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Steroid-Responsive Encephalopathy Associated With Hashimoto's Thyroiditis in an Adolescent With Chronic Hallucinations and Depression: Case Report and Review

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CONCLUSION

Vit B_{12} deficiency may be seen in infants and present with distinct clinical and MRI findings.

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REFERENCES

1. Schenck UV, Bender-Götze C, Koletzko B. Persistence of neurological damage induced by dietary vitamin B-12 deficiency in infancy. *Arch Dis Child*. 1997;77:137–139
2. Kalaycıı Ö, Çetin M, Kıirel B, et al. Neurologic findings of vitamin B-12 deficiency: presentation of 7 cases. *Turk J Pediatr*. 1996;38:67–72
3. Graham SM, Arvela OM, Wise GA. Long-term neurologic consequences of nutritional vitamin B_{12} deficiency in infants. *J Pediatr*. 1992;121: 710–714
4. Renault F, Verstıchel P, Ploussard JP, Costil J. Neuropathy in two cobalamin-deficient breast-fed infants of vegetarian mothers. *Muscle Nerve*. 1999;22:252–254
5. Grattan-Smith PJ, Wilcken B, Procopis PG, Wise GA. The neurological syndrome of infantile cobalamin deficiency: developmental regression and involuntary movements. *Mov Disord*. 1997;12:39–46
6. Jadhav M, Webb JKG, Vaishnava S, Baker SJ. Vitamin-B_{12} deficiency in Indian infants. *Lancet*. 1962;ii:903–907
7. Louwman MW, van Dusseldorp M, van de Vijver FJ, et al. Signs of impaired cognitive function in adolescent with marginal cobalamin status. *Am J Clin Nutr*. 2000;72:762–769
8. Goebels N, Soyka M. Demantia associated with vitamin B_{12} deficiency: presentation of two cases and review of the literature. *J Neuropsychiatry Clin Neurosci*. 2000;12:389–394
9. Emery SE, Homans AC, Colletti RB. Vitamin B_{12} deficiency: a cause of abnormal movements in infants. *Pediatrics*. 1997;99:255–256
10. Frenkel H. Abnormal fatty acid metabolism in peripheral nerve of patients with pernicious anemia. *J Clin Invest*. 1973;52:1237–1245
11. Cooper BA, Rosenblatt DS. Inherited defects of vitamin B12 metabolism. *Annu Rev Nutr*. 1987;7:291–320
12. Carmel R, Wathuis D, Goodman SI, Rosenblatt DS. Heriditary defects of cobalamin metabolism (CbIG-mutation) presenting as a neurological disorder in childhood. *N Engl J Med*. 1988;318:1738–1741
13. Allen RH, Stabler SP, Savage DG, Lindenbaum J. Metabolic abnormalities in cobalamin (vitamin B_{12}) and folate deficiency. *FASEB J*. 1993;7: 1344–1353
14. Higginbottom MC, Sweetman L, Nyhan WL. A syndrome of methylmalonic aciduria, homocystinuria, megaloblastic anemia and neurologic abnormalities in a vitamin B_{12}-deficient breast-fed infant of a strict vegetarian. *N Engl J Med*. 1978;299:317–323
15. Kuhne T, Bubl R, Baumgartner R. Maternal vegan diet causing a serious infantile neurologic disorder due to vitamin B_{12} deficiency. *Eur J Pediatr*. 1991;150:205–208
16. Funada U, Wada M, Kawata T, et al. Changes in CD4+CD8−/ CD4−CD8+ ratio and humoral immune functions in vitamin B_{12}-deficient rats. *Int J Vitam Nutr Res*. 2000;70:167–171
17. Sharma S, Khilnani GC, Berry M. Case of season. Megaloblastic anemia with subacute combined degeneration (SCD) of the spinal cord. *Semin Roentgenol*. 1999;34:2–4
18. Bassi SS, Bulundwe KK, Greeff GP, Labuscagne JH, Gledhill RF. MRI of the spinal cord in myelopathy complicating vitamin B_{12} deficiency: two additional cases and review of the literature. *Neuroradiology*. 1999;41: 271–274

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ABBREVIATIONS. MRI, magnetic resonance imaging; SPECT, single-photon emission computed tomography; TSH, thyroid-stimulating hormone; EEG, electroencephalogram background activity; TPO, thyroperoxidase; CSF, cerebrospinal fluid; SREHT, steroid responsive encephalopathy associated with Hashimoto's thyroiditis.

