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Increased female sexual response after oxytocin

Dr M ANDERSON-HUNT and Associate Professor L DENNERSTEIN (Key Centre for Women's Health in Society, University of Melbourne, Carlton, Victoria 3053, Australia) write: A 26 year old woman presented 17 months after the birth of her second child for some temporary help with the breast feeding of her nephew. The breast feeding of her child had been intermittent after 6-8 months and ceased at 15 months. Lactogenesis had continued with minimal milk expressed. A 28-32 day menstrual cycle had returned by 12 months post partum. A progestogen only pill (levonorgestrel 30 μg) had been used since 15 months.

A synthetic oxytocin spray (Syntocinon; Sandoz) was used on day 17 of her irregular cycle. Let down of milk occurred but left the infant unsatisfied. No further attempts were made to breast feed. About 2 hours after the use of two activations (8 IU) of oxytocin, she noticed copious vaginal transudate trickling down her leg. Intense sexual desire had followed, and she reported that her cervix had opened slightly. She had initiated sex with her partner and commented that the uterine and vaginal contractions were intensified at orgasm, along with heightened subjective pleasure.

Oxytocin was repeated on day 19 with similar effects lasting again for three hours after their onset. Seven to 10 days later she ceased taking levonorgestrel and restarted barrier contraception. Two weeks later she readministered the spray but no sexual responses occurred.

In a detailed sexual history she revealed that she had been sexually active during her second pregnancy with orgasm about three times a week. Most sexual activity ceased for the months after caesarean section and then resumed as before. She also described feelings of mild arousal with breast feeding her children, an experience common among other women.

To our knowledge, no similar cases have been reported,[1] although reviews of animal studies indicate that interactions between sex steroids and oxytocin have been documented in mammals.[2-4] Contextual issues surrounding other prosexual substances and oxytocin as potential aphrodisiacs for women have been discussed.[5]

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5 Anderson-Hunt M, Dennerstein L. Hormones and sexual arousal: developing a method for research. In: International Women's Health Coalition—Population Council Sexuality and Gender Working Group. *Learning about sexuality*. New York: IWHC—Population Council (in press).

Hearing loss and tinnitus with carbimazole

Dr D HILL, Mr H WHITTET, and Dr H SIMPSON (Royal Berkshire Hospital, Reading, Berkshire RG1 5AN) write: We present a previously undocumented complication of carbimazole treatment occurring in a 28 year old woman with Graves' disease. Four months after starting carbimazole (40 mg, dropping to 20 mg daily after six weeks) she developed left sided otalgia and presented with high pitched tinnitus. An audiogram showed a unilateral 25 dB high frequency loss which could not be accounted for by family or occupational history. Brain stem audiometry showed no evidence of an acoustic neuroma; the only abnormal result was a raised titre of antibodies to DNA (106 IU, normal range 0-50). Carbimazole hypersensitivity was diagnosed, and she started taking propylthiouracil instead (100 mg twice daily). Four months later the hearing loss had subjectively improved and an audiogram showed that her hearing was within normal limits, although the tinnitus persisted. The DNA antibody titre had dropped substantially to 54 IU, and she had not developed any further symptoms of hypersensitivity.

Acute ototoxicity has been reported in only two patients receiving thiourea derived antithyroid drugs. One patient developed polyarthritis, fever, and bilateral deafness four days after starting propylthiouracil, with incomplete recovery of hearing on drug withdrawal.[1] The other patient developed unilateral deafness, tinnitus, and polyarthritis 10 months after starting propylthiouracil, the tinnitus persisting after complete recovery of hearing.[2] Both these patients were acutely unwell and had serological evidence of systemic lupus erythematosus. An association between carbimazole and propylthiouracil and the development of a lupus-like syndrome have been previously noted, with evidence suggesting a causal role being particularly strong for propylthiouracil.[3] Cross sensitivity to these two drugs has been reported, but it remains unclear why thiourea derived drugs should have this association.[4]

Autoimmune inner ear disease is well recognised.[5] Patients are typically young, have a bilateral, occasionally unilateral, hearing loss, and often have frank signs of autoimmune disease. The relevant antigens are unknown. In our patient deafness and tinnitus developed in association with serological evidence of lupus, both of which improved on drug withdrawal. We suggest that this could represent an autoimmune phenomenon due to the development of antibodies to connective tissue or neural antigens in the cochlea. It is interesting that the symptoms we ascribe to carbimazole hypersensitivity developed after a prolonged period of treatment and that these did not worsen with propylthiouracil.

