HHS Public Access

Author manuscript

Am J Psychiatry. Author manuscript; available in PMC 2015 April 29.

Published in final edited form as:

Am J Psychiatry. 2010 September ; 167(9): 1033–1037. doi:10.1176/appi.ajp.2010.09071043.

Identification and Treatment of a Pineal Gland Tumor in an Adolescent with Prodromal Psychotic Symptoms

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Abstract

An adolescent male patient originally presented to a prodromal clinical research program with severe obsessive-compulsive behaviors and subthreshold symptoms of psychosis, which eventually developed into Schneiderian first-rank psychotic symptoms. The patient was followed over a two-year period. His symptoms did not respond to psychotherapy or pharmacological interventions. However, when a pineal gland tumor was discovered and treated with chemotherapy and autologous stem cell rescue, both psychotic symptoms and psychosocial functioning reverted towards baseline. Although subcortical brain structures have been implicated in the pathophysiology of idiopathic psychosis, reports of psychiatric sequelae of treatment of subcortical tumors are extremely rare. Etiological pathways that may have played a role in symptom development are of particular interest, as understanding these mechanisms may shed light on the pathophysiology of psychotic disorders more generally.

Case Presentation

"David" is a Hispanic male, who was referred to the University of California Los Angeles (UCLA) Center for Assessment and Prevention of Prodromal States (CAPPS) clinical research program at age 17 by his psychologist due to subthreshold psychotic symptoms and behaviors believed to represent a potential precursor to an Axis I psychotic disorder (1). At the intake interview, David's mother reported that his early developmental history was unremarkable, with no mental health-related difficulties during childhood aside from some mild attentional problems. Notably, David had received "A" grades and was also accepted into a gifted program in elementary school.

David began to show a marked behavioral decline at the onset of puberty. During the 8th grade (age 13), David's parents were undergoing a divorce, and both his teachers and parents reportedly viewed his presentation of mild depressive symptomatology and attentional problems as an adjustment disorder related to this situation. However, David continued to show signs of disturbance, and in the ninth grade his marks had dropped to the "D" range.

Disclosure: There are no potential conflicts of interest to report.

David's mother had also noticed a decline in verbal expression, and around this time he began to exhibit unusual behaviors (e.g., making idiosyncratic hand gestures, exhibiting a preoccupation with checking the time, insisting the bed covers were folded a certain way before he could fall asleep, and excessively over-eating salty foods such as ketchup and peanuts). As the year progressed, David shifted from being preoccupied with salty foods to excessively drinking water, which was diagnosed as psychogenic polydipsia. He was referred for psychiatric treatment at this time, although psychological and pharmacological interventions (fluvoxamine 300 mg) did not ameliorate the symptoms. David remained in psychotherapy for the next several years, and fluvoxamine was eventually discontinued.

Throughout the tenth grade David's verbal expression continued to decline. His mother reported that David was only expressing himself in brief phrases, consisting of a few words at a time. As his academic performance continued to drop, David was subsequently transferred to a charter school in the eleventh grade. That winter, David also began to exhibit unusual thought content (detailed below), and his treatment providers referred him to the CAPPS program for further evaluation and follow-up. He was evaluated using the Structured Interview for Prodromal Syndromes (SIPS), a semi-structured diagnostic interview consisting of five components: the 19-item Scale of Prodromal Symptoms (1), a version of the Global Assessment of Functioning with well-defined anchor points, DSM-IV schizotypal personality disorder checklist, family history of mental illness, and a checklist for the Criteria of Prodromal Syndromes (2-3). The evaluation revealed that David had a euthymic mood state, but was exhibiting attenuated positive symptoms of psychosis. In particular, he endorsed a belief in mind-reading (reporting about 50% conviction) and indicated that he felt as if he was at the center of people's attention at times. This concern sometimes prevented him from leaving the house. David also reported that in the past month he had repeatedly heard clapping and hissing sounds, but was unable to find the source of these noises. Based on these symptoms, David met research criteria for Attenuated Positive Prodromal Syndrome (1-3) and was invited to participate in the CAPPS program. The two-year longitudinal course of each of David's positive prodromal symptoms from the SIPS evaluation is presented in Table 1.

