

#### CASES THAT TEST YOUR SKILLS

Ms. D says 'impostors' posing as family members have invaded her house, and 'stalkers' are out to get her. What is causing her paranoid delusions?

# When your brother becomes a 'stranger'

Elizabeth Cerceo, MD Resident, department of medicine University of Pennsylvania, Philadelphia Jeffrey Dunn, MD Professor Thomas Newmark, MD Professor

Department of Psychiatry Robert Wood Johnson Medical School, Cooper Hospital, Camden, NJ

#### HISTORY 'THEY'RE MAKING ME CRAZY'

s. D, age 22, is brought to the emergency room by her older brother for psychiatric evaluation after a family argument. He tells us that his sister is out most nights, hanging out at nightclubs. When she's home, he says, she locks herself in her room and avoids him and his younger brother, who also lives with them.

Recently, her brother says, Ms. D signed a contract to appear in pornographic videos. When he found out, he went to the studio's producer and nullified the contract.

Ms. D, frustrated with her brother's interference, tells us she dreams of becoming a movie star and going to college, but blames him for "holding me back" and keeping her unemployed.

Worse, she says, he and her two sisters are impostors who are "trying to hurt me" and are

"making me go crazy." She fears her "false brother" will take her house if she leaves, yet she feels unsafe at home because strangers—envious of "my beauty and intelligence"—peek into her windows and stalk her. She tells us her father is near and guards her—even though he died 4 years ago.

Ms. D, who lost her mother at age 2, began having psychotic episodes at age 19, a few months after her father's death. At that time, she was hospitalized after insisting that her father had faked his death because of a conspiracy against him. A hospital psychiatrist diagnosed bipolar disorder and prescribed a mood stabilizer, but she did not take the medication and her psychosis has worsened.

Ms. D's Mini-Mental State Examination score of 30 indicates that she is neither grossly confused nor has underlying dementia. However, she is emotion-



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ally labile with grossly disorganized thought processes and paranoid and grandiose delusions.

We could not locate other family members, so Ms. D's family psychiatric history is unknown. She has casual relationships with men but does not have a boyfriend. She acknowledges that she frequents local nightclubs but denies using alcohol.

Blood work and other medical examination results are normal. Negative urine toxicology screen suggests she not abusing substances, and electrolytes and thyroid-stimulating hormone levels are normal. Negative rapid plasma reagin rules out tertiary syphilis. We do not order radiologic studies because her presentation does not suggest focal abnormality, and neurologic exam results are benign.

#### Ms. D's symptoms suggest:

- a) schizophrenia, paranoid type
- b) pharmacologic intoxication/toxic encephalopathy
- c) bipolar disorder with psychotic features
- d) organic brain pathology

#### The authors' observations

Patients with both paranoid delusions and manic features are challenging. Prognoses and treatment options for each group of symptoms differ substantially.

Ms. D's grandiosity, pressured speech, tangential flight of ideas, and hypersexuality strongly suggest bipolar disorder. We could not rule out schizophrenia, however, because of her prominent hallucinations and paranoia.

Pharmacologic intoxication was not likely based on laboratory results and the longstanding, progressive course of Ms. D's disorder. Organic pathology also was unlikely, given her normal neurologic examination and lack of other medical issues.

#### TREATMENT TALK THERAPY

e tentatively diagnose Ms. D as having bipolar disorder type I with a manic episode and psychotic features. She does not meet DSM-IV-TR criteria for schizophrenia and lacks affective flattening, poverty of speech, avolition, and other negative symptoms typical of the disorder. We admit her to the inpatient psychiatric unit and prescribe lithium, 300 mg tid, and quetiapine, 50 mg bid.

An internal medicine (IM) resident visits Ms. D for 30 to 45 minutes daily during her hospitalization to check her medical status and to allow her to vent her frustration. A resident in psychiatry also interviews Ms. D for about one half-hour each day. The patient rarely interacts with other patients and speaks only with physicians and nurses.

Ms. D appears to trust the IM resident and confides in her about her brother. During their first meeting, she appears most disturbed that a man who "claims" to be her brother is sabotaging her life. She does not fear that this "impostor" will physically harm her but still distrusts him. She repeatedly reports that her late father is nearby or in the room above hers. She adds that she feels much safer in the hospital, where the "stalkers" cannot reach her.

