Letters to the Editor

Parkinsonism and Elevated Lactic Acid With Sertraline

Dear Editor: A few reports of sertraline-induced akathisia and dystonia are available, but reporting on sertraline-induced Parkinsonism is negligible (1). We report a case of Parkinsonism associated with sertraline use. To our knowledge, this is the first case in the literature to highlight a possible relation between selective serotonin reuptake inhibitors (SSRIs) and lactic acid metabolism.

Case Report

Mrs L, aged 45 years, is a married, middle-class housewife. She was premorbidly well adjusted and had no significant psychiatric or medical history and no significant family history. She presented with a 2-month history of persistent sadness, frequent crying spells, suicidal ideation, agitation, feelings of hopelessness and worthlessness, disturbed biological functioning, and weight loss of 5 kg. Her illness was precipitated by severe financial losses in her husband’s business. Moreover, he had taken a large loan to start his business, and his creditors were demanding payment. Their visits to the home caused her to become markedly restless and apparently unresponsive to her surroundings for periods of 30 minutes to 2 hours. She emerged from these spells when cold water was sprinkled on her face. Mrs L was hospitalized in a private nursing home and given tablet alprazolam 0.5 mg daily and intramuscular promethazine 50 mg, electrolyte, and lactic acid metabolism. Under physiological conditions, lactic dehydrogenase converts lactate to pyruvate, which is metabolized through the citric acid cycle. An elevated lactic acid level may imply a disturbance in mitochondrial oxidative metabolism. Mrs L’s elevated lactic acid correlated clinically with her Parkinsonism symptoms. The reversible elevated lactic acid level in this case is perhaps attributable to reversible disturbances in the mitochondrial oxidative metabolism. However, Mrs L’s muscle biopsy report was normal, which suggests neither mitochondrial encephalopathy nor disturbances in the oxidative metabolism (3). This case report shows that Parkinsonism can occur with sertraline therapy, although it may be a rare side effect.

References


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Delusion of Oral Parasitosis in a Patient with Major Depressive Disorder

Dear Editor: Delusion of parasitosis is a false belief in which sufferers have a strong conviction that they are infested with small, insect-like organisms. Patients believe that these organisms live and thrive in their skin and, sometimes, in other parts of their bodies. This condition is also named Ekbom’s syndrome (1). The delusion may exist as a core symptom in patients with delusional disorder, somatic type, or it may be one of the symptoms in other psychiatric disorders. We describe a patient with major depressive disorder who suffered from delusion of parasitosis of the oral cavity. To our knowledge, there is only one previous report of oral parasitosis.

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New Delhi, India
Case Report
The patient is a married housewife, aged 31 years, with a Grade 10 education. She lives in Kerman, a city in the south of Iran. The patient’s symptoms started 4 months prior to her referral. She was restless and suffered from deep depression. She said that she hated the world, that there was no future for her, and that everything was meaningless. She stated that she was only a moving corpse. Since the beginning of her illness, she had heard voices telling her that she would return to life on the day of resurrection and that, because she would be hungry, she would have to eat lizards, beetles, and crickets. After hearing these voices, she felt these organisms in her mouth. To remove them, she repeatedly chewed and swallowed, also feeling the taste of chewed materials. She thought that she was sinful and being punished by God. She had insomnia and anorexia and lacked energy to do her duties.

A mental state examination revealed that she had gustatory, tactile, and auditory hallucinations associated with somatic delusions, together with the conviction of being orally infested with tormenting organisms. Her cognition was intact, physical and neurologic examinations were normal, and brain magnetic resonance imaging showed no abnormality. The patient was treated with 150 mg imipramine and 8 mg pimozide daily. She became well 2 months later.

Discussion
Our patient suffered from the delusion that she was infested with several living organisms, including lizards. To our knowledge, all previous case reports describe patients who complained of being infested with only a single small organism; a large organism has not been previously reported. Delusion of parasitosis mainly targets the skin, although a report exists of 4 patients with ocular parasitosis associated with self-inflicted trauma (2). There is only one prior report of delusion of oral parasitosis; Maeda and others reported the case of an old man with a previous left-sided cerebral infarct who suffered from a delusional conviction that something like a thread was coming out from between his teeth. He could feel worms but not see them. This patient improved with pimozide treatment (3).

