ceitfulness, oppositionality, and defiance. Besides mood lability, distractibility, and acts of self-harm, there was no evidence of depression or mania. The patient refused mood stabilizers and atypical antipsychotics because of the possibility of weight gain.

Doses of topiramate started at 25 mg once in the morning for 5 days and then increased to 25 mg twice daily. After 15 days taking 50 mg/day, the patient's dose of topiramate was increased to 25 mg in the morning and 50 mg at bedtime. For the next 7 days, she was administered a dose of 75 mg/day and had no behavioral outbursts. She became more cooperative with staff and did not require restraints. The staff noted a significant improvement in her disruptive behaviors as well.

Although her mania was much improved, she continued to display some emotional lability and distractibility. Therefore, her disruptive behavior symptoms improved independently and more significantly than her mood-related symptoms. She had CGI improvement ratings of 2 for mania and 1 for overall illness. Despite her reports of poor concentration and transient hyperactivity, there was no evidence for these or other side effects, such as weight gain or difficulties with verbal communication.

As illustrated, topiramate monotherapy may be effective in treating disruptive behavior disorders, independent from its therapeutic effect for mania that is possibly related to its efficacy in decreasing impulsivity in binge eating disorder, borderline personality disorder, and pathological gambling disorder in adults. Because of a favorable side effect profile, including possible weight loss, medication compliance may be less of a problem. Further randomized controlled studies of adjunctive and monotherapy topiramate treatment are needed. Future studies should assess aggression as an outcome measure with the Overt Aggression Scale.

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DREW H. BARZMAN, M.D.
MELISSA P. DELBELLO, M.D.
Cincinnati, Ohio

Binge Eating Associated With Internal Carotid Artery Aneurysm

TO THE EDITOR: Prevalence rates of DSM-IV binge eating disorder and intracranial aneurysm have been estimated at 1% (1) and 2%–5% (2). To our knowledge, there has been no report of comorbidity of these two conditions. We describe a case of a patient with internal carotid artery aneurysm presenting with symptoms similar to binge eating disorder.

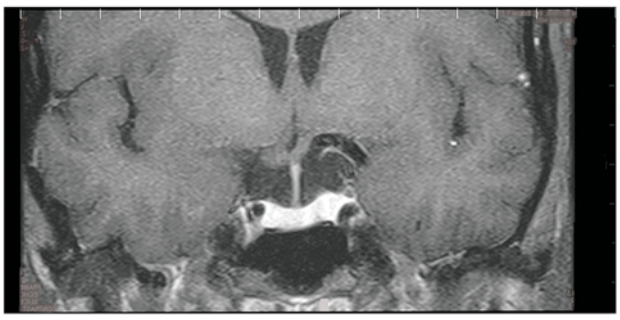
"Mrs. A," a 32-year-old married woman with two children, presented at a psychiatric clinic in Jan. 2003 with depressive symptoms and uncontrollable binge eating episodes that had been recurrent for 2 months. She became fixated with her body weight and waist circumference. Psychosexual stressors from her husband's extramarital affairs were also identified. Neither compensatory behavior nor a history of anorexia was elicited.

Despite alleviation of her depressed mood and preoccupation with body weight following treatment with paroxetine (30 mg/day) and weekly psychotherapy for 4 months, her binge eating episodes persisted. She was then lost to follow-up for several months. Subsequently, she relapsed and presented with the following at her visit in Jan. 2004: binge eating episodes, overconcern with waist circumference, depressed mood, decreased self-esteem, psychomotor agitation, episodic headaches, blurred visions, increased body weight (from 47 kg to 54 kg in 4 months), and increased severity of stress from marital conflict. There was no identified temporal relationship between her headaches and hyperphagia episodes. Similar to prior pharmacotherapy, fluoxetine doses (40 mg/day) were effective in reducing her symptoms of depression and body image distortion rather than controlling her binge eating impulse.

Her physical examination revealed no negative findings except galactorrhea. Neither papilledema nor other focal neurological sign was identified at neurological examination. Her serum level of prolactin was within normal range (8.27 ng/ml). A magnetic resonance image of her pituitary disclosed an engorged vessel from the right internal carotid artery A1 segment compressed on the right optic chiasma and hypothalamus (Figure 1). An angiography showed a wide-based aneurysm (maximum width: 5 mm; height: 4 mm) at the supraclinoid segment of the right internal carotid artery. Her blurred vision and headaches subsided after microsurgical decompression. She did not suffer from any binge eating episode or body image distortion during the postoperation follow-up for 20 months.

An association between bulimia and central nervous system lesions and increased intracranial pressure has been reported previously (3). To our knowledge, Mrs. A is the first reported case of internal carotid artery aneurysm presenting with a binge eating disorder. The disproportional treatment responses and evidence for focal neurological signs suggest the necessity for organic workup. It is advisable to perform detailed physical assessment, endocrinological evaluation, and possible neuroimaging studies in patients with binge eating disorder who have unexpected responses to traditional treatment strategies. Furthermore, our case report may provide additional evidence of a neurological basis of binge eating disorder. One limitation to this report is that it lacks analysis of the potential neural substrates for binge eating disorder.