At times, Ms. D tells the IM resident she has a twin. Other times, she believes her family is much larger than it is, and she sometimes laments that she is losing her identity. She often perseverates on Judgment Day, at which time she says her "fake" relatives will answer for their actions against her.

Ms. D's delusions of grandiosity, tangentiality, circumferential speech, and flight of ideas persist



through 4 days in the hospital. Her affect is extremely labile and occasionally inappropriate. She sometimes cries when discussing her father's death, then stops, thinks a moment, and begins laughing. At this point, we increase lithium to 600 mg tid and quetiapine to 100 mg tid. She is suffering no side effects and infrequently requires haloperidol as a demand dose only.

Ms. D's symptoms now indicate:
a) bipolar disorder
b) schizophrenia
c) another disorder associated with paranoia

#### The authors' observations

A patient such as Ms. D who lives in a minimally supportive environment and has paranoid delusions could fabricate an explanation for what she perceives as family members' incongruent behavior. She could create a reality in which these relatives are impostors.

Although this behavior is not unusual, Ms. D's extreme reaction toward her siblings suggests Capgras syndrome, a rare misidentification disorder (*Box*). The syndrome is often missed in clinical practice, and its prevalence has not been quantified.

Capgras syndrome is seen most often in patients with paranoid schizophrenia—the highest functioning and most preserved schizophrenia patients. This association may indicate that both neurologic dysfunction and psychological background are necessary to produce the syndrome.

The belief that family members are impostors could point to a conspiracy theory or paranoid delusion. Ms. D's suspicion and distrust toward her older brother indicate a paranoid state, and her other delusions—such as her belief that others are stalking her—suggest that her Capgras symptoms are another manifestation of paranoia. **Capgras' causes.** Capgras delusions can occur sec-

## Capgras syndrome: A disorder that distorts identity

Capgras syndrome—named for Jean Marie Joseph Capgras, a French psychiatrist who first described the disorder—is characterized by paranoid delusions that close friends or relatives are impostors or "doubles" for the family member/friend or are somehow feigning their identity.

Depersonalization and derealization symptoms are common, as is inability to endorse the verity of another's identity. Misidentifications—defined as misperceptions with delusional intensity—can also involve people who do not prompt negative or ambivalent feelings or even inanimate objects.

Capgras syndrome may be neurologically and structurally similar to prosopagnosia which describes inability to recognize familiar faces—but may also be a variation of a paranoid delusion in which the patient seeks to explain affective experiences. The disorder's coexistence with paranoid delusions also suggests an association with schizophrenia.

ondary to neurologic lesions and often appear to have an organic cause, such as abnormal focal paroxysmal discharges.<sup>1</sup> These delusions can occur secondary to systemic infections, thyroid dysfunction, seizures, concussion, intoxication dementia, toxic encephalopathy, or head trauma.<sup>12</sup> Theories vary as to physiologic, structural, and neurologic causes (*Table, page 76*).

For Ms. D, structural brain deficits probably interacted with her psychosocial milieu to create Capgras delusions, though we did not perform confirmatory brain imaging or functional neurologic testing. Whereas right cortical lesions might impair recognition while preserving familiarity, Capgras syndrome preserves recognition but

## Table Proposed causes of Capgras syndrome

#### Physiologic

Frontal lobe damage may distort visual stimuli monitoring, thus impairing facial recognition.<sup>4</sup>

Disruption of neuronal connections within the right temporal lobe scrambles memories needed for facial recognition.<sup>5</sup>

#### Neurologic

Disconnection between brain hemispheres lead to cognitive but not affective recognition.<sup>6</sup>

Bifrontal pathology or other organic cause blurs "judgment of individuality or uniqueness," as in prosopagnosia.<sup>3</sup>

Dorsal pathway impairment alters affective response to faces.<sup>7</sup>

Dissociation in the amygdala may distort affective response to faces.8

#### **Psychological\***

In depression, misidentification develops secondary to rationalizing feelings of guilt and inferiority. $^{\circ}$ 

"Two-armed recognition"—one automatic and almost instantaneous, the other attentive and mnemonic—begins to falter.<sup>10</sup>

Suspicion, preoccupation with details leads to "agnosia through too great attention."<sup>11</sup>

