Psychiatria Danubina, 2009; Vol. 21, No. 4, pp 518 *Letter to editor*

RARE PSYCHOTROPIC ADVERSE EVENT OF MOXONIDINE

Krunoslav Fuckar, Nenad Lakušić & Duško Cerovec

Division of Cardiology, Special hospital for medical rehabilitation Krapinske Toplice, Croatia

Moxonidine is a centrally acting antihypertensive agent with high affinity to the imidazolin-I1 receptor which induces peripheral sympathetic inhibition reflected by lowering of blood pressure and heart rate (Ernsberger et al. 1993). To a lesser extent it also binds to the α2-receptors whose activation is responsible for side-effects of central antihypertensive drugs such as sedation and dry mouth (Van Zwieten 1999). Because it decreases the total sympathetic tone without reducing exercise capacity moxonidine appears suitable for treatment of patients with high sympathetic activity and physical or mental stress induced hypertension (Wenzel et al. 2004). It is well tolerated and has a low potential for drug interactions. However there are reports which show that when co-administered with a low dose of lorazepam, moxonidine increases the impairment of attentional tasks (Wesnes et al. 1997). Because of excess early mortality and morbidity moxonidine is contraindicated in patients with chronic heart failure (Cohn et al. 2003).

During the course of our clinical practice we recently treated a peri-menopausal female patient with signs of metabolic syndrome and arterial hypertension resistant to various antihypertensive drugs. Due to the beginning of the menopause she occasionally suffered from restlessness and mild anxiety episodes. Among other antihypertensive agents we started her on moxonidine in the usual dosage profile. Four days after the beginning of moxonidine therapy the patient developed horrible nightmares. Immediately after withdrawal the nightmares stopped and no further sleep disorders were registered. Although this adverse event was not listed in the product characteristics summary and was unexpected as such, the positive de-challenge along with other known central effects of moxonidine make the connection between this adverse event and the drug itself highly probable. The exact mechanism has not yet been thoroughly clarified but it most likely pertains to type A adverse events which are associated with the drugs pharmacological effect.

In the World Health Organization (WHO) adverse events database there are so far eight cases of moxonidine induced nightmares reported since the year 1999.

According to the criteria of the Council for International Organizations of Medical Sciences (CIOMS) the above mentioned adverse event was listed as non serious. Nevertheless if not recognized as a side effect of moxonidine, severe repeating nightmares may cause unnecessary psychiatric evaluation with possible prescription of various psychotropic drugs as well as significantly decreased quality of life. An interdisciplinary medical approach is crucial in order to avoid unnecessary treatment in such cases.

In conclusion we believe one must bear in mind that psychotropic symptoms occurring when treating hypertensive patients with moxonidine, especially menopausal women, could in some cases be the result of the side effects of the drug itself.

REFERENCES

1. *Cohn JN, Pfeffer MA, Rouleau J, Sharpe N, Swedberg K, Straub M, Wiltse C & Wright TJ: MOXCON Investigators. Adverse mortality effect of central sympathetic inhibition with sustained-release moxonidine in patients with heart failure (MOXCON). European Journal of Heart Failure 2003; 5: 659-67.*
2. *Ernsberger P, Damon TH, Graff LM, Schafer SG & Christen MO: Moxonidine, a centrally acting antihypertensive agent, is a selective ligand for I1-imidazoline sites. Journal of Pharmacology and Experimental Therapeutics 1993; 264:172-182.*
3. *Van Zwieten PA: The renaissance of centrally acting antihypertensive drugs. Journal of Hypertension 1999; 17: 15-21.*
4. *Wenzel RR, Mitchell A, Siffert W, Bührmann S, Philipp T & Schäfers RF: The I1-imidazoline agonist moxonidine decreases sympathetic tone under physical and mental stress. British Journal of Clinical Pharmacology 2004; 57: 545-51.*
5. *Wesnes K, Simpson PM, Jansson B, Grahnén A, Weimann HJ & Küppers H: European Journal of Clinical Pharmacology 1997; 52: 351-8.*

Correspondence:

Krunoslav Fuckar, MD
Division of Cardiology, Special hospital for medical rehabilitation
Gajeva 2, 49217 Krapinske Toplice, Croatia
E-mail: krunoslav.fuckar@kr.t-com.hr