At this point, David was receiving “D” and “F” marks in school. David was administered a neuropsychological test battery, which indicated a Full Scale Intelligence Quotient (FSIQ) estimate of 88 (21st percentile), based on the Wechsler Abbreviated Scale of Intelligence (WASI) (4). Although David had not received any prior cognitive evaluations, it is very likely that these scores represented a marked drop from his premorbid level of cognitive function, given his previous level of achievement. David had a neurological consultation including a magnetic resonance imaging (MRI) scan without contrast that did not reveal any irregularities, although it was terminated early when David was unable to lie still.

Over the course of follow-up assessments, examiners learned that David was exhibiting additional behaviors that were not characteristic prodromal symptoms, and may have been suggestive of neurological dysfunction. He continued to exhibit stereotyped and idiosyncratic hand gestures resembling gang signs and adopted a bizarre and elaborate series of handshakes. In addition, he had begun to wander away from home, becoming lost and talking in a familiar manner to strangers. David lost orientation to time and date as well. He

also began to engage in self-injurious behavior, such as scraping his arm against the wall, which his mother noted may have been an attempt “to get attention” at school. During a subsequent interview, David's mother reported that her son had recently begun to occasionally drag his right foot. As the year progressed, David expressed an interest in learning to write with his non-dominant (left) hand, although he explained this by saying it was a strategy to avoid schoolwork.

During the 6-month period following David's inclusion in the CAPPS program, he did not appear to benefit from weekly psychotherapy or several consecutive regimens of atypical antipsychotic treatment, including sertraline, quetiapine furmarate, and finally aripiprazole. Further, the polydipsia had begun to severely impair his functioning and on one occasion David was hospitalized due to a disruption of his equilibrium secondary to electrolyte imbalance. After David had been participating in our program for 6 months, he began to express overtly psychotic symptoms including a belief in mind reading (at this time with 100% conviction), thought withdrawal, and both olfactory and auditory hallucinations (e.g., he reported a man and a woman's voice alternating with the message, “You should have stayed in bed”). Given David's severe impairment, disorganization, and need for constant supervision, he was enrolled in short-term residential treatment. His treatment providers believed that David was showing signs of an emerging psychotic disorder.

By age 17, David had not responded well to treatment, and the family was planning to transfer him to a long-term treatment facility. As part of a routine entry examination to this program, David was administered another MRI scan, both with and without contrast, and this time the results revealed a soft tissue mass in the region of the dorsal midbrain/pineal cistern. A subsequent biopsy indicated a diagnosis of pineal mixed germ cell tumor (29 mm in maximum dimension). The scan also revealed evidence of a small left-sided basal ganglia stroke, which was estimated to have occurred corresponding to the onset of lateralized motor difficulties. The tumor also resulted in hydrocephalus with distention of the third and lateral ventricles. Immediately following the diagnostic biopsy, a Rickham reservoir (shunt) was put in place (entering the frontal horn of the right lateral ventricle) to treat edema. David was also taken out of school, antipsychotic medication was permanently suspended, and he was immediately placed on a regimen of chemotherapy.

Over the next six months, David was treated with a total of six cycles of high-dose chemotherapy at which point the tumor had rescinded to 7mm. To help his body tolerate the high doses of chemotherapy, David began procedures for an autologous stem cell transplant in his last month of chemotherapy; the procedure was implemented over the next seven weeks. This combination of treatments was successful, and the tumor had almost entirely rescinded (3.7 mm) at the conclusion of therapy. Figure 1 illustrates the tumor prior to treatment compared to post-treatment. However, the tumor's sequelae included pan-hypopituitarism and diabetes insipidus. David had also begun to complain of peripheral vision loss during his treatment that has continued to the present date. This persistent symptom may also be secondary to the tumor, reflecting pressure from the swelling and hydrocephalus induced by the tumor that may have caused the pituitary to press against the optic chiasm.

