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Case Report

Escitalopram-induced epistaxis: A case report

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الملخص

مثبطات استرداد السيروتونين الانتقائية هي مجموعة من الأدوية التي تم تطويرها لعلاج الأمراض النفسية المختلفة، مثل اضطراب الاكتئاب، القلق والوسواس القهري. بالرغم من كونها آمنة بشكل عام إلا أنه يمكن أن يكون لها عدة أعراض جانبية بما في ذلك زيادة احتمالية النزف. تؤثر مثبطات استرداد السيروتونين الانتقائية على تنشيط الصفائح الدموية وتجمعها، مما قد يؤدي إلى زيادة خطورة حدوث نزيف عند استخدامها في حالات معينة. ومن الممكن حصول النزيف في مواقع وأعمار مختلفة وعند استخدامها مع أدوية أخرى. غالبا ما تشمل عوامل الخطر المذكورة بشكل شائع في البحوث السابقة استخدامها مع أدوية أخرى، تليف الكبد أو الفشل الكبدي. في هذه الورقة، تقرير لعامل آخر محتمل وأقل شيوعا للنزيف عند استخدام مثبطات استرداد السيروتونين الانتقائية. هذه الورقه تشمل تقرير حالة لذكر، بالغ، شرق أوسطي وبدون أي تاريخ مرضي سابق عدا عن الحساسية الموسمية، أصيب بالرعاف مع استخدام دواء مثبط للسيروتونين بعلاج اضطراب اكتئابي. نظرا لأن وجود تاريخ مرضي للحساسية الموسمية من الممكن أن يشكل عامل خطورة للنزيف مع استخدام مثبطات السيروتونين، فيجب استكشاف عوامل الخطر للنزيف بخلاف الأسباب الشائعة لتحسين العلاج والوقاية.

الكلمات المفتاحية: رعاف؛ إسيتالوبرام؛ التهاب الأنف التحسسي؛ نزيف؛ مثبطات امتصاص السيروتونين الانتقائية

Abstract

Selective serotonin reuptake inhibitors (SSRIs) are a group of drugs used to treat various psychiatric disorders such as major depression, generalised anxiety, and obsessive-compulsive syndrome. Although generally safe, SSRIs can lead to various adverse effects, including an increased risk of bleeding due to their effect on platelet activation and aggregation. Unexpected bleeding can occur at different sites, in people of different age groups, and in combination with other medications. The commonly reported risk factors associated with medication-induced bleeding in patients with mental disorders include co-administration of other drugs and liver cirrhosis or failure. We report a relatively less common adverse effect of SSRIs. This is the case of a Middle Eastern man, known to have seasonal allergic rhinitis, who developed self-limiting epistaxis following the use of escitalopram for a depressive disorder. Since a history of seasonal allergy can precipitate bleeding when using SSRIs, risk factors for bleeding associated with SSRIs, excluding the common causes, should be explored for better management and prevention.

Keywords: Allergic rhinitis; Bleeding; Epistaxis; Escitalopram; Selective serotonin reuptake inhibitors

Introduction

Selective serotonin reuptake inhibitors (SSRIs) are a group of drugs developed in the 1970s.[1] They inhibit the serotonin reuptake by serotonin transporters into the synapses, thereby causing an increased serotonin concentration in the synaptic clefts. In addition, these drugs might also bind to and work on postsynaptic receptors other than serotonin receptors such as adrenergic, histamine, muscarinic, dopamine,[2] and acetylcholine receptors; however, the effects are negligible or minimal.[3]

SSRIs have been approved for the treatment of various psychiatric conditions, such as major depressive disorder, generalised anxiety disorder, and obsessive-compulsive

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disorder.[3] Although these drugs are as efficacious as the classical antidepressants, namely, tricyclic antidepressants[1] and monoamine oxidase inhibitors,[3] they are often more widely used because of their safety and tolerability profiles.[1] Despite their safety profile, SSRIs can still have various adverse effects, including gastrointestinal problems, weight gain, sexual dysfunction, hyponatraemia, and bleeding.[4] These complications might be mediated by different subtypes of serotonin receptors and increased serotonin levels.[1]

SSRIs are also known to affect platelet activation and aggregation, thereby increasing the bleeding time.[5] A previous study showed an increase in the bleeding time following the use of fluoxetine.[6] There are also reports of abnormal bleeding tendency and haemostatic changes following the use of fluoxetine, sertraline, and paroxetine.[5] The use of SSRIs might increase the risk of bleeding by 36 %.[7] A relationship between depression and increased platelet activity was also observed.[5] It was also found that SSRIs downregulate serotonin receptors on the platelets,[8] this, in turn, affects haemostasis homeostasis. In addition to causing gastric acidity and ulceration, increasing the likelihood of bleeding,[9] and disrupting platelet aggregation.[10] Certain risk factors for bleeding have been identified, including co-administration of SSRIs with other drugs (warfarin and aspirin)[8] and cirrhosis or liver failure.[11] Additionally, previous studies have shown that alcohol affects haemostasis primarily through platelet destruction, decrease in platelet aggregation and release, and other platelet-related factors, increasing the risk of bleeding; such effects are reversible with discontinuation of alcohol intake.[12] Alcohol decreases platelet aggregation by affecting the response to adenosine 5′-diphosphate (ADP), collagen, epinephrine, and thrombin. An increase in alcohol consumption increases the effect on platelet activity, which returns to normal after discontinuation of alcohol. However, there are contradictory reports about alcohol leading to partial platelet activation within the first few minutes of ingestion. In addition, alcohol might affect coagulation factors, which is exhibited by decreased levels of fibrinogen, von Willebrand factor, and Factor VII, and by an increase in fibrinolysis.[13] More specifically, it was found that alcohol exerted a statistically significant, dose-related antiplatelet effect on patients with idiopathic epistaxis, in addition to increased blood pressure, which might cause rupture of the nasal artery.[14]

