Attention-Deficit Hyperactivity Disorder in a Sample of Omani Schoolboys

Dear Editor: Attention-deficit hyperactivity disorder (ADHD) is a common disorder affecting schoolchildren and adolescents (1,2). Studies conducted elsewhere (especially in the West) suggest a prevalence rate ranging from 3% to 15% among schoolchildren, although even higher figures have been reported. ADHD affects boys 3 to 10 times more than girls and is characterized by high comorbidity. Delayed diagnosis and inadequate treatment of ADHD can lead to repeated school failure, antisocial behaviour, road traffic accidents, family problems, and delinquencies. Research on ADHD from developing countries is scarce, despite the preponderance of youth in their communities. Conners’ Rating Scale is a screening tool that is widely used in both community and hospital studies to detect hyperactivity, inattention, and behaviours related to ADHD (3,4). It has been translated into Arabic and validated (5,6).

We report on a study, the objective of which was to quantify the rate of hyperactivity and to examine its psychosocial and academic correlates in schoolboys in the Sultanate of Oman, a country with a youth-based population of 2 million, situated in the southeastern part of the Arab Peninsula. This is the first such study among Omani schoolboys.

During 2002, we obtained informed consent and studied 1502 schoolboys, aged 6 to 14 years, from 8 randomly selected public elementary schools in Muscat. Their teachers completed the short form of Conners’ Teacher Rating Scale (CTRS) and also gave their subjective view of the pupil’s behaviour regarding aggression, stealing, and lying. Pupils with a CTRS score ≥ 15 were considered probable cases of ADHD. Social workers assisted in providing the children’s demographic and school achievement variables. School performance was determined by results in both mathematics and Arabic, as well as by school failure(s) in previous years. Pupils whose grades were under 50% in one or both subjects were considered to be the poorly achieving group. SPSS software (7) was used for the data analysis, and the results of the chi-square test were considered significant if \( P \leq 0.05 \).

Of the 1502 schoolboys, 117 (7.8%) had a CTRS score ≥ 15 and were considered to represent the hyperactive group. This group exhibited aggressive, stealing, and lying behaviour more than their counter group (\( P < 0.001 \)). Also, they had poor school achievement in both the first term of the current school year, as well as failure(s) in the previous year (\( P < 0.001 \)). The birth order of the group exhibiting hyperactivity and the total number of their siblings did not differ significantly from the rest of the sample (\( P = 0.10 \) and \( P > 0.05 \), respectively). The paternal education of the boys showing hyperactivity was lower than that of the normative group (\( P = 0.05 \), while the maternal education was more or less similarly distributed in both groups (\( P = 0.25 \)). This study confirms the presence of hyperactivity in Omani boys and is consistent with results of other studies in different countries. This finding is of high importance with regard to planning for primary, secondary, and tertiary prevention of this disorder in Oman. The poor school performance among the group with hyperactivity may be attributable to the disturbed cognitive functions that characterize such children (8,9). Because ADHD is a neurobiological (within-child) disorder, environmental factors do not play a role in its causation; however, the nature of that environment could influence its outcome, positively or negatively.

Acknowledgement
We are grateful to Ms Zena Al-Sharbati, University of Toronto, for helping in the preparation and the statistical analysis of this letter.

References
praying). The obsessive–compulsive symptoms first appeared after the remission of his first manic episode; however, embarrassment and feelings of guilt prevented him from reporting them.

He received a diagnosis of OCD according to DSM-IV criteria. His original score on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) was 20. Olanzapine 15 mg daily was added to his therapeutic regimen to treat the obsessive–compulsive symptoms. This medication was preferred over antidepressants because the latter may precipitate manic episodes. The addition of olanzapine led to clear improvement in the obsessive–compulsive symptoms over a period of 6 weeks, at the end of which time his Y-BOCS score was 6.

**Discussion**

The treatment of patients with comorbid BD and OCD appears rather problematic. It seems that mood stabilizers alone do not suffice to control obsessive–compulsive symptoms (5), although there have been some positive experiences with lithium (6), as well as with topiramate and lamotrigine (1). The use of antidepressants risks inducing mania or rapid cycling (1), and treatment of mania with classic neuroleptic drugs may lead to worsened obsessive–compulsive symptoms (3). However, there seems to be some hope in the use of the newer, atypical antipsychotic agents. The role of these drugs in treating BD is now well established (7), and there is some evidence that such agents are efficacious in treating OCD. Most reports concern cases of refractory OCD (8–10), but it has been suggested that such cases could imply bipolarity (5).