A comprehensive clinical and neuropsychological twelve-month follow-up assessment was conducted at the CAPPS program. At this stage in treatment, David's tumor had receded from 29 mm to 7 mm, and he was preparing for the stem cell transplant stage of the treatment regimen. Overall, during the assessment it became clear that his behavior had begun to improve towards premorbid functioning. David's psychotic symptoms had remitted to the mild range; he continued to exhibit mild unusual thought content, some paranoia, and perceptual abnormalities, although these symptoms did not reach the psychotic threshold. The water drinking “compulsion” had abated, likely as a result of treatment for David's diabetes insipidus. In addition, David's academic performance had begun to improve as the tumor receded; standardized scholastic testing in shortly before the diagnosis of the tumor had indicated that David was performing at a third to fifth- grade level. However, more recent testing during his treatment revealed that his performance had improved to the sixth to eighth-grade level.

Despite the overall improvements, several ongoing complications were noted. David's motor functioning on the right side of his body remained impaired. He also continued to exhibit mild depressed mood. While many of the unusual behaviors had remitted (e.g., staring at the clock, unusual hand gestures, waving at people), some behaviors persisted; for example, David continued to frequently make the sign of the cross at unusual times during his follow-up interview. His mother indicated that this may have begun as a release of anxiety during the difficult periods of treatment, although it is possible these gestures may be etiologically related to the other unusual motor symptoms.

In the year subsequent to his tumor treatment, David has worked closely with a multi-disciplinary treatment team (including a psychotherapist, endocrinologist and a physical therapist) and his physical health has continued to improve. At the time of this writing (twenty-four months after the baseline assessment at CAPPS), David has returned to public high school to attend the twelfth grade and is participating in class three days a week. Although he requires some accommodations (e.g., help with note taking due to the right-sided motor difficulties), the family plans for David to receive the standard curriculum and graduate from high school. His overall mental health status and social and role functioning have considerably improved; although David continues to exhibit mild depression and some compulsive motor behaviors (e.g., making the sign of the cross frequently), he has not displayed any recurrence of psychotic symptomatology since the treatment for the tumor began. Given this longstanding period of stability, we feel confident in re-conceptualizing the case as “Psychosis due to General Medical Condition” (DSM-IV-TR).

Discussion

Our patient originally presented with a pattern of behavioral decline and unusual thought content suggestive of the prodromal phase of psychotic illness. However, over the course of longitudinal assessment, he developed increasingly severe signs suggestive of potential underlying neurological etiology (e.g., foot-dragging, lateralized motor symptoms, polydipsia). David's presentation ultimately progressed to fully psychotic symptoms, which remitted subsequent to the discovery and treatment of a pineal gland tumor. Although causality cannot be conclusively determined, the timeline of symptom development strongly

suggests that the pineal gland tumor and small left-sided basal ganglia stroke played a primary role in the onset of psychotic and obsessive-compulsive symptomatology in this case.

Despite the heavy genetic loading associated with psychotic disorders, organic or environmental origins underlying psychosis have also been well documented. Although the specific pathogenic mechanisms of central nervous system (CNS) damage leading to psychosis are unknown, exposure to trauma is associated with an almost five-fold increase in psychotic-like phenomena (5), and psychotic symptoms can also manifest secondary to traumatic brain injury, particularly when a pre-existing head injury occurred prior to adolescence (6). These examples also point to the complex gene-environment interactions that are likely to be relevant to the pathophysiology of psychosis (7). Although speculative, it is possible that in David's case an unidentified genetic liability may have interacted with pathological processes either directly or indirectly resulting from the emerging pineal tumor. With regard to the present case, there is also evidence that cerebral neoplasms can be associated with both delusions and hallucinations (8). Notably, a long-term follow-up study of survivors of childhood CNS malignancies found that several of these individuals later developed persistent psychosis, many years after the tumor had been successfully treated (9).

