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Case Report

Hyperprolactinaemia induced by proton pump inhibitor

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Abstract

A case of a 13 year old girl who manifested hyperprolactinaemia and galactorrhea induced by Omeprazole, a commonly used proton pump inhibitor is presented.

Introduction

Hyperprolactinaemia is a condition of elevated serum prolactin levels, manifesting as galactorrhoea and menstrual irregularities in women and erectile dysfunction and decreased libido in men. Hyperprolactinaemia has a long list of causes including physiological, pathological and pharmacological.^{1,2} Drug induced hyperprolactinaemia is common and a number of drugs have been identified as the cause. e.g. antipsychotics, antidepressants, H₂ receptor antagonist, antiandrogens, estrogen, prokinetics, anticonvulsants, opiates and cholinomimetics. These drugs raise the serum prolactin to a level that is frequently associated with symptoms.¹⁻⁵

We describe here a case of hyperprolactinaemia presenting with galactorrhea induced by Omeprazole (losec) a proton pump inhibitor.

This to our knowledge has not been reported before.

Case Report

A 13 year old girl presented to her physician with a history of migranous headache and was prescribed mefenamic acid. Other than these bouts of headache for last 3 months, she had no active medical issues. On taking mefenamic acid she developed dyspeptic symptoms and was found helicobacter antibody positive and was prescribed Omeprazole. After 4 days of treatment with Omeprazole 20mg BD, she developed bilateral galactorrhea. Her prolactin level was found to be 288ng/ml (normal being 2.7-22.4ng/ml). On detailed interrogation she refused to have taken any other drugs in particular antiemetics or H₂ receptor antagonists. An MRI of hypothalamo-pituitary area revealed a bulky pituitary probably due to pubertal change. She was given a course of anti helicobacter regimen and Omeprazole was discontinued. The galactorrhea resolved and three weeks later her serum prolactin levels returned to 17.5ng/ml (normal).

Considering the temporal relationship with omeprazole, with the patient and parent's consent, she was rechallenged with omeprazole after six weeks and her serum prolactin level was checked again. The serum prolactin level was increased from a base line of 26.6 to 70ng/ml and returned

back to normal in 2 weeks after withdrawal.

As part of laboratory work up, serum calcium, phosphorus, gastrin was reported normal.

Later, she reported with a bout of headache to some other centre and was given injection Domperidone and tablets for nausea as take home medication. Forty eight hours later she again developed galactorrhea and her domperidone was discontinued. This time her prolactin level was 161ng/ml. She remained normal with episodes of headache and nausea, which used to settle with acetaminophen and NSAID, alongwith antacids, sucralfate and ranitidine which was tolerated well.

After about six months she again developed a severe bout of headache with nausea, vomiting and dizziness and attended a nearby medical centre where she was given injection diclofenac. On insistence by her parents about the past medical history of galactorrhea induced by omeprazole (losec), this time she was prescribed lansoprazole instead of omeprazole. Three days later she again developed galactorrhea and her serum prolactin was 49.7ng/ml which returned to normal in a week after stopping lansoprazole.

These three documented instances of hyperprolactinemia by proton pump inhibitors is to our knowledge, has not been reported before.

Discussion

Hyperprolactinemia, a very common biochemical abnormality, is frequently associated with the use of certain medications. There is a long list of medications that are associated with a rise in serum prolactin and symptoms of hyperprolactinemia.¹⁻⁵

Hyperprolactinemia induced by medication should return to normal after the withdrawal of the offending drug. In this case the administration of proton pump inhibitor provoked a rise in serum prolactin level, followed by a return towards the baseline after its discontinuation. Although proton pump inhibitors are generally well tolerated and they are the most commonly prescribed classes of medication in the treatment of acid peptic disease,⁶ an important adverse effect is a rise in serum prolactin level.

To our knowledge this is the first case report incriminating the proton pump inhibitors as a cause of symptomatic hyperprolactinemia. Hyperprolactinemia

induced by medication does not need to be managed with dopamine agonists like cabergoline or bromocriptine, as in the case of hyperprolactinemia caused by some pathological cause.¹

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