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2 Smith KE, Spaulding JS. Ototoxic reaction to propylthiouracil. *Arch Otolaryngol Head Neck Surg* 1972;96:368-70.
3 Horton RC, Shepperd MC, Emery P. Propylthiouracil-induced systemic lupus erythematosus. *Lancet* 1989;ii:568.
4 Smith A, Gledhill RF, Jenkins P. Cross sensitivity to thyroid drugs. *BMJ* 1989;298: 1253.
5 Hughes GB, Barna BP, Kinney SE. Clinical diagnosis of autoimmune inner ear disease. *Laryngoscope* 1988;98:251-3.

Peripheral neuropathy with bezafibrate

Drs C J ELLIS, W E WALLIS, and M CARUANA (Middlemore Hospital, Auckland 1006, New Zealand) write: Neurological side effects with bezafibrate are uncommon. Fatigue, weakness, drowsiness, dizziness, and headache have previously been recorded.[1,2] Peripheral motor neuropathy has been seen with clofibrate,[3] and gemfibrozil has been implicated in six patients with paraesthesia.[4] To our knowledge this is the first report of peripheral neuropathy due to bezafibrate and substantiated by nerve conduction studies.

A previously well 52 year old European man started taking bezafibrate 200 mg three times daily two months after a myocardial infarction. His serum cholesterol concentration was 6·9 mmol/l (normal range 3·6-5·2 mmol/l). He had also been taking aspirin 150 mg daily since the infarction. After one month his fingers and toes developed a persistent painful tingling without weakness, which progressed over six months. A neurological history confirmed good health with mild alcohol intake and no known toxic exposure. General examination showed nothing abnormal, while neurological examination showed loss of all sensation in a glove and stocking distribution (to mid-calves and wrists bilaterally) and depressed ankle reflexes.

The following investigations and tests gave normal results: chest radiography: assays of serum urea, electrolytes, glucose, vitamin B-12, and folate concentrations; iron studies; liver and thyroid function tests; syphilis serology; assays of nuclear antibodies and rheumatoid factor; Coombs' test; protein electrophoresis; and full blood count. The erythrocyte sedimentation rate was 5 mm in the first hour. Results from nerve conduction studies in the arms were within normal limits, while they showed a mild reduction in sensory action potential amplitudes in the legs. The right sural nerve stimulated 14 cm from the ankle produced an amplitude of 3 μV (normal laboratory range 15 (5) μV), although conduction velocities were normal. Results from motor studies, including F wave and reflex latencies, and from needle electromyography of distal leg muscles were normal.

The neurophysiological evidence suggested a mild peripheral sensory neuropathy of an axonal type, the apparent discrepancy between the results of nerve conduction studies of the arms and legs often being found early in a peripheral neuropathy. On stopping bezafibrate treatment the symptoms completely resolved, although nerve conduction studies three months later showed similar results: a mildly low amplitude in the sural nerve without any abnormality in the latency (implying axonal damage, not demyelination). The patient declined any further studies.

Monitoring bodies have generally received less well documented reports: New Zealand monitoring programme (three reports), British Committee on Safety of Medicines (five), Boehringer Mannheim drug safety department (17); Data from the World Health Organisation list four cases of neuropathy and 13 of paraesthesiae. The cause of peripheral neuropathy due to bezafibrate is unknown.

We thank the New Zealand Intensive Medicines Monitoring Programme, the British Committee on Safety of Medicines, and Boehringer Mannheim New Zealand for information. Interpretation of the figures from the Committee on Safety of Medicines is our own.

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