Avoidance of unconscious desires leads to recognition problems.<sup>12</sup>

Patient "projects and splits" family member into two persons; directs love toward real person and hate toward imagined impostor.<sup>13</sup>

In schizophrenia, world is viewed through primitive mechanisms, such as doubles and dualism.<sup>14</sup>

\*Dependent on psychiatric comorbidity

deadens the emotion that makes faces seem familiar. When focal lesions are found to cause Capgras delusion, however, the right hemisphere—specifically the frontal cortex—usually is affected.<sup>2,3</sup>

## How would you diagnose Capgras syndrome?

- a) thorough patient interview
- b) neurologic examinations
- c) discussion with trusted family members

#### The authors' observations

When interviewing a patient with paranoid delusions, get as much detail as possible about

his or her close relationships. Try to interview one or two family members or friends. The information can help determine whether Capgras symptoms underlie paranoia.

Brain imaging might uncover pertinent abnormalities, but the cost could outweigh any benefit. No evidence supports use of CT to diagnose Capgras syndrome. Some evidence supports use of brain MRI, but more research is needed.

No specific treatment exists for Capgras delusions apart from using antipsychotics to treat the psychosis based on clinical suspicion and constellation of symptoms.

Studies have shown no difference in response to atypical antipsychotics between

vasodilatation, thinking abnormal, decreased libido, and sweating. Commonly Observed Adverse Events in Controlled Clinical Trials for MDD, GAD, SAD, and PD—Body as a Whole: asthenia, headache, flu syndrome, Controlled Clinical Trials for MDD, GAD, SAD, and PD—Body as a Whole: asthenia, headache, flu syndrome, accidental injury, abdominal pain. <u>Cardiovascular</u>: vasodilatation, hypertension, papirtation. <u>Digostive</u>: nausea, constipation, anorexia, vomiting, flatulence, diarrhea, eructation. <u>Metabolic/Nutritional</u>: weight loss. <u>Nervous</u> <u>System</u>: dizziness, somnolence, insomnia, dry mouth, nervousness, abnormal dreams, tremor, depression, hypertonia, paresthesia, libido decreased, agitation, anxiety, twitching. <u>Respiratory System</u>: pharyngitis, yawn, sinusitis. <u>Skin</u>: sweating. <u>Special Senses</u>: abnormal vision. <u>Urogenital System</u>: abnormal ejaculation, impotence, orgasmic dysfunction (including anorgasmia) in females. <u>*Vital Sign Changes*: Effexor XR was</u> associated with a mean increase in pulse rate of about 2 beats/min in depression and GAD trials and a mean increase in pulse rate of 4 beats/min in SAD trials. (See WARNINGS-Sustained Hypertension). Laboratory Changes: Clinically relevant increases in serum cholesterol were noted in Effexor XR clinical trials. Increases Charges: Chinaday relevant incleases in seruin choiseator where noted in chikor An chinada indas incleases were duration dependent over the study period and tended to be greater with higher doses. **Other Events Observed During the Premarketing Evaluation of Effexor and Effexor XR**—N=6.670. "Frequent"=events and the least 1/100 patients; "infrequent"=1/100 to 1/1000 patients; "rare"=fewer than 1/1000 patients. **Body as a whole** - Frequent: chest pain substernal, chills, fever, neck pain; Infrequent: face edema, intentional injury, malaise, monitiasis, neck rigidity, pevice pain, photosensitivity reaction, sucide attempt, withdrawal syndrome; Rare: appendicitis, bacteremia, carcinoma, cellulitis. **Cardiovascular system** - Frequent migraine, postural hypotension, tachycardia; Infrequent; angina pectoris, arrhythmia, extrasystoles, hypotension, peripheral vascular disorder (mainly cold feet and/or cold hands), syncope, thrombophlebitis, Rare: aortic aneurysm, arteritis, first-degree atrioventricular block, bigeminy, bundle branch block, capillary radio and a diabatic and a second sec infarct, pallor, sinus arrhythmia. Digestive system - Frequent: increased appetite; Infrequent: Ibruxism, colitis, dysphagia, tongue edema, esophagiis, gastriitis, gastorenteritis, gastrointestinai ducer, gingvitis, glossitis, rectal hemorrhage, hemorrhoids, melena, oral moniliasis, stomatitis, mouth ulceration; Rare: abdominal distension, billary pain, chelitis, cholecystitis, choleithihaisis, esophageal spasms, duodenitis, hematemesis, gastroesophageal reflux disease, gastrointestinal hemorrhage, gum hemorrhage, hepatitis; lieitis, laundice, intestinal obstruction, liver tenderness, parotitis, periodontitis, proctitis, rectal disorder, salivary gland enlargement, increased salivation, soft stools, tongue discoloration. Endocrine system - Rare; galacotrhoea, goiter, hyperthyroidism, hypothyroidism, thyroid nodule, thyroiditis. <u>Hemic and lymphatic system</u> - Frequent: ecchymosis; Infrequent: anemia, leukocytosis, leukopenia, lymphadenopathy, thrombocythemia; Rare; basophila, bleeding time increased, cyanosis, eosinophila, lymphadopotysis, multiple myeloma, purpura, thrombocytopenia. <u>Metabolic and nutritional</u> - Frequent: edema, weight gain; Infrequent: alkaline obsobatase increased dehvdration. hvoercholesteremia. hvoerolveenia. hyposphatase increased, dehydration, hyporcholesteremia, hyporglycemia, hyporlipemia, hypoglycemia hypokalemia, SGOT increased, SGPT increased, thirst; Rare: alcohol intolerance, bilirubinemia, BUN increased, creatinine increased, diabetes mellitus, glycosuria, gout, healing