We describe the case of a 14-year-old girl who presented with a 5-year history of hallucinations and depression. She had significantly elevated thyroperoxidase (TPO) antibody titers consistent with the diagnosis of Hashimoto's thyroiditis. A magnetic resonance imaging (MRI) scan of the brain showed white matter changes affecting the frontal lobe, and cerebral hypoperfusion deficits were observed on serial single-photon emission computed tomography (SPECT) scans. The patient had significant clinical improvement and showed resolution on neuroimaging after corticosteroid treatment. Steroid responsive encephalopathy associated with Hashimoto's thyroiditis (SREHT) is a more accurate description of the previously named "Hashimoto's encephalopathy." This is a condition with neuropsychiatric symptoms associated with high anti-thyroid antibody titers which shows marked improvement following corticosteroid treatment.

The medical evaluation of adolescents who present with psychiatric symptoms requires a full clinical assessment to exclude organic disease. The list of potential etiologies in these patients can be long, particularly if presenting symptoms do not fall into recognized patterns. We present the case of an adolescent with recent disclosure of long-standing hallucinations and depression who was found to have elevated thyroid-stimulating hormone (TSH) and anti-thyroid antibody titers. This case reinforces the importance of evaluating thyroid function in pediatric patients who present with ill-defined neuropsychiatric symptoms.

CASE REPORT

The patient is a 14-year-old girl with no significant past medical history who described visual and auditory hallucinations beginning at age 9. Her visual hallucinations comprised seeing animals and unknown people on walls often engaged in violent acts and she began hearing commanding and denigrating voices. Her symptoms caused significant distress associated with decreased mood and energy.

After normal electroencephalogram background activity (EEG), the patient was treated with numerous psychotropic medications over a 6-month period by her psychiatrist including valproic acid,

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sertraline, and quetiapine fumarate. These medications reduced the intensity, but not the frequency, of her hallucinations and resulted in intolerable adverse effects including marked weight gain and hypersomnolence.

The patient had menarche at 11 years. The patient's father suffers from epilepsy, her mother has rheumatoid arthritis, and her twin sister has also been diagnosed with Hashimoto's thyroiditis. She was an above-average eighth grade student whose school performance had recently declined.

On evaluation, the patient had an intact mental status examination with a score of 38/38 on the Kokman scale. Temperature was 37.4°C, heart rate 90 beats per minute, respiratory rate 19 breaths per minute, blood pressure 110/78 mm Hg. Height 168.3 cm (25%), weight 129 kg (≫97%), and body mass index 45.5 kg/m^2. Her formal neurologic examination did not reveal any focal findings. Thyroid examination was normal. Aside from flesh-colored striae on the abdomen, the remainder of the examination was unremarkable.

A thorough laboratory and multidisciplinary evaluation was undertaken. Initial results revealed a TSH of 77.4 mIU/mL (0.3–5.0 mIU/mL), free thyroxine of 0.5 ng/dL (0.8–1.8), and TPO antibody titer of 6320 (normal <20 IU/mL). Hemoglobin was 10.7 g/dL, MCV 79 fL, with iron studies and peripheral blood smear indicative of a hypochromic, microcytic anemia. Other laboratory studies including white blood cells, platelets, erythrocyte sedimentation rate (3 mm/hour), electrolytes, liver and renal function, ceruloplasmin, lactate, AM cortisol, corticotropin, C-reactive protein, complement, glutamic acid decarboxylase, antinuclear antibody, anti-endomysial antibody, muscle acetylcholine receptor antibody, heavy metal screen, urine-free cortisol, angiotensin-converting enzyme, and syphilis serology were normal. A chest film, electrocardiograph, and echocardiogram were also normal. Cerebrospinal fluid (CSF) analysis revealed normal cell count, protein, glucose, and was negative for oligoclonal banding. MRI of the head showed scattered foci of T2 hyperintensity in the white matter of the frontal lobe. A brain perfusion scan (SPECT) was also abnormal with patchy decreased perfusion most notable in the left temporal lobe, inferior aspect of the frontal lobes as well as decreased perfusion to the right basal ganglia. Short and prolonged video electroencephalography (24-hour) did not reveal epileptiform activity despite hallucinatory episodes.

