Neuroleptic malignant syndrome revisited!

Sir,

I have read carefully a very interesting case report by Zolezzi and Al-Hathloul.[1] However, there are few relevant points, which need more elaboration. According to my analysis, this young female patient presented for the first time to hospital Accident and Emergency (A&E) with acute catatonic excitement characterized by severe aggression, agitation and positive psychotic features, which invited immediate administration of haloperidol and lorazepam. A variety of interlocking factors including hospital admission and discharge policies, key relative as well as patient' decision, and attending doctor's assessment play an important role in hospitalizing a patient. However, this female patient could have been admitted, even involuntarily, for managing first psychotic episode or preventing or aborting untoward events. Beside the issue of admission, this case report raises some other queries. Was this patient discharged home without any recommendations to follow? Was she prescribed any psychiatric treatment from A&E? Was she advised to consult a psychiatrist or was a psychiatric consultation sought when she presented to hospital A&E? Clinical experience guides that a single 5 mg dose of haloperidol combined with suitable benzodiazepine more often than not aborts the excitement phase of catatonic schizophrenia. Subsequently, patients switch into its retarded/withdrawn phase unaccompanied or rarely accompanied by impending features of neuroleptic malignant syndrome (NMS), as probably happened in this case. In contrast to excited phase, the retarded phase, usually inherent with negativism together with overall poor compliance, is usually ignored by the immediate naïve relatives in terms of overall care including early psychiatric consultation, drug treatment, and food and fluid intake. But newly emerging dangerous features in terms of extrapyramidal manifestations, thermoregulatory dysregulation, impaired consciousness, and other dysautonomic functions warn the relatives to seek medical help. Like the present case, this help is often sought late by a few days to one week or more. However, direct admission of this patient from A&E to medical/psychiatric ward could have curtailed this delay. Further, clinicians could have suspected early the diagnosis of NMS along with proper immediate intervention. Although catatonia could be caused by a variety of medical conditions and psychiatric disorders, neither provisional psychiatric diagnosis nor etiology of catatonia was extensively explored in this case. However, several investigations were carried out to exclude any organic cause for hyperthermia and related symptoms. This diagnostic clinical point is relevant because patients with catatonia-caused by psychiatric disorders such as schizophrenic psychosis and bipolar disorder usually need re-initiation of neuroleptic therapy, of course different from offending medication. The neuroleptic therapy or novel antipsychotic drug should be given after 2-weeks of complete recovery from NMS that is better indicated by return of creatinine phosphokinase to normal level. The authors did not provide any such information. Children developing NMS with poor prognosis associated with low potency neuroleptics are known to recover over a long period of time, sometimes up to 119 days.[2] This particular case appears to be atypical as regards the resolution of NMS over 86 days. A patient with uncomplicated NMS usually takes 7-10 days to recover. Continuation of oral lorazepam could have enhanced the recovery phase in particular catatonic symptoms, which are usually confused with extrapyramidal rigidity. In cases of NMS coupled with persistent psychotic symptoms, electroconvulsive therapy is a good option. Finally, the authors have not highlighted any risk factor for developing NMS. Catatonia is the harbinger of NMS and high potency neuroleptic, constant agitation, aggression, stress of psychological disorder, and physical exhaustion are other obvious risk factors in this patient. Our research team has discussed the risk factors[3] and also extensively reviewed the relevant literature together with clinical case presentations.[4-6] Sympathoadrenal hyperactivity, stress of psychological disorders, genetic trait vulnerability, CNS dopamine neurotramission disruptions, and calcium-mediated signal transduction mechanisms and regulatory proteins are some of the new speculative etiopathogenic hypothesis of NMS.[7,8,9]

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Reply from the author

We are flattered that our case report on NMS has been able to generate interest in the part of the readers of the Saudi Medical Journal. All the questions raised by Dr. Qureshi about this case are valid, in particular, with regards to differential diagnosis. Catatonia was certainly thoroughly investigated in our case and it was part of the

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provisional psychiatric diagnosis for this patient, although for reasons inherent to case report publications, we did not explain in detail. As an extensive review of NMS was not the overall objective of presenting this case, we did not highlight risk factors associated with NMS. Our main objective was to stress the importance of early recognition of this adverse reaction as delayed intervention has been recognized in resulting in slow recovery and prolonged hospitalizations. We thank Dr. Qureshi for his comments, as they have been able to clarify a few points regarding NMS that we did not mention in our case report.

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Erratum

In manuscript "Are women at an increased risk of gestational thyrotoxicosis?" Saudi Medical Journal 2002; Vol. 23 (6) 651-657, the spelling of an authors name in the article should have appeared as "Tsuruta E".

Erratum

In manuscript "Restorative proctocolectomy with ileal reservoir" Saudi Medical Journal 2002; Vol. 23 (6) 667-671, an author's name mentioned in the references section should have appeared through the text as "Parks and Nicholls.[6]

Erratum

In manuscript "Growth status of Saudi patients with cleft lip and palate" Saudi Medical Journal 2002; Vol. 23 (7): 823-827, the authors names should have appeared as follows:
Sahar F. Barakati, Eman A. Alkofide. Also Table 2, column 3, should have appeared as follows:
UCLP and BCLP