Owing to the prognostic and therapeutic implications mentioned, it is most important to carefully assess BD patients for the existence of obsessive–compulsive symptoms. It should be borne in mind that specifically oriented questioning is necessary to disclose such symptoms, given that patients often conceal them out of ignorance or embarrassment (4), as in the case we report. Atypical antipsychotics such as olanzapine act favourably on obsessive–compulsive symptoms, as well as on BD, and could thus be of use in patients with comorbid BD and OCD.

**References**


Petros Petrikis, MD, Christina Andreou, MD Vasilis P Bozikas, MD PhD; Athanasios Karavatos, MD, PhD

**Monoamine Oxidase Inhibitors and Subarachnoid Hemorrhage**

**Dear Editor:** Hypertensive crisis with monoamine oxidase inhibitor (MAOI) use has been reported in the literature and carries an overall risk of less than 1% (1). The reported reactions were triggered by diet noncompliance or by the use of concomitant medications. Specific agents responsible for these crises include tricyclic antidepressants (TCAs), meperidine, levodopa, and hay fever and cold remedies (2–4). However, spontaneous hypertensive reactions have also been described (1). Here, we present the first case of a subarachnoid hemorrhage (SAH) observed over the last 35 years that was likely caused by a hypertensive crisis secondary to MAOI use (5).

**Case Report**

A man, aged 52 years, with a long-standing history of treatment-refractory atypical depression presented to hospital complaining of a severe headache. The patient’s medical history did not suggest any risk factors for SAH. Five weeks prior to admission, he had begun taking oral tranylcypromine with a dosage escalating to 20 mg daily. One week after starting this medication, he complained of the "worst headache of his life" after consuming a meal of chicken teriyaki that included a half-tablespoon of soy sauce. When he presented at a local emergency room, his blood pressure was elevated to a systolic pressure of 210 mmHg. A noncontrast CT scan was performed. The scan revealed 2 small, hyperdense foci in the left frontal lobe and left lateral occipital lobe, with surrounding edema, consistent with an SAH. There was no evidence of hydrocephalus. His GCS score was found to be 13 to 14, and we were consulted in regard to delirium. The patient was unable to follow commands and, interestingly, had evidence of perseveration. Routine investigations were normal. A cerebral angiogram showed no evidence of aneurysm or vascular malformation and only showed vasospastic changes along branches of the left middle cerebral artery. An investigation for vasculitis was also negative.

His delirium was left untreated so that his level of consciousness could be accurately followed. The tranylcypromine and a neuroleptic for the treatment of delirium were withheld owing to concerns on the part of the neurosurgery service. The delirium resolved, and he was discharged after 17 days.

On follow-up, the patient opted for no further treatment with medication and was offered a trial of interpersonal therapy. He relied upon Tylenol 3 to manage his headache symptoms, which dissipated over time.

**Discussion**

Multiple factors led to the conclusion that this SAH was triggered by MAOI use. The patient’s consumption of soy sauce, a recent increase in tranylcypromine dosage, ingestion of tranylcypromine close to mealtime, and a medical history devoid of SAH risk factors all suggest that MAOI use was a precipitating factor. Further, negative angiography and a laboratory workup helped rule out other possible causes. It is interesting to note that the patient’s outpatient psychiatrist considered treating his depression with a TCA, which has been cited to precipitate SAH in some patients (4). MAOI agents can be effective antidepressants, but as this case highlights, physicians and patients should be aware of their propensity to cause SAH.