The pineal gland is located at the posterior extent of the third ventricle, extending over the tectal plate of the midbrain. Through the production of melatonin and subsequent modulation of sex hormones, the gland is a key structure involved in sexual development and the onset of puberty. Tumors in this region are extremely rare (accounting for about one percent of all brain tumors), occurring most often in children and young adults (10). The most common of these are germ cell tumors (germinomas and teratomas), which arise from embryonic remnants of germ cells. These tumors are malignant, invasive and may be life-threatening. Although it is not possible to pinpoint when David's tumor first began to develop, it likely coincided with the onset of his behavioral decline during puberty. Previous case reports have indicated tumors involving the pineal body can result in an irregular course of sexual development in pediatric populations (11). However, it should be noted that David did not display signs of either precocious or delayed sexual development; there were no reports of irregular sexual maturation and during his participation in the CAPPS program, David continued to receive Tanner Staging scores (12) indicative of age-appropriate development. In David's case, it is possible that the tumor manifested after the onset of puberty, and thus did not observably alter sexual maturation.

However, given the pineal gland's importance in pubertal development, and the timing of the tumor and symptoms in David's presentation, this case may provide important clues regarding the pathophysiology of psychotic disorders and their typical onset in adolescence. Several lines of evidence suggest a possible relationship between pineal gland abnormalities and symptoms of thought disorder. In particular, pineal gland irregularities (e.g., calcification) have been observed in patients with schizophrenia (13), and others have noted that these abnormalities may be related to abnormal nocturnal melatonin secretion in patients with schizophrenia (14). Anecdotal reports have also suggested that high doses of melatonin may exacerbate psychotic symptoms in patients with schizophrenia (15). It is also

noteworthy that the pineal area holds a rich concentration of sigma receptors, which bind haloperidol and other antipsychotic agents (16).

There have been very few case reports of psychosis associated with subcortical tumors in children. We were able to identify only three such cases in the literature (17-19), which bore interesting similarities to our patient. Benjamin and colleagues (17) described a 9-year old boy presenting with a choroid plexus papilloma in the anterior third ventricle and worsening psychotic symptoms, including auditory hallucinations, which remitted following resection of the tumor. Although the origin of psychotic symptoms in this case is unclear, it is possible that the tumor location in the third ventricle - in close proximity to the pineal gland- played a role. Within this context it is notable that Sandyk (20) identified third ventricular width and pineal calcifications as the sole neuroradiologic indicators that were related to thought disorder severity in chronically psychotic patients.

Mordecai and colleagues (18) described a 13-year-old boy with a suprasellar germinoma involving the bilateral basal ganglia. Similar to our patient, this case involved left-sided weakness, diabetes insipidus, marked decline in academic function, and both obsessive-compulsive symptoms and psychosis, including a loosely organized delusional system and auditory hallucinations. However, unlike David, this patient's symptoms and cognitive functioning did not substantially improve with treatment of the tumor.

Finally, Craven (19) described a 15-year-old girl who developed intermittent but severe psychotic symptoms, including paranoid delusions and auditory perceptual abnormalities, approximately 14 months after a pineal germinoma was successfully treated. This patient also developed diabetes insipidus and panhypopituitarism secondary to the tumor. Given the atypical course of psychotic symptomatology, the author speculated that the symptom onset may have been related to pituitary hormone replacement therapy, as cases of corticosteroid-induced psychosis have been reported (21).

The MRI that identified the pineal tumor also indicated that David had suffered a small left-sided basal ganglia stroke. Although it bore no obvious relationship to the development of the tumor, it is possible that the enlarging tumor may have resulted in cerebral vascular accident (CVA). David's stroke-related basal ganglia dysfunction, along with other potential contributing factors (i.e., the pineal tumor and neuroendocrine dysregulation) may have also additively or interactively affected the development of prodromal and ultimately psychotic symptomatology. Indeed, cortico-striato-pallido-thalamic circuit malfunction, involving striatal hyperdopaminergia, may predate onset of psychosis in at-risk individuals (22-23).

It is also possible that the basal ganglia stroke contributed to the onset and/or exacerbation of obsessive-compulsive symptoms in the present case (e.g., preoccupation with checking the time, insisting the bed covers were folded a certain way before he could fall asleep). Disorders that involve the basal ganglia (e.g., Sydenham's chorea, Huntington's disease) can present with both psychotic and obsessive-compulsive symptoms (24-26). Structural and functional neuroimaging studies have demonstrated abnormalities of the basal ganglia and associated corticostriato-thalamocortical circuitry in the pathophysiology of OCD (27). Of particular relevance to David's case, Peterson and colleagues (28) reported three cases in

which preexisting OCD symptoms became more severe with the development of cerebral malignancies affecting corticostriato-thalamocortical circuitry, and then improved with successful treatment of the tumor. When David first began showing excessive consumption of salty foods and then developed polydipsia, these behaviors were originally conceptualized as another manifestation of his OCD behavior, which also included compulsive checking and other routines. However, in hindsight it is clear that the compulsive water drinking actually reflected diabetes insipidus, likely related to his subcortical brain pathology.