The patient was started on 100 μg of L-thyroxine daily in addition to iron supplementation and her previous psychiatric medications. Because the patient was having some gradual improvement in symptoms and CSF analysis did not reveal elevated protein suggestive of an inflammatory process, steroids were held pending the patient returning for repeat thyroid function testing and SPECT imaging.

On follow-up visit to the clinic 1 month after her initial assessment, the patient noted mild improvements in concentration and memory but remained hallucinatory, fatigued, and dysthymic. Her L-thyroxine dose was increased after repeat thyroid testing showed an elevated TSH, and her TPO antibodies remained elevated. A second SPECT scan remained abnormal. Cortical uptake was decreased in both frontal and fronto-parietal regions, worse on the right. Improvement was noted in temporal lobe and right basal ganglia perfusion. In light of the minimal improvement in symptoms of the patient on thyroid replacement, steroid therapy was initiated with methylprednisolone 1 g intravenously per day for 3 days, followed by prednisone at a dose of 60 mg orally per day.

At the outset, the patient had a partial response to prednisone treatment with resolution of visual hallucinations, but persistence of auditory hallucinations. In an effort to lose weight, the patient stopped taking her prednisone 3 months into her steroid course. She immediately noticed an increase in frequency and intensity of her hallucinations and depressed mood, which were eliminated on resumption of corticosteroids. The patient returned for evaluation at 6 months after prednisone was initiated. She denied any hallucinations and described improved mood. Her school performance improved significantly and her weight stabilized on resumption of a regular exercise routine. At her latest visit, she had normal thyroid function tests (TSH = 2.1 mIU/L, free thyroxine = 1.2 ng/dL) and a decreased TPO antibody titer (2300 IU/mL). A repeat SPECT scan showed complete resolution of her prior perfusion deficits affecting the temporal lobe with continued decreased uptake in the frontal and parietal lobes. The steroid was decreased and the patient was advised to taper her dose monthly.

DISCUSSION

Hashimoto's thyroiditis is the most common cause of acquired hypothyroidism in children and adolescents.[1] It is a chronic autoimmune thyroiditis characterized by diffuse lymphocytic infiltration, which may result in firm goiter with a high titer of circulating anti-thyroid antibodies. Although a minority of cases present with thyrotoxicosis, most children are euthyroid or hypothyroid at diagnosis. Clinical manifestations in hypothyroid patients may include impaired growth, myxedema, cold intolerance, and lethargy as well as behavioral changes. Psychiatric symptoms include impaired short-term memory and other cognitive dysfunction in 66% to 90% and depression in 40%.[2] The term "myxedema madness," coined in 1949, described a florid psychotic illness associated with profound hypothyroidism.[3,4] This condition has become rare with the advent of sensitive thyroid screening tests and effective treatment.[5] The first description of neuropsychiatric disease associated with autoimmune thyroid dysfunction was by Brain et al[6] in 1966. He described the case of a 40-year-old coachbuilder with known thyroid-antibody positive Hashimoto's disease who subsequently developed focal neurologic deficits and coma successfully treated with steroids and thyroxine replacement. This patient was noted to have elevated CSF protein as well as slowing on EEG. Shaw and colleagues[7] further reported 5 cases with subacute onset of encephalopathy with elevated titers of anti-thyroid antibodies and proposed the term "Hashimoto's encephalopathy" as a distinct clinical entity.

SREHT is a clinical condition encompassing persistent or relapsing seizures, myoclonus, focal neurologic deficits, and delusions or hallucinations along with elevated thyroid antibody titers.[8,9] A workup must exclude other etiologies and the neuropsychiatric deficits show significant clinical improvement with corticosteroid treatment. The patient, typically female, may be euthyroid or hypothyroid and anti-thyroid antibody titers do not correlate to the degree of encephalopathy.[7–9] Although heterogeneous in presentation, 2 subtypes have been described. The vasculitic type is characterized by acute stoke-like episodes associated with focal neurologic features and seizures. The diffuse progressive type is associated with insidious onset with progressive impairment of mental status with confusion, somnolence, and psychosis.[8] Neither presentation is exclusive and significant overlap may occur. Furthermore, EEG, computerized tomography, MRI, and CSF analysis cannot distinguish between the 2 types.