**References**

Dear Editor: In emergency departments, haloperidol is the antipsychotic most frequently used to manage violent patients (1). Although psychiatrists use various typical and atypical antipsychotics for this indication (2), emergency physicians continue to favour haloperidol. It is a safe and reliable medication for acute aggression (1), but there are additional factors to consider. For example, most patients requiring an antipsychotic for ongoing treatment will be prescribed a novel or atypical antipsychotic such as risperidone, olanzapine, or quetiapine. Using the same medication for both acute and ongoing treatment improves continuity, reduces risk of relapse while switching medications, and reduces the risk of medication interactions. Moreover, alternate strategies are useful if a patient states a strong medication preference or has experienced an allergic or side effect reaction to a particular medication. Further, patients will sometimes require intramuscular (IM) antipsychotics. However, in Canada, only typical antipsychotics are available in IM formulation. In the US, ziprasidone IM has recently been approved for treatment of acute agitation in patients with schizophrenia (3), while olanzapine IM is moving toward approval in Canada and in the US (3). Many typical antipsychotics are as safe and effective as haloperidol and can be given IM. These include trifluoperazine, fluphenazine, thiothixene, and loxapine (1.2). With regard to the latter, loxapine has a 5-HT₂–dopamine D₂ receptor occupancy ratio more characteristic of atypical than typical antipsychotics and, thus, a lower risk of extrapyramidal symptoms (2).

Many hospitals have psychiatrists readily available to the emergency department, yet emergency physicians still regularly manage patients showing acute aggression. Emergency physicians are intermittently exposed to continuing medical education, to pharmaceutical company presentations, and to informal discussions with psychiatry staff regarding medication management of aggression. Nevertheless, haloperidol remains the antipsychotic of choice.

At our centre, postgraduate year 1 emergency medicine (EM) residents are required to complete a 1-month psychiatry rotation. During residency year 2002–2003, EM residents for the first time completed an emergency psychiatry rotation. Each resident was specifically educated in the management of agitation and aggression. This included a didactic session, teaching focused on cases, and supervised case management. At the beginning of their rotations, all 4 EM residents were aware of the haloperidol 5 mg plus lorazepam 2 mg approach to managing aggression. By the end of the rotation, residents were comfortable with some other approaches, including use of loxapine, risperidone, and olanzapine. One resident had the opportunity in the emergency department to use loading dosages of olanzapine 20 mg for 3 different patients showing agitation, with excellent benefit (4). Moreover, in ensuing months, EM residents continued to use these strategies and to discuss them with colleagues.

EM residents benefit from a specific rotation in emergency psychiatry because it is so relevant to their future practice. Further, psychiatry patients benefit from a more patient-centred, individualized approach to medication management, even in the acute setting.

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References

Susan Finch, MD, CM, FRCPC
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Leon Sher, MD
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A Romanian Adoptee’s Journey From Latency Age to Adolescence

Dear Editor: Studies on attachment and prenataI environment demonstrate difficulty with attachment and physical and neurocognitive functioning in children who suffered significant early adversity (1). The plight of children in Romanian orphanages became known following the fall of the Ceausescu regime in 1989. Some of these children were adopted into Canadian families (2), and as they reach adolescence, some are presenting with psychiatric morbidity consistent with research findings (3).

I report a case wherein the developmental challenges of adolescence brought to the fore the effects of disrupted attachment without cognitive damage that had no harbinger in the patient’s latency years.

Case Report
Jane, aged 15 years, was adopted at age 2 years by a Canadian family. Prior to adoption, she had lived for 8 months in a Romanian orphanage. At her initial introduction to the family, Jane was seen to be overfriendly (that is, she showed indiscriminuous approach behavior). She adapted rapidly to her new home in Canada. Upon starting school, she coped well socially and academically.

Jane’s first contact with child psychiatry was at age 11 years and was initiated by her adoptive mother as a proactive consultation prior to taking her to Romania to meet her biological mother. Her adoptive mother wanted to ensure that this meeting would not have a detrimental effect on Jane’s psychological well-being. Jane was seen after she returned from the visit and appeared to be coping very well. No evidence of any psychiatric disorder was observed. Her adoptive mother was noted to be a warm and caring woman who provided well for Jane’s needs.

At age 14 years, Jane presented in crisis to the emergency room after having expressed homicidal thoughts toward a younger sibling. The imminent arrival of a fourth adoptee into the home was noted to be a precipitant. While obviously distressed by these thoughts, Jane was adamant that she was capable of carrying them out and stated that she had similar thoughts toward a teacher. Her family’s assurance of their commitment to her and reminder about the implications of such threats had no effect. Jane stated that she would happily move to a different family.

