Milnacipran for the drastic improvement of refractory pain in a patient without depressive symptoms: a case report

Dear Editor,

Milnacipran is a selective and potent dual serotonin and noradrenaline reuptake inhibitor. It has been reported that milnacipran is beneficial for the treatment of chronic pain [2] and fibromyalgia [5]. However, to our knowledge, drastic effectiveness of milnacipran for the treatment of refractory pain has not yet been reported. We present a patient with refractory pain who responded drastically to single-agent treatment with milnacipran despite a lack of depressive symptoms.

Mr. A, a 59-year-old man who had been suffering from a severe pain such as thrusting a wimble into his left lateral femoral region for 2 months, came to our outpatient clinic. Through X-ray of the femur, magnetic resonance imaging of the brain and the spinal cord, and a blood examination, however, his physical and neurological examinations were normal, and he had no abnormal findings causing his severe pain. His daily life was impaired extremely because nonsteroidal anti-inflammatory drugs and anticonvulsants were ineffective in his severe pain.

Mr. A had not received any prior psychiatric treatment and no past history of alcohol and substance abuse. After psychiatric interview with him, depression and dementia were excluded, and he was diagnosed with pain disorder according to DSM-IV criteria. We evaluated his severe pain in left lateral femoral region with visual analogue scale (0–100) [1]. His score on the VAS was 94. We prescribed 50 mg/day of milnacipran. After 2 weeks of his taking milnacipran, his severe pain was not alleviated and his VAS score was 90. After 4 weeks, he improved dramatically and his VAS score decreased from 90 to 38. We increased a dose of milnacipran to 100 mg/day. After 7 weeks of his taking milnacipran, his pain resulted in complete resolution, and his score on the VAS was 0. No adverse effects of milnacipran were observed.

Tricyclic antidepressants that have serotonin and noradrenaline reuptake inhibitor properties are used for the treatment and management of pain [3]. The mechanism of action is thought to modulate descending pain inhibitory pathways due to the inhibition of reuptake of serotonin and noradrenaline released predominantly in the raphe nucleus and the locus coeruleus of the brainstem [4]. Therefore milnacipran, a selective serotonin and noradrenaline reuptake inhibitor, have utility in the treatment of pain. Further studies are necessary to establish the efficacy of selective serotonin and noradrenaline reuptake inhibitors in the treatment and management of pain.

References

[1] Carlsson AM. Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. Pain 1983;16:87–101.
[2] Kamata M, Takahashi H, Naito S, Higuchi H. Effectiveness of milnacipran for the treatment of chronic pain: a case series. Clin Neuropharmacol 2004;27:208–10.
[3] Lynch ME. Antidepressants as analgesics: a review of randomized controlled trials. J Psychiatry Neurosci 2001;26:30–6.
[4] Stamford JA. Descending control pain. Br J Anaesth 1995;75:217–27.
[5] Vitton O, Gendreau M, Gendreau J, Kranzler J, Rao SG. A double-blind placebo-controlled trial of milnacipran in the treatment of fibromyalgia. Hum Psychopharmacol 2004;19(Suppl 1):S27–35.

Shinsuke Kito *
Toru Nakajima
Yoshihiko Koga
Department of Neuropsychiatry, Kyorin University School of Medicine, 6-20-2 Shinkawa, Mitaka, Tokyo 181-8611, Japan
E-mail address: kito@kk.iij4u.or.jp (S. Kito).

Received 16 March 2005; accepted 10 April 2005

* Corresponding author.

doi:10.1016/j.eurpsy.2005.04.005

Sertraline for Klüver–Bucy syndrome in an adolescent

Dear Editor,

Klüver–Bucy syndrome (KBS), a neurobehavioral syndrome first described in rhesus monkeys, is characterized by visual agnosia, placidity, hypermetamorphosis (strong tendency to react to visual stimulus), increased oral exploration, altered dietary habits, and hypersexuality [3]. In addition, human cases have features such as fluent aphasia, amnesia, dementia and seizures [3]. Although this disorder is generally considered to have an unfavorable outcome, we wish to report a case of KBS who didn't respond to a combination of an antipsychotic and carbamazepine but responded when sertraline was added.