Although numerous reports have been described in adults, few cases are described in children. In our review of the literature (see Table 1), we found a total of 16 cases (including our own) aged 9 to 17 years. All but 2 were female with a mean age of 13.7 years. Thirteen cases presented with seizures, and all of the cases had an intense initial workup to rule out traumatic and infectious etiologies. Only 2 primarily behavioral presentations are described: ours and a 10-

TABLE 1. Clinical Characteristics of Pediatric Patients With SREHT

Age (Yr)	S	Presentation	Thyroid Function				Imaging	SPECT	EEG	CSF	Treatment	Outcome	Reference
			St	TSH	T4	AB							
14	F	Seizures, syncope	E*	N	N	+	CT normal	—	Slowed	+, ↑ protein	Thyroxine steroid	Relapse, cognitive impairment	16
14	F	Seizures, depression	H	↑	N	+	CT normal	—	Slowed	N	Thyroxine steroid	Relapse, disturbed behavior	7
15	F	Seizure, ataxia	E*	N	N	+	MRI ↑ T2 hippocampus	—	Slowed	+, ↑ protein	Thyroxine steroid	Myoclonus, cortical atrophy	10
16	F	Seizures	H	↑	↓	+	CT/MRI normal	—	N	N	Thyroxine steroid	Relapse, eventual full recovery	9
13	F	Hyperactivity, depression	E*	N	N	+	—	—	—	—	Thyroxine steroid	Improved cognition	9
12	F	Unconscious, seizures	H	↑	↓	+	CT/MRI normal	—	Slowed	+, ↑ protein	Thyroxine	Full recovery normal cognition	13
14	M	Confusion, hemiplegia	H	↑	N	+	CT/MRI normal	L hemisphere	Slowed	N	Thyroxine flunarizine	Full recovery	25
17	F	Seizure, anxiety	E*	N	N	+	MRI normal	—	Sharp waves	+, ↑ protein	Thyroxine steroid	Full recovery	20
9	F	Seizures, confusion	H	↑	↓	+	CT/MRI normal	—	Sharp waves	N	Thyroxine	Mild cognitive deficits	15
14	F	Seizures, hemiplegia	H*	↑	N	+	MRI normal	Normal	Slowed	+, ↑ protein	Thyroxine steroid	Mild cognitive deficits	17
12	F	Seizure, headache	E*	N	N	+	CT/MRI normal	—	Sharp waves	N	Thyroxine steroid	Relapse, eventual full recovery	17
15	F	Seizure, myoclonus	E	N	N	+	MRI cerebellar atrophy	Bifrontal	Slowed	+, ↑ protein	Steroid	Relapse, eventual full recovery	24
12	F	Seizure, hemiparesis	H	↑	↓	+	CT/MRI normal	L prefrontal parietoccipital	Slowed	+, ↑ protein	Thyroxine	Full recovery	14
14	F	Seizures, hemiparesis	H	↑	↓	+	MRI ↑T2 frontal/parietal	Frontal & parietal	Sharp waves	+, ↑ protein	Steroid	Full recovery	11
14	M	Seizures, ↓cognition	E	N	↓	+	CT/MRI normal	Bifrontal	Slowed	+, ↑ protein	Steroid	Full recovery	11
14	F	Hallucinations, depression	H	↑	↓	+	MRI ↑T2 frontal lobe	L temporal, frontal, BG	N	N	Thyroxine steroid	Full recovery	†

Yr indicates year; S, sex; M, male; F, female; N, normal; St, status, refers to thyroid status at diagnosis; E, euthyroid; H, hypothyroid; SPECT, localized perfusion deficits; T4, serum thyroxine (total or free); AB, thyroid antibody positivity, including cytoplasmic, microsomal or thyroperoxidase; L, left; BG, basal ganglia, outcome summarizes clinical course as described in cited reference.

* Patients in whom diagnosis of Hashimoto's thyroiditis preceded neuropsychiatric presentation who were on thyroid replacement therapy.

† Current report.

year-old patient with longstanding attentional difficulties which improved with steroid treatment.[9] Our case is unique because of the long (5-year) history of hallucinations and depressed mood.