FIGURE 1. Coronal View of T2-Weighted (TR: 400 msec, TE: 120 msec) Image of Brain MRI Revealing Engorged Vessel Rising From the Right Internal Carotid Artery A1 Segment With Compression on the Right Optic Chiasma and Hypothalamus



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LINEN LIN, M.D.
SUSAN SHUR-FEN GAU, M.D., PH.D.
MING-BEEN LEE, M.D.
Taipei, Taiwan

Facilitation in Inducing Folie a Deux Through Healthy Precipitator

TO THE EDITOR: Folie a deux is a psychotic disorder characterized by a shared delusion that is instigated by a psychotic inducer and a healthy recipient (1). We describe an unusual case in which the psychosis in the recipient appeared only after the most influential member of the family adapted the psychotic ideas of the inducer.

"Mr. G" was a 23-year-old man with no history of psychiatric disorder. He was hospitalized with a paranoid delusion that his brother-in-law was a Mafia leader who had plans to murder him. Mr. G also heard voices telling him to kill himself or he would be subjected to torture. Consequently, he tried to strangle himself in the hospital.

Additional history revealed that Mr. G's sister developed similar symptoms prior to his hospitalization and that she had been in close contact with him during that time. Their mother was aware of her daughter's delusional ideas. While the mother did not believe in the delusions, she provided her daughter with unconditional support and even checked her house for surveillance equipment without asking questions. The mother felt an obligation to maintain family cohesion, even if her actions contradicted her own comprehension of reality. Therefore, she avoided any confrontation with her daughter out of fear the daughter might commit suicide, which is what an uncle did several years previously.

Subsequent to his sister's delusions, Mr. G's first psychotic symptoms appeared when he realized that his mother was fully supportive of his sister's beliefs. Notably, his psychosis exacerbated only after his mother's visits on the psychiatric ward, and his attempt to strangle himself occurred after a visit from her. He received intensive treatment with doses of diazepam (30 mg/day) as well as individual and family psychotherapy that focused on reality testing of both the patient and the family.

It became apparent that the mother played a major role in the family and had indirectly supported Mr. G's psychosis. Following intervention to clarify the mother's influence on Mr. G, she changed her attitude and denied explicitly the existence of his delusions. Consequently, Mr. G began to improve rapidly. After six sessions of family psychotherapy, he was free of psychosis and could be discharged.

It is generally accepted that a dyad composed of a charismatic psychotic inducer and an induced person with dependent character traits is necessary for the development of shared psychosis (2). To our knowledge, the case presented here is the first documented case in which the pathogenic influence of a noncharismatic psychotic inducer was enhanced by a healthy charismatic family leader who was fully supportive of psychotic ideas without sharing those ideas. This case introduces the possibility that a psychotic inducer does not have to be a dominant person in a family. On the other hand, it does emphasize the role of a family leader in transmitting an induced psychosis (3).

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IGOR SALGANIK, M.D.
SHINKARENKO EVGENY, M.D.
PERELROYZEN GALINA, M.D.
near Hadera, Israel

Immobilization Panic

TO THE EDITOR: Fear-associated freezing/immobilization is a well described, adaptive, defensive behavioral phenomenon that is common in many species of animals and occurs during conditions of natural threat or fear. Although freezing behavior has been assessed in humans utilizing stress/anxiety paradigms in the laboratory (1), no study, to our knowledge, has explicitly examined the prevalence of freezing or immobilizing behaviors in a clinical sample.

In our study, we used the NIMH Panic Questionnaire (2), a self-report instrument designed to elicit detailed, syndrome-specific information in patients with panic disorder, to obtain information regarding panic-related, freezing/immobilizing behavior. The frequency ("never," "rarely," "sometimes," "always") and severity ("mild," "moderate," "severe," "extreme") of 44 panic attack symptoms were obtained in a mixed treatment- and community-based sample of 1,118 people who met self-reported DSM criteria for panic disorder. In our analysis, we focused on a single item that determined whether the subjects were actually immobilized during a panic attack.

Among the participants, 198 (18%) reported "always" being immobilized during panic attacks. Fifty-three percent of the participants reported varying frequencies of immobilization panic ("sometimes" [N=405] and "rarely" [N=188]). Thus, 71% of the participants reported lifetime episodes of immobilization panic. Notably, subjects with positive lifetime histories of immobilization panic were 2.3 times (95% confidence interval [95% CI]=1.73–2.95, $p<0.001$) and 1.6 times (95% CI=1.21–2.09, $p<0.001$), respectively, more likely to suffer from disabling chronic anxiety and sleep panic attacks relative to panic disorder patients who had never experienced immobilization panic. Moreover, panic disorder patients who had experienced immobilization panic were 2.4 times (95% CI= 1.64–3.57, $p<0.001$) more likely to also experience work impairment relative to panic disorder patients who did not report immobilization panic. This latter finding is noteworthy because work absenteeism is increased in panic disorder patients relative to nonpanic disorders in primary care settings.

Data presented in this report indicate that immobilizing/freezing behaviors are common, yet clinically underappreciated, events during panic attacks. The fact that patients with