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Serotonin syndrome: not a benign toxidrome

Philippe Birmes and associates[1] suggest that serotonin syndrome is a less serious condition than neuroleptic malignant syndrome (NMS), but this has not been our experience.[2-5] In our prospective study of serotonin syndrome,[4,5] 6 of the 16 patients experienced disseminated intravascular coagulation (DIC), rhabdomyolysis and hypotension necessitating admission to the intensive care unit. Acute renal failure developed in 2 patients, and 1 patient died.

Table 2 in the article by Birmes and associates[1] does not capture the key differences between NMS and serotonin syndrome. Both conditions can be fulminant, and patients may present with delirium, hyperthermia, rhabdomyolysis, dilated pupils, tachycardia, daphoresis, rigidity and blood pressure changes[2-5] (see Table 1 with this letter). The main difference lies in the clinical gestalt: typically a patient with serotonin syndrome is agitated, speaks incoherently and has prominent myoclonus, whereas a patient with NMS is immobile, mute and staring. Although rhabdomyolysis is a complication of both toxidromes, DIC, seizures, ventricular tachycardia and severe hypotension are extremely rare in NMS.[2]

We agree with the mainstays of treatment suggested by Birmes and associates,[1] but we also advise monitoring of vital signs, platelet count, muscle enzymes and myoglobin twice daily for at least 72 hours. We have serious concerns about the use of chlorpromazine and propranolol for serotonin syndrome. Both drugs decrease blood pressure, which will exacerbate the hard-to-treat hypotension that can occur in serotonin syndrome; in addition, chlorpromazine may precipitate NMS. An absolute contraindication for the use of propranolol is a history of asthma, which is difficult to elicit if the patient is delirious. Finally, it is important to advise patients taking serotonergic agents about the risks of this potentially serious and fulminant syndrome.

Table 1: Clinical characteristics, laboratory abnormalities, complications, and risk factors for neuroleptic malignant syndrome and serotonin syndrome[2-5]

Characteristic	Neuroleptic malignant syndrome	Serotonin syndrome
Typical clinical presentation	Rigid, mute, staring, immobile	Agitated, incoherent speech, myoclonic twitching, bruising
Cognitive	Mild confusion to delirium; difficult to assess because of mutism	Mild confusion to delirium
Autonomic	Fever, tachycardia, diaphoresis, dilatation of pupils, blood pressure instability	Fever, tachycardia, diaphoresis, dilatation of pupils, blood pressure instability (hypertension in moderate cases, hypotension in severe cases)
Gastrointestinal	Constipation, ileus	Nausea, vomiting, diarrhea
Neurologic	Severe muscular rigidity (cogwheel), rigours, tremulousness	Hyperreflexia, myoclonus, tremulousness, clonus, fasciculations, ataxia, with or without rigidity
Psychiatric	Facial expression "fearful," underlying psychosis, premorbid mood disorder	Underlying mood or anxiety disorder; delirium may be misinterpreted as psychosis
Common laboratory abnormalities	Leukocytosis, elevation of muscle enzymes (CPK, ALT, AST, LDH), low serum iron	Leukocytosis, elevation of muscle enzymes (CPK, ALT, AST, LDH), thrombocytopenia
Complications	Aspiration pneumonia, renal failure, pulmonary embolus, contractures, postepisode muscle weakness	Falls, seizures, severe hypotension, ventricular tachycardia, disseminated intravascular coagulation, renal failure, coma (mortality rate unknown)
Risk factors	Antipsychotic drug use (all types), polypharmacy, rapid increase in neuroleptic dosage, concurrent use of lithium, dehydration, catatonia, agitation, benzodiazepine withdrawal during neuroleptic treatment	Use of serotonergic agents (all types), polypharmacy, concurrent use of lithium, MAOIs plus demerol (other risk factors unknown)

Note: CPK = creatine phosphokinase, ALT = alanine aminotransferase, AST = aspartate aminotransferase, LDH = lactic dehydrogenase, MAOIs = monoamine oxidase inhibitors.

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Smith-Magenis syndrome

Waleed Al Busairi and Fawzi Ali[1] describe a 15-year-old boy with mental retardation and a history of putting inedible objects into his mouth. The authors might want to investigate

for Smith-Magenis syndrome if this has not previously been considered.

Smith-Magenis syndrome is associated with mental retardation, sleep disturbances, few facial dysmorphic features, self-injurious behaviour and putting objects into orifices. This trait of bodily insertions is known as polyembolokoilamania.[2] The definitive diagnosis is based on absence of the 17p11.2 region (a band on the short arm of chromosome 17), determined by cytogenetic examination (in more than 95% of cases[2,3]) or by fluorescence in situ hybridization (also known as FISH).

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[One of the authors responds:]

We did not consider Smith-Magenis syndrome for the patient described in our article.[1] This chromosomal microdeletion syndrome is associated with a clinically recognizable pattern of physical, developmental and behavioural features.[2] The facial appearance is characterized by broad, square shape, brachycephaly, prominent forehead, synophrys, upslanting palpebral fissures, deep-set eyes, broad nasal bridge, midfacial hypoplasia and prognathism. The behavioural phenotype includes sleep disturbance, attention deficit disorders, attention-seeking, aggression, self-injurious behaviour and stereotypes, especially the self-hug and lick-and-flip movements.

We suspect that Chitra Prasad raised the possibility of Smith-Magenis syndrome because the patient was mentally retarded and ingested foreign bodies. However, 2 important distinctions must be made. First, most people with Smith-Magenis syndrome have mild to moderate mental retardation, whereas this patient had severe to profound retardation. Second, the syndrome is associated with polyembolokoilamania, the insertion of objects into body orifices such as the rectum, vagina, urethra, nose and ear, rather than pica, in which ingestion is restricted to the oral route, as in the patient we described. Smith-Magenis syndrome is rare, occurring in 1 of 25 000 births, but pica affects some 20% of mentally retarded people.[3]

Other facts about this patient, not given in the article, made a diagnosis of Smith-Magenis syndrome unlikely. For example, the patient did not show the distinctive facial appearance or behavioural phenotype of this syndrome. Furthermore, virtually all cases of Smith-Magenis syndrome occur de novo, whereas the patient's family included other mentally retarded siblings, which indicated an inherited abnormality.

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Correction

In a recent article on adaptation of Inuit children to a low-calcium diet,[1] the units for the urinary calcium to creatinine ratio were given incorrectly. The units in the text and the table should have been moles per mole (mol/mol). Note that the numeric values for both the study results and the normative values are correct as presented.

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