Mirtazapine Relieves Limb Paralysis of Unknown Etiology: A Three-Year Case Follow-Up

To the Editor: "Mrs. M" is a housewife who has had bilateral limb paralysis (LP) of her legs for the last 3.3 years; she had no significant brain or lumbar spine findings in MRI of brain or lumbar-spine, biochemistry examination, and substance or toxicity screenings by many departments of internal and surgical medicine. She was transferred to our clinic because there were no pathologic results of medical examinations. No significant psychosocial stressors, depression (Hamilton Rating Scale for Depression [Ham-D]), or anxiety symptoms (Hamilton Rating Scale for Anxiety (Ham-A) were noted, except bilateral LP of legs, with decreased muscle power (MP: score 2), mild anorexia, low back pain (visual analog scale (VAS) score: 5), and insomnia. No other neurologic signs were found except bilateral LP of legs. Mirtazapine (30 mg/day) was used for the above complaints with unknown etiology, and Mrs. M had relief of LP with less pain feelings (VAS: 3), increased mp, better sleep, and appetite within 2 weeks. LP improved progressively in the following 1 month, with MP returning to normal (score: 5), pain relief (VAS: 1), and ability to walk without assistance. She continued mirtazapine use during the last 3 years; however, she complained that LP and pain would relapse with cessation of mirtazapine (MP: 3 and VAS: 4 if she discontinued mirtazapine use for longer than 1 month). She was still under continuous therapy with mirtazapine (30 mg/day) with good control of LP and pain up to the present (MP: 5; VAS: 1). There has been no significant psychopathology of depression (Ham-D: 0–1) or anxiety (Ham-A: 0–2) during these 3 years.

Discussion

Mirtazapine could enhance firing rates of dopaminergic neurons and adrenergic neurons in frontocortical and corticolimbic areas, such as frontal cortex, striatum, nucleus accumbens, and hippocampus.[1] Dopaminergic-enhancing agents, such as *L*-dopa, has been reported to facilitate motor recovery in hemiplegic stroke patients.[2] The precursor of norepinephrine or aromatic amino acid decarboxylase inhibitor could elevate norepinephrine level in cerebral cortex and hippocampus of sensorimotor cortex-ablated rats. The increase in norepinephrine level was correlated with motor-function recovery.[3] Serotonin could give a significant number of stroke patients better outcomes of motor function after fluoxetine treatment, above and beyond the depression-treating effects.[4]

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Chien-Han Lai, M.D., M.Sc.
Dept. of Psychiatry
Buddhist Tzu-Chi General Hospital
Taipei Branch
Taipei, Taiwan
Institute of Brain Science
National Yang Ming University
Taipei City, Taiwan
e-mail: t122336257@yahoo.com.tw.

References

1. Millan MJ, Gobert A, Rivet JM, et al: Mirtazapine enhances frontocortical dopaminergic and corticolimbic adrenergic, but not serotonergic, transmission by blockade of alpha$_2$-adrenergic and serotonin$_{2C}$ receptors: a comparison with citalopram. Eur J Neurosci 2000; 12:1079–1095
2. Scheidtmann K, Fries W, Muller F, et al: Effect of levodopa in combination with physiotherapy on functional motor recovery after stroke: a prospective, randomised, double-blind study. Lancet 2001; 358:787–790
3. Kikuchi K, Nishino K, Ohyu H: Increasing CNS norepinephrine levels by the precursor L-dopa facilitates beam-walking recovery after sensorimotor cortex ablation in rats. Brain Res 2000; 860:130–135
4. Dam M, Tonin P, De Boni A, et al: Effects of fluoxetine and maprotiline on functional recovery in poststroke hemiplegic patients undergoing rehabilitation therapy. Stroke 1996; 27:1211–1214