The MRI findings observed in our patient, with small T2 weighted white matter changes, has been described in other pediatric patients.[10,11] Nevertheless, cranial imaging performed in most pediatric cases reported normal computerized tomography or MRI scans of the head. This differs from the adult literature, as a recent review of patients with SREHT followed at the Mayo Clinic revealed abnormal MRI scans in 57% of patients, a minority (45%) of which resolve with treatment.[12]

An interesting aspect of our case relates to the changes seen in serial SPECT imaging studies over the course of the patient's illness. When treated with thyroid hormone alone, the SPECT showed improved temporal lobe perfusion mirroring clinical improvement in her memory and cognition. These deficits completely resolved on initiation of steroids, although decreased perfusion in the frontal and parietal lobes persisted. Hypothyroid patients with "Hashimoto's encephalitis" with abnormal SPECT scans at diagnosis have been described with full resolution of symptoms on treatment with thyroid hormone alone.[13–15]

Although formal treatment studies are absent, most reports have shown that glucocorticoids are effective. We recommend use of high-dose steroids, initially with intravenous methylprednisolone, followed by a prednisone (1–2 mg/kg/days, max 60 mg/days) for 6 to 8 weeks. The dose should be gradually reduced with expected improvement in symptoms over 3 to 6 months. Despite treatment, patients may experience relapses, and adolescents with this condition may experience residual cognitive deficits.[7,15–17]

Etiologically, SREHT is a controversial diagnosis, as it lacks a precise pathophysiologic basis. We are not aware of any evidence indicative of a causative link between thyroid autoimmunity and encephalitis. CSF analysis has not shown the presence of TPO antibodies or thyroglobulin antibodies in symptomatic adolescent and adult cases.[11,18,19] This condition best describes an association that is postulated to include aspects of endocrine dysfunction, cerebral vasculitis, and impaired autoimmunity.[8,9,17,20,21] In the case of our patient, subclinical hypothyroidism and abnormalities on SPECT scanning, indicative of altered cerebral microvascular perfusion, were found. Thus far, our patient has not shown any evidence of other autoimmune disease, which has been reported in a significant proportion of patients with Hashimoto's thyroiditis.[22]

Despite the speculative origin of SREHT, it remains a treatable encephalopathy. Pediatricians must be aware that younger patients may present with a diverse clinical picture, including seizures, myoclonus, focal neurologic deficits, delusions, and hallucinations. This case is unique as it illustrates that the neuropsychiatric component of this condition may be chronic. SREHT requires a high index of suspicion, and our case stresses the importance of evaluating thyroid function and thyroid antibody titers in adolescent patients with psychiatric presentations.

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REFERENCES

1. Rallison MK, Dobyns BM, Keating FR, Rall JE, Tyler FH. Occurrence and natural history of chronic lymphocytic thyroiditis in childhood. *J Pediatr*. 1976;86:675–682
2. Leigh H, Kramer SI. The psychiatric manifestations of endocrine disorders. *Adv Intern Med*. 1984;29:413–445
3. Asher R. Myxedema madness. *BMJ*. 1949;2:555–562
4. Garrard P, Hodges JR, De Vries PJ, et al. Hashimoto's encephalopathy presenting as myxoedematous madness. *J Neurol Neurosurg Psychiatry*. 2000;68:102–103
5. Hall RCW. Psychiatric effects of thyroid hormone disturbance. *Psychosomatics*. 1983;24:7–18
6. Brain L, Jellinick EH, Ball K. Hashimoto's disease and encephalopathy. *Lancet*. 1966;2:512–514
7. Shaw PJ, Walls TJ, Newman PK, Cleland PG, Cartlidge NEF. Hashimoto's encephalopathy: a steroid-responsive disorder associated with high anti-thyroid antibody titers—report of 5 cases. *Neurology*. 1991;41(Suppl 1):228–233
8. Kothbauer-Margreiter I, Sturzenegger M, Komor J, Baumgartner R, Hess EW. Encephalopathy associated with Hashimoto thyroiditis: diagnosis and treatment. *J Neurol*. 1996;243:585–593
9. Vasconcellos E, Pina-Garza JE, Takoury F, Fenichel GM. Pediatric manifestations of Hashimoto's encephalopathy. *Pediatr Neurol*. 1999;20:394–398
10. Takahashi S, Mitamura R, Itoh Y, Suzuki N, Okuno A. Hashimoto encephalopathy: etiologic considerations. *Pediatr Neurol*. 1994;11:328–331
11. Maydell BV, Kopp M, Komorski GV, Jeugling FD, Korinthenberg R. Hashimoto encephalopathy—is it underdiagnosed in pediatric patients? *Neuropediatrics*. 2002;33:86–89
12. Castillo PR, Broeve PF, Caselli RJ, et al. Steroid-responsive encephalopathy associated with thyroid autoimmunity; clinical and laboratory findings. *Neurology*. 2002;58(Suppl 3):A248
13. Sybesma CA, Pinxtreren-Nogler EV, Sinnige LGF, van Oostrom JCH, deVrieses TW. Hashimoto encephalopathy in a 12 year old girl. *Eur J Paediatr*. 1999;158:876–868
14. Querol Pascual MR, Aguirre Sanchez JJ, Velicia Mata MR, Gahete Jimenez C, Duran Herrera MC, Gonzalez Dorrego F. Encephalitis de Hashimoto: un nueva caso con remission espontanea. *Neurologia*. 2000;15:313–316
15. Watemberg N, Willis D, Pellock JM. Encephalopathy as the presenting symptom of Hashimoto's thyroiditis. *J Child Neurol*. 2000;15:66–69
16. Shien MA, Apter Z, Dickerman S, Tyano N, Gadoth N. Encephalopathy