Epistaxis is defined as bleeding from the nose due to any cause.[15] The causes can be classified as local or systemic.[16] Local causes include neoplasia, inflammatory disease, and trauma, and systemic causes include bleeding diathesis, alcoholism, age (elderly and children), and dryness in the winter season.[16] Some medications, such as nonsteroidal anti-inflammatory drugs, clopidogrel, and warfarin[16] are associated with an increased risk of epistaxis. One report described a case of epistaxis induced by topiramate.[17] In addition to reports of intranasal steroids, antibiotics, and SSRIs, anticoagulants, and antiplatelet agents have been reported as major medications that induced epistaxis.[18] A retrospective cohort study found that warfarin alone or in combination with aspirin increases the risk of epistaxis; however, no such risk was seen with SSRIs.[19] In contrast, another retrospective cohort study reported allergic rhinitis ($P < 0.0001$) and anticoagulants ($P < 0.0001$) as predictors of epistaxis, which was seen in 24.14% and 64.40% of the cases, respectively. Alcohol abuse, nonetheless, was not a predictor ($P = 0.3711$), with epistaxis occurring in only 1.20% of the cases; however, SSRI use was not assessed in that study.[20]

There are few reports about escitalopram (ESC)-induced epistaxis. However, this is the first report of a patient with seasonal allergy developing epistaxis with ESC. Herein, we report the case of an adult male with seasonal allergic rhinitis who developed epistaxis after starting ESC, although the laboratory parameters were normal.

Case presentation

The patient was a middle-eastern Arabian man in his early 20s, who was otherwise healthy except for a history of seasonal rhinitis during the spring season that usually presented with symptoms of nasal congestion, itching, and ocular symptoms. He first presented with situational anxiety and symptoms of depression. He consumed alcohol occasionally, and was initiated on ESC 5 mg in the month of January, and the dose increased to 10 mg per day after a few days, with a good initial response. The patient experienced epistaxis after taking 5 mg ESC, although it subsided spontaneously, a few days after increasing the dose to 10 mg, which is the adult therapeutic dose of ESC. He did not report a recurrence of epistaxis during the assessment, two weeks after starting the medication. There was no bleeding at other sites, ecchymosis, or purpura, and no other medication was administered simultaneously.

The results of the laboratory parameters investigated while the patient was on a 10 mg dose, were within normal range [complete blood count: red blood cells, $5.20 \times 10^6/\mu L$ (reference: $4.50-5.50 \times 10^6/\mu L$); haemoglobin, 15.2 g/dL (reference: 13−17.50 g/dL); haematocrit, 45.2% (reference: 40−54); mean corpuscular volume (MCV), 87 fL (reference: 75−98 fL); mean corpuscular haemoglobin (MCH), 29.2 pg (reference: 26−32 pg); mean corpuscular haemoglobin concentration (MCHC), 33.6 g/dl (reference: 32−36 g/dL); red cell distribution width-coefficient of variation (RDW-CV), 12.9% (reference: 11−14%); WBC, $10.6 \times 10^3/\mu L$ (reference: $4-11 \times 10^3/\mu L$); platelet count, $232 \times 10^3/\mu L$ (reference: $150-450 \times 10^3/\mu L$); coagulation profile: prothrombin time (PT), 13.6 s (reference: 12−15 s); partial thromboplastin time (PTT), 26.5 s (reference: 22−32.6 s); international normalised ratio (INR), 1.05 (reference: 0.8−1.2)].

The dose of ESC was then increased to 15 mg, and the patient again experienced similar episodes of epistaxis accompanied by other common adverse effects associated with SSRIs, such as dry mouth and a headache, which started after initiating ESC therapy and was unresponsive to analgesics. After exploring the possible options due to an inadequate response to treatment and with the reported adverse effects, the medication was switched to fluoxetine (20 mg per day), which is also associated with epistaxis.

The patient never had a history of epistaxis despite having allergic rhinitis, and no symptoms of active rhinitis were seen at the time of presentation. The episodes of epistaxis had the same severity with both SSRI and did not change with the administration of a higher dosage. There were no obvious deformities of the nose or septal deviation; however, the

patient did not undergo a complete otorhinolaryngological examination at the time of the episodes.

Discussion

SSRIs cause bleeding by affecting haemostasis, as they cause serotonin depletion in the platelets, which leads to vasoconstriction and platelet aggregation. SSRIs might also cause intracranial bleeding, postpartum haemorrhage, ecchymosis, gum bleeding, and vaginal bleeding, with upper gastrointestinal bleeding being the most common.[9] Previous studies have shown drug–drug interactions through cytochrome-P450 (CYP) that might be associated with a risk of bleeding when combined with medications that have a similar bleeding risk such as duloxetine, fluvoxamine, fluoxetine, paroxetine [prominent (CYP) inhibitors], citalopram, sertraline, ESC, and venlafaxine [weak CYP inhibitors].[21] Furthermore, it was found that antidepressants with a higher affinity towards serotonin transporters, such as fluoxetine, paroxetine, and sertraline, have a higher risk of bleeding. Citalopram, fluvoxamine, and ESC have an intermediate affinity, and mirtazapine, agomelatine, and desipramine have a low affinity towards serotonin transporters.[21,22] In another study, ESC was classified as having a high affinity towards serotonin transporter along with paroxetine, sertraline, and fluoxetine.[23] Atar et al. examined the antiplatelet activity of ESC in adults without depression, having more than two risk factors for coronary artery diseases. ESC, at a high dose, caused significant inhibition of ADP-induced platelet aggregation and decreased collagen-induced