This led to hospitalization for crisis intervention and stabilization. Cognitive assessment showed average intelligence. Attachment problems, together with early neglect and deprivation, were considered etiologic factors in a diagnosis of undersocialized aggressive conduct disorder. Difficulties in Jane’s attachment patterns were noted. She received no other psychiatric diagnosis. She was charged for her threats and remanded to a youth facility for a period of 1 month. The family was very reluctant to have her return home. Family therapy was offered to address problems within the family. This was difficult, because they felt they had successfully assimilated their children and saw Jane’s behaviors as a problem limited to her alone.

The developmental tasks of adolescence, including the bid for autonomy and identity formation with the ambivalent need for security, serve as additional stressors for the insecurely attached and can disrupt a tenuous attachment. In Jane’s case, a nurturing and consistent environment had contained and provided security during her latency age, but it was unable to contain the adolescent thwarted by yet another younger child needing to share in her mother’s affections. This could have caused feelings of abandonment to emerge, together with escalating attention seeking alternating with hostile rejection of the family. The family felt bewildered by the changes in this girl, and it required an expert family therapist to help them come to terms with the limitations imposed on her by early experiences.

References


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Stéatose hépatique non alcoolique secondaire à la clozapine


Cas
Suivi depuis 1990 pour un trouble schizo-affectif de type bipolaire, Mme K, 46 ans, souffre d’angoisse envahissante avec pensées catastrophiques, hallucinations auditives, illusions visuelles et idées suicidaires malgré plusieurs approches pharmacothérapeutiques. Un essai à la clozapine jusqu’à concurrence de 200 mg par jour s’est avéré le seul traitement efficace. On a ainsi noté la disparition des symptômes psychotiques, une nette amélioration des habiletés sociales et un soulagement des velléités suicidaires. Elle
recevait de façon concomitante citalopram, à raison de 20 mg par jour. Après deux ans, il y a malheureusement eu un gain pondéral significatif (35 kg; IMC = 34) et une constipation sévère réfractaire. Un suivi métabolique périodique a d’ailleurs démontré une élévation des enzymes hépatiques : AST à 55 et ALT à 100, soit le double de leurs valeurs normales (10 à 40 U/L). Les sérologies virales pour les hépatites, le HIV et le CMV se révèlent négatives. Une échographie abdominale objective une stéatose hépatique diffuse très importante sans autre anomalie des voies biliaires. Une évaluation en gastroentérologie conclut à une stéatose sévère secondaire à la prise de poids rapide et à une légère cytolysie concomitante. Le sevrage de la clozapine est décidé en raison de l’inconfort marqué de la patiente. Malgré la reprise d’un antipsychotique atypique, il y a eu retour des symptômes psychotiques et des idées suicidaires. Compte tenu de l’état clinique précaire et après plus de 5 mois de normalisation des enzymes hépatiques, la décision fut prise de reprendre la clozapine, actuellement à 1 dose de 200 mg par jour depuis 6 mois sans récurrence d’anomalie hépatique, comme en témoignent les résultats obtenus par Eggert et coll (2).

**Bibliographie**


Caroline Bell, MD
Marc Delisle, MD, FRCPC
Quebec, Canada

**Re: A Case–Control Study on Psychological Symptoms in Sleep Apnea-Hypopnea Syndrome (SAHS)**

**Dear Editor:** I write to notify you of some rather strange results in the above-noted paper, published in the June 2003 issue of your journal (1).

Table 3 presents results from the Symptom Checklist-90. The General Severity Index (GSI) is, as noted by the authors, the mean of the responses on all 90 items, which are answered on a 0-to-4 Likert scale. On inspection, however, it appears that the tabulated GSI values in Table 3 are obviously too high, and as the mean GSI for the SAHS patients is stated as 5.25 (SD 4.94), there are definitely errors in the table.

**Reference**


Jan Ivanouw, PhD
Copenhagen, Denmark

**Reply: A Case–Control Study on Psychological Symptoms in Sleep Apnea-Hypopnea Syndrome (SAHS)**

**Dear Editor:** It is very kind of Dr Ivanouw to bring to our attention the fact that the data in Table 3 are too high. In fact, the data given for the General Severity Index of the Symptom Checklist-90 (SCL-90) represent the total factorial score, which is the sum of all 9 factorial scores. The total factorial score of the SCL-90 is commonly used to evaluate psychological status in the Chinese population. I apologize for confusing the 2 terms.

Thank you very much for your instruction.

Yue Weihua, MD
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