruitment of cortex in the contralateral normal hemisphere. The second is the expansion of the damaged representation into cortex adjacent to the lesion. It is not clear under what circumstances these 2 mechanisms begin to operate. Muller et al concluded that multiple brain areas beyond the motor cortex, such as secondary motor and fronto-parietal nonmotor cortices, can participate in motor processes following a Rolandic lesion within the first years of life and that this potential plasticity is reduced when the lesion occurs later in life.[5] Other studies report that the contralateral intact hemisphere takes over the motor function of the damaged one.[6]

We suggest that the severe behavioral disorder that developed in our case may have been a consequence of injury

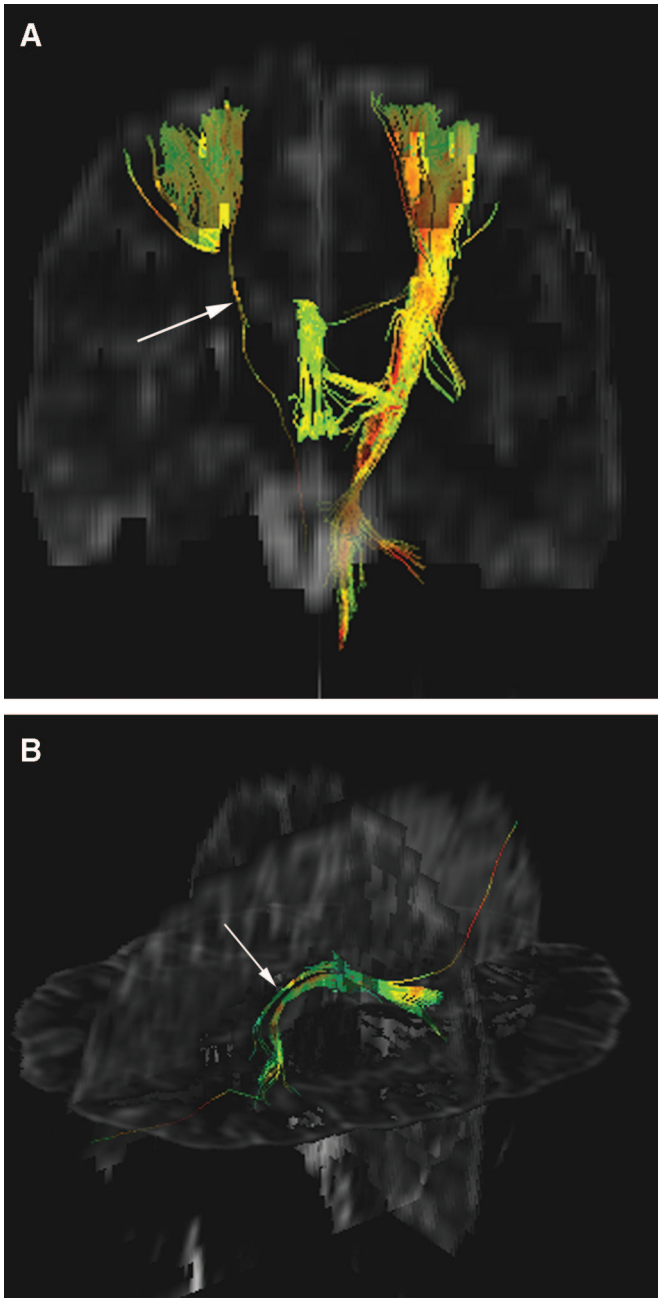

FIGURE 4. Diffusion tensor imaging-based fiber tracking shows dramatically decreased caliber of the right corticospinal tract (A) (arrow) and fornix (B) (arrow).

to the right-sided limbic system at the time of surgery. Other case studies demonstrate that incident bipolar disorder after brain injury is strongly associated with lesions involving areas of the right hemisphere that are connected with the limbic system. Cohen and Niska reported 2 patients who developed secondary mania after right hemisphere lesions and suggested there was an association between secondary mania and right hemisphere damage.[7] Starkstein et al showed that patients with bipolar disorder had a high frequency of lesions on the right side involving the caudate and thalamus.[8]

Magnetic resonance imaging studies of pediatric bipolar patients have detected structural abnormalities in a number of brain regions implicated in affective and cognitive processes. Botteron et al reported increased asymmetry of the cerebral hemispheres and ventricles in adolescent bipolar patients.[9] A study conducted by DelBello et al found that adolescents with bipolar disorder had smaller amygdala and larger putamen volume compared with healthy controls.[10] Finally, Frazier et al recently demonstrated that youths with bipolar disorder have smaller hippocampal volume than normal controls.[11] Based on these prior reports, injury to right side limbic structures in our case may have facilitated, if not caused outright, the development of a severe behavioral disturbance similar to bipolar disorder.

In conclusion, we report the case of a child who suffered significant brain injury during revision of a ventricular shunt. Despite persistence of severe disruption of the cortical spinal tract at age 14 years, as demonstrated by DTI, the patient recovered completely from initial hemiplegia. This attests to the marked plasticity of the brain during childhood even to severe injury. In addition, we propose that the patient's development of childhood-onset bipolar disorder may be related to the extensive limbic system damage incurred during the shunt revision.

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