One interesting aspect of our patient is her auditory hallucination, which is not directly related to her somatic delusion. A second interesting aspect is that, to remove the disturbing organisms, she repeatedly chewed and swallowed them and then experienced a gustatory hallucination of chewed materials. This phenomenon has not been reported before and can only be observed where delusion of oral parasitosis exists.

Many patients with depression have irrational guilt feelings. Our patient had such guilt feelings; in her case, they were rooted in her religious beliefs, which could have had a special role in forming her delusion.

Our patient improved with combined drug therapy that included pimozide. Since its introduction, pimozide has been regarded as the specific treatment for delusion of parasitosis (4).

References

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Pathological Gambling and Cross-Addiction

Dear Editor: I read with enthusiasm the triad of articles dedicated to pathological gambling in the August 2004 issue of the CJP (1–3), but when finished, I felt somehow disappointed. The persistent avoidance of even mentioning 12-step–based models of addiction as alternatives to understanding pathological gambling in fact greatly limits the 3 papers.

The editorial by Dr Ladouceur attempts to explain problem gambling in terms of a pure cognitive model (1) wherein pathological gambling stems from the sufferer’s inability to understand the independent randomness of chance events, confirmed by the fact that most individuals will hold nonscientific and false beliefs if exposed to gambling. The typical example offered is the situation in which, after the tossing of a coin has resulted in several consecutive tails, most people will believe that chances for heads have increased with the next toss. However, this phenomenon does not explain pathological gambling; it just explains how most people think. It is almost like saying that people gamble because they have 2 feet! As Dr Ladouceur admits, two-thirds of adults gamble, and most people find it difficult to understand randomness, preferring to interpret reality within a deterministic framework. Dr Ladouceur suggests that these false beliefs are more strongly held among problem gamblers than among the general population, and for that reason, they cannot stop gambling, even in the face of loss and self-destruction. This line of reasoning equates to using our knowledge of why the general population drinks moderately to explain why people become dependent on alcohol; it is obviously fallacious. If Dr Ladouceur considered instead a cross-addiction model of pathological gambling, he would not find it difficult to observe the similarities between problem gamblers’ insistence on gaming, despite financial ruin, and the persistent addictive behaviour of patients dependent on alcohol or opiates, despite the tragic consequences.

Although Dr Shaffer and others highlight the high levels of comorbidity between chemical addictions and pathological gambling (2), they never consider that this comorbidity could be the manifestation of a common pathology. If the authors had included in the comorbidity with problem gambling not only chemical addictions but also such other addictive behaviours as binge eating and sex addiction, they would have found a concordance close to 100%. Looking at addiction, including pathological gambling, as a unitary problem, would also have helped the authors to understand the life trajectories described in their article. The fact that pathological gamblers are not constantly involved in gambling is not surprising, according to a cross-addiction model. All clinicians involved in addiction treatment observe their patients switching among different addictive behaviours during their lifetime. The addiction is lifelong; the way to express it changes. I am confident that, if the authors were to observe patients who seem to recover from their gambling problem longitudinally, they would realize that, in reality, most have just transferred their addiction to other addictive behaviours such as alcohol abuse, substance
Letters to the Editor

Marco Procopio, MD, MRCPsych
Hove, East Sussex

Reply: Pathological Gambling and Cross-Addiction

Dear Editor: Dr Procopio read the editorial and 2 articles (1–3) published in the August 2004 issue of the CJP on pathological gambling with enthusiasm; however, he ultimately felt somewhat disappointed. He believes that some important issues were left out and that some comments may not adequately reflect our understanding of this disorder. In the following comments, we address the main issues raised by Dr Procopio.

Dr Procopio criticizes the editorial as follows:

The editorial by Dr Ladouceur attempts to explain problem gambling in terms of a pure cognitive model (1), according to which pathological gambling stems from the sufferer’s inability to understand the independent randomness of chance events, confirmed by the fact that most individuals will hold nonscientific, false beliefs if exposed to gambling.

Ladouceur’s editorial goal was not to present an exhaustive critical review of the literature on the causes of pathological gambling or to explain its etiology. The editorial described a central attribute associated with the experience of pathological gambling—the hope to win money. Contrary to Dr Procopio’s statement, the cognitive theory was not focused on the etiology of pathological gambling. Rather, “the central consequence, and possibly the core factor in causing gambling problems, are the financial losses” (1, p 501).