abnormal, hemochromatosis, hypercalcinuria, hypperkalemia, hyperphosphatemia, hyporatemia, gud, neaming admirma, hyponatemia, hypophatemia, hypo Introporteinemic and a structure intervential and the structure interventis and the struct dermatuis, incriencio dermatuis, nar oiscoloration, sixin oiscoloration, nurniculosis, instisuism, ieukodermia, miliaria, petechial rash, puritic rash, pustular rash, vesiculobullous rash, seborrhea, skin atrophy, skin hypertrophy, skin striae, sweating decreased. <u>Special senses</u> - Frequent: abnormality of accommodation, mydriasis, taste perversion; infrequent: conjunctivitis, diplopia, dry eyes, eye pain, hyperacusis, otitis media, parosmia, photophobia, taste loss, visual field defect; Rare: blepharitis, cataract, chromatopsia, conjunctivial edema, corneal lesion, deafness, exophthalmos, eye hemorrhage, glaucoma, retinal hemorrhage, subconjunctival hemorrhage, keratitis, labyrinthitis, miosis, papilledema, decreased pupillary reflex, otitis subconjunctival nemormage, keratus, ladynninus, miosis, papiliedema, oecreaseb opupiliary reitex, outis externa, scientis, uveitis, **Urogenital system** - Frequent: prostatic disorder (prostatitis, enlarged prostate, and prostate irritability), urination impaired; Infrequent: albuminuria, amenorrhaa, breast pain, cystitis, dysuria, hematuria, kidney calculus, kidney pain, leukorrhea, menorrhagia, metrorrhagia, nocturia, polyuria, pyuria, urinary incontinence, urinary referiton, urinary urgency vaginal hemorrhage, vaginitis; Rare: abortion, anuria, batetina (Expression beast discharge, breast engorgement, breast enlargement, endometriosis, female Instellis, bladder pain, breast discharge, breast engorgement, breast enlargement, endometriosis, female creation. lactation, fibrocystic breast, calcium crystalluria, cervicitis, orchitis, ovarian cyst, prolonged erection, gynecomastia (male), hypomenorrhea, kidner function abnormal, mastitis, menopause, pyelonephritis, oliguria, asplngitis, urolithiasis, uterine hemorrhage, uterine spasm, vaginal dryness. **Postmarketing Reports**: agranulocytosis, anaphylaxis, aplastic anemia, catatonia, congenital anomalies, CPK increased, deep vein fibrillation, supraventricular tachycardia, ventricular extrasystoles, and rare reports of ventricular fibrillation and ventricular tachycardia, including torsades de pointes; epidermal necrosis/Stevens-Johnson syndrome. and vonitcular and the second se second sec who may have been taking methylphenidate, was treated and recovered), neutropenia, night sweats, pancreatitis, pancytopenia, panic, prolactin increased, pulmonary eosinophilia, renal failure, rhabdomyolysis, serotorin syndrome, shock-like electrical sensations or tinnitus (in some cases, subsequent to the discontinuation of venlafaxine or tapering of dose), and SIADH (usually in the elderly). Elevated clozapine levels that were temporally associated with adverse events, including seizures, have been reported following the addition of venlafaxine. Increases in prothrombin time, partial thromboplastin time, or INR have been reported when venlafaxine was given to patients on warfarin therapy. DRUG ABUSE AND DEPENDENCE: Efferor XR is not a controlled substance. Evaluate patients carefully for history of drug abuse and observe such patients closely for signs of misuse or abuse. **OVERDOSAGE**: Electrocardiogram changes (e.g., prolongation of QT interval, bundle branch block, QRS prolongation), sinus and ventricular tachycardia, bradycardia, hypotension, altered level of consciousness (ranging from somnolence to coma), rhabdomyolysis, seizures, vertigo, liver necrosis, and death have been reported. Treatment should consist of those general measures employed in the management of overdosage with any antidepressant. Ensure an adequate airway, oxygenation and ventilation. Monitor cardiac rhythm and vital signs. General supportive and symptomatic measures are also recommended. Induction of emesis is not recommended. Gastric lavage with a large bore orogastric tube with appropriate airway protection, if needed, may be indicated if performed soon after ingestion or in symptomatic patients. Activated charcoal should be administered. Due to the large volume of distribution of this drug, forced diversis, dailysis, hemoperfusion, and exchange transition are unlikely to be of benefit. No specific antidotes that were temporally associated with adverse events, including seizures, have been reported following the diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. No specific antidotes for venlafaxine are known. In managing overdosage, consider the possibility of multiple drug involvement. Consider contacting a poison control center for additional information on the treatment of overdose. Telephone numbers for certified poison control centers are listed in the Physicians' Desk Reference® (PDR), DOSAGE AND Numbers for certified poison control centers are used in the Physicians Desk Reference (PCN), DUSAGE AND ADMINISTRATION: Consult full prescribing information for dosing instructions. Switching Patients to or From an MAOI—At least 14 days should elapse between discontinuation of an MAOI and initiation of therapy with Effexor XR. At least 7 days should be allowed after stopping Effexor XR before starting an MAOI (see CONTRAINDICATIONS and WARNINGS). This brief summary is based on Effexor XR Prescribing Information W10404C019, revised November 2005.