in compensated Hashimoto's encephalopathy: a clinical expression of autoimmune cerebral vasculitis. *Brain Dev*. 1986;8:60–64
17. Byrne OC, Zuberi SM, Madigan CA, King MD. Hashimoto's thyroiditis—a rare but treatable cause of encephalopathy in children. *Eur J Paediatr Neurol*. 2000;4:279–282
18. Seipelt M, Zerr I, Nau R, et al. Hashimoto's encephalitis as a differential diagnosis of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry*. 1999;66:172–176
19. Archambeaud F, Galinat S, Regouby Y, et al. Encephalopathe de Hashimoto. Analyse de quatre observations. *Rev Med Interne*. 2001;22:653–659
20. Canton A, de Fabregas O, Tintore M, Mesa J, Codina A, Simo R. Encephalopathy associated to autoimmune thyroid disease: a more appropriate term for an underestimated condition? *J Neurol Sci*. 2000; 176:65–69
21. Boers PM, Colebatch JG. Hashimoto's encephalopathy responding to plasmapheresis. *J Neurol Neurosurg Psychiatry*. 2001;70:132–135
22. Becker KL, Ferguson RH, McConahay WM. Connective tissue diseases and symptoms associated with Hashimoto's thyroiditis. *N Engl J Med*. 1963;268:277–280
23. Forchetti CM, Katsamakis G, Garron DC. Autoimmune thyroiditis and a rapidly progressive dementia: global hypoperfusion on SPECT scanning suggests a possible mechanism. *Neurology*. 1997;49:623–626
24. Gucuyener K, Serdaroglu A, Bideci A, et al. Tremor and myoclonus heralding Hashimoto's encephalopathy. *J Pediatr Endocrinol Metab*. 2000; 13:1137–1141
25. Balestri P, Grosso S, Garibaldi G. Alternating hemiplegia of childhood or Hashimoto's encephalopathy? *J Neurol Neurosurg Psychiatry*. 1999;66: 548–549

IS DIVERSITY OVERRATED?

"One of the most comprehensive studies ever undertaken of diversity in higher education indicates that this contention is at least questionable. The study's findings show that college diversity programs fail to raise standards, and that a majority of faculty members and administrators recognize this when speaking anonymously. . . To find out, in 1999 we surveyed a random sample of >1600 students and 2400 faculty members and administrators at 140 American colleges and universities, asking them to evaluate the quality of education at their institution, the academic preparation and work habits of the student body, the state of race relations on campus, and their own experiences of discrimination. . . If diversity works as advertised, we surmised, then those at institutions with higher proportions of black enrollment should rate their educational and racial milieus more favorably than their peers at institutions with lower proportions. . . The results contradict almost every benefit claimed for campus diversity. Students, faculty members, and administrators all responded to increasing racial diversity by registering increased dissatisfaction with the quality of education and the work ethic of their peers. Students also increasingly complained about discrimination. . . One cannot help but wonder why the public and private views of higher education's leadership differ so greatly."

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