A careful reading of Dr Shaffer and others’ article reveals that they do support a syndromal approach to addiction—exactly the view espoused by Dr Procopio. Shaffer and others refer to a syndrome approach in the paper, but perhaps not sufficiently, because the paper’s primary purpose was to focus on epidemiology. For example, Shaffer and others note that although the DSM-IV currently classifies pathological gambling as an impulse control disorder, many clinicians consider it to be an addiction, a label not yet included in the APA nomenclature. Indeed, the DSM criteria for gambling disorders closely parallel the signs and symptoms of substance use disorders, which are commonly considered addictions (2, p 505–6).

Similarly, Many aspects of problem behaviours emerge during adolescence. Compared with adults or those younger, adolescents are more likely to take drugs, act delinquently, and commit serious crimes... Jessor and Jessor have posited a ‘problem behaviour syndrome’ to explain this multifaceted increase. Some researchers... suspect that gambling may be another facet of this syndrome, implying that the prevalence of gambling-related problems in adolescents may be similarly inflated. Indeed, prevalence studies have consistently shown that adolescents evidence higher rates of problem and pathological gambling than adults (2, p 509).

We encourage readers interested in a syndrome approach to gambling and other addictions to read Shaffer and others’ publication, in which they review addiction as a syndrome (4).

Concerning the treatment paper (3), self-help groups were not included because Dr Toneatto only examined controlled studies, and he could find none that evaluated 12-step approaches. This paper’s objective was not to review treatments per se, but rather, empirically supported treatments.

Regarding the comment about substitute addictions, Dr Procopio is making assumptions. In our experience, many individuals recover from an addiction and do not transfer their addictive tendency to another behaviour. Certainly, this does happen, but of course, it may not be true for everyone.

The comments about cognitive distortions are not clear. The point seems to be that the key to understanding gamblers is to look also at other addictive behaviours. Again, this statement is not supported by empirical data.

References


Howard Shaffer, PhD
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Tony Toneatto, PhD
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Robert Ladouceur, PhD
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The Psychiatric Emergency Service Patient

Dear Editor: For most patients, the psychiatric emergency service (PES) is the major point of entry into the mental health system (1). Several functionally and structurally dissimilar PES models are in use today (2–4); consistent epidemiologic data supporting any particular model are lacking. Presently, data obtained from one PES cannot be easily compared with data obtained from another. Differing observation periods, patient volumes, and data-acquisition strategies, as well as the lack of a standardized PES classification scheme, have likely all contributed to the inability to obtain the stable and reproducible patient profiles crucial for assessing PES efficacy.

Using a rigorous PC-based data acquisition strategy, we aimed to more definitively assess the clinical and demographic characteristics of a local PES population and the presence of clinically significant subpopulations. Over a period of 4½ years, we acquired up to 70

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abuse, smoking, overeating, and pathological sexual promiscuity.

As a last remark, it is unacceptable to review treatments available for pathological gambling (3) without mentioning 12-step fellowships and treatment centres that follow this philosophy, given that most patients who try to fight addiction on both sides of the Atlantic are helped by this model.

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Using a rigorous PC-based data acquisition strategy, we aimed to more definitively assess the clinical and demographic characteristics of a local PES population and the presence of clinically significant subpopulations. Over a period of 4½ years, we acquired up to 70
variables prospectively from patients visiting the PES of a large metropolitan general hospital (5). We subsequently added a preexisting 10½-year, prospectively acquired patient log of 8 variables to the database. We compared these results with those derived from a 72-journal review of the PES literature (from 1965 to 2003).

Overall, 14 826 patients made 29 577 PES visits throughout the 15½-year observation period. There was only a moderate degree of agreement between the present data and data obtained from the literature review. Demographic and socioeconomic variables were partially consistent with those of previous reports, whereas more complex clinical data were poorly correlated. We could not derive a single, all-inclusive typical local PES user profile from our data. Rather, we observed several different profiles, varying according to the age and sex of the patients. We also identified clinically significant sub-populations, for example, frequent users and patients with a primary diagnosis of substance abuse, and quantified their overall impact on PES functioning.

The present report benefited from the longest observation period of any published PES study. Gold standards for several variables, such as sex and age, were obtained and, by corollary, more definitive and precise local PES user profiles than were previously available. Using the same methodology, we are currently undertaking a 2-year multicentre study collecting data in 4 structurally and functionally dissimilar PES’s to determine whether the above local data can be generalized, as well as the effects of different PES models on PES efficacy. Useful information will be obtained regarding the type of model possessing a greater potential for future development.

References


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