Conclusion

This report illustrates a compelling case in which the psychotic symptom onset tracked with the development of the pineal tumor – and remitted when the tumor rescinded – such that we believe that the pineal gland tumor is the most likely etiology of these symptoms, although other factors, particularly the basal ganglia stroke, may have also contributed. Previous case reports describing subcortical tumors associated with psychosis and/or worsening OCD symptoms provide further support for the hypothesized causal relationship (17-19).

Acknowledgments

This work was supported by National Institutes of Health Grants MH52857 (T.D.C.), L30 MH087258-01 and NRSA T32 MH14584-33 (V.A.M.), and NARSAD (C.E.B). Funding was also provided by a gift to the UCLA Foundation from the Staglin Music Festival for Mental Health.

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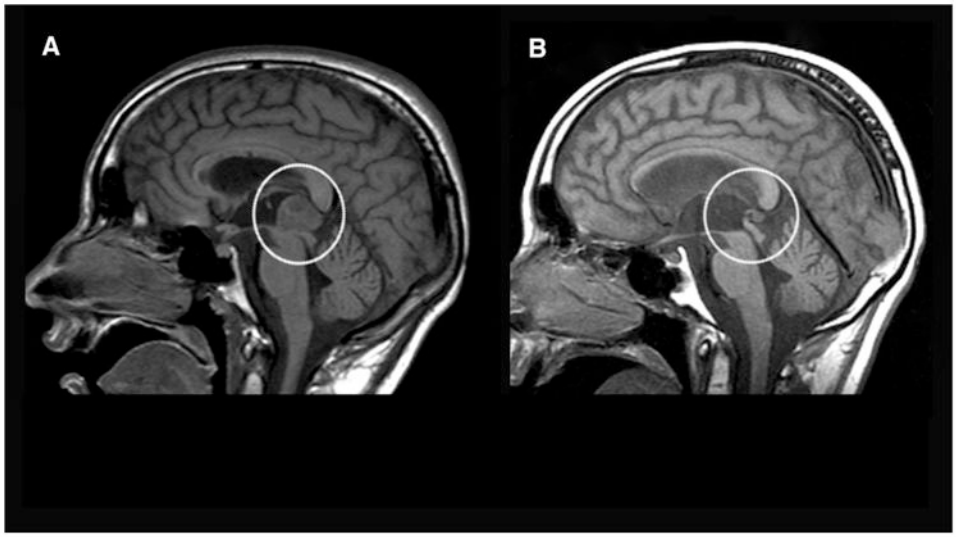

Figure 1.

Baseline and 16-Month Follow-up Magnetic Resonance Images Demonstrating Pineal Region Mass Before and After Chemotherapy Treatments and Stem Cell Transplant[a].

[a]The scan image presented in “A” represents the soft tissue mass in the dorsal midbrain/pineal cistern (circled; 29mm). The image represented in “B” shows the post-treatment rescinded pineal germ cell tumor 16 months later (circled; 3.7mm).

Table 1
Longitudinal Progression of Positive Symptoms

Positive Symptoms	Baseline	Six-Months	Twelve-Months	Twenty-Four-Months
	Initial Ratings	Psychosis Onset	Ongoing Tumor Treatment	Post-Treatment Recovery
Unusual Thought Content	4	6	3	1
Suspiciousness	5	5	4	1
Grandiosity	1	3	5	0
Perceptual Abnormalities	3	6	4	0
Disorganized Communication	4	6	1	1

Structured Interview for Prodromal Syndromes Scores (1): 0 = absent, 1 = questionably present, 2 = mild, 3 = moderate, 4 = moderately severe, 5 = severe but not psychotic, 6 = psychotic level of severity.