Ms. A, a 14-year-old adolescent presented with history of hypersexuality, hyperphagia, oral tendency, hyperactivity, misidentification and poor personal care of 4 years duration. Pre-morbidly she was an intelligent and social girl with a good academic record, but 4 years ago she developed an illness characterized by severe headache, high-grade fever, and was in a comatose state for 15 days. Although her parents were not sure about the exact nature of this illness, the picture was consistent with encephalitis. While recovering from her ill-

ness, the patient started exhibiting disinhibited behaviors such as disrobing in front of others, manipulating her genitals, and making sexual advances toward her father. She would lick any object lying on the ground and whenever she got an opportunity, she would rush to the toilet and try to put urine and feces in to her mouth (urophagia and coprophagia, respectively). She also exhibited hyperphagia and would eat three times the usual amount of meal at a time. She was impulsive and would try to snatch any object and slap others without reasons. She would misidentify parents and relatives and would call them by different names. A meaningful mental state examination was not possible in view of her irrelevant speech and impulsive and disorganized behavior. Her neurologic examination did not show any long tract signs. Her blood work up was unremarkable and so was an EEG. However, a head CT scan showed diffuse mild cerebral atrophy with no focal abnormalities.

The patient was started on carbamazepine titrated up to 600 mg per day without any improvement in symptoms. Three weeks later, risperidone 4 mg per day was added, but only mild improvement was noticed. Six weeks after this combination, sertraline 25 mg was added which was increased to 100 mg over a period of 2 weeks. Surprisingly, 6 weeks after commencing sertraline, all her symptoms improved remarkably, including hypersexuality, disinhibited behavior, hyperorality, urophagia and coprophagia. On follow-up 2 months later, she had stopped taking sertraline for 2 weeks (while continuing other drugs) and had worsening of the above-mentioned symptoms; reinstituting sertraline again ameliorated the symptoms. The patient was doing well for next 6 months till her last follow-up.

Our case exhibited most of the symptoms of KBS. Although KBS has been shown to be associated with demonstrable bilateral lesions in the anterior temporal horn or amygdale [4], other authors have reported that lesions in the amygdala are not necessary for KBS in animals or humans [1]. This is consistent with our patient who showed diffuse brain atrophy but no localized lesions. Carbamazepine and antipsychotics have been previously reported to be effective in managing some of the behavioral symptoms of KBS [2]. There is only one report describing the successful use of selective serotonin reuptake inhibitors (SSRIs) in two patients with KBS secondary to head trauma [5]. In one of these cases, the patient didn't respond to carbamazepine alone but responded only after being started on fluoxetine. In the second case, the patient responded to a combination of antipsychotics and sertraline. That sertraline may have an independent effect on symptoms of KBS is further bolstered by relapse of symptoms on withdrawing this drug and their resolution again on rechallenge in our patient. Some of the symptoms of KBS are compulsive in quality and may reflect a disturbance of aggression regulation and impulse control. It may be worth exploring if the known efficacy of SSRIs against these symptoms cluster may play a role in treating KBS.

References

[1] Carroll BT, Goforth HW, Carroll LA. Anatomic basis of Klüver–Bucy syndrome. J Neuropsychiatry Clin Neurosci 1999;11:116.
[2] Hooshmand H, Sepdham T, Vries J. The Klüver–Bucy syndrome. Successful treatment with carbamazepine. J Am Med Assoc 1974; 229:1782.
[3] Jha S, Patel R. Kluver–Bucy syndrome—an experience with six cases. Neurol India 2004;52:369–71.
[4] Lily R, Cummings JL, Benson DF, Frankel M. The human Klüver–Bucy syndrome. Neurol 1983;33:1141–5.
[5] Slaughter J, Bobo W, Childers MK. Selective serotonin reuptake inhibitor treatment of post-traumatic Klüver–Bucy syndrome. Brain Inj 1999;13:59–62.

D.N. Mendhekar
Delhi, India

H.S. Duggal *
Western Psychiatric Institute and Clinic, 3811 O'Hara Street, 15213 Pittsburgh, PA, USA
E-mail address: bobduggal@yahoo.com (H.S. Duggal).

Received 23 March 2005; accepted 19 April 2005

* Corresponding author.

0924-9338/$ - see front matter
doi:10.1016/j.eurpsy.2005.04.007

Brief psychotic disorder associated with Sturge–Weber syndrome

Keywords: Sturge–Weber syndrome; Phakomatoses; Brief psychotic disorder

Sturge–Weber syndrome (SWS) is a rare, sporadic neurocutaneous syndrome with an estimated frequency of one per 50,000 live births [5]. Although mental retardation is a well-studied sequelae of SWS [5], little attention has drawn on the psychological well being of SWS patients. SWS is devastating, especially when children experience recurrent seizures, pervasive learning and behavioral problems, and disabling visual impairment [5], all predisposing to problems in behavioral functioning [1].

Despite the above, literature on psychotic symptoms in these patients is scarce.

We report on the case of a 33-year-old male with an extended nevus covering his whole face except for a small area surrounding his right eye and vascular deformities extending to the left temporo-parietal lobes. He was suffering from glaucoma and was on carbamazepine for complicated seizures. The patient had no personal or family history of major psychiatric disorder and had a normal psychosocial development. He was working as a freelancing software engineer but