#### continued from page 76

patients with schizophrenia and comcomitant Capgras symptoms and those with schizophrenia alone. In clinical practice, we have found that treating Capgras symptoms does improve schizophrenia's course.

Adjunctive psychotherapy has not been studied in Capgras syndrome, and directed, insightguided therapy might not resolve deeply rooted delusions for some patients. With Ms. D, however, "talk therapy" helped us build rapport and gave us insight into her strained familial relationships. Establishing a therapeutic alliance with the patient and encouraging healthy relationships with his or her family and friends can mitigate the effects of Capgras paranoia.

#### CONTINUED TREATMENT GRADUAL CHANGE

ay by day Ms. D's mania subsides gradually, though she still fears that a stranger posing as her brother is stalking her. She talks about her brother less frequently, though she is clearly holding fast to her delusional beliefs.

We discharge Ms. D after 10 days. Although her symptoms have not resolved, she is markedly less manic and less agitated than at admission. We arrange treatment with outpatient psychiatry. She does not follow up with her original psychiatrist and is lost to follow-up.

**C**apgras syndrome can underlie paranoid delusions and can manifest as suspicion toward family and friends. Although its impact on outcomes has not been established, clinical experience suggests that recognizing Capgras symptoms and gaining the patient's trust can improve his or her course.

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#### Related resources

- PsychNet-UK. Disorder information sheet: Capgras (delusion) syndrome. www.psychnet-uk.com/dsm\_iv/capgras\_syndrome.htm.
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#### DRUG BRAND NAMES

Haloperidol • Haldol Lithium • Eskalith, others Quetiapine • Seroquel

DISCLOSURES

The authors report no financial relationship with any company whose products are mentioned in this article, or with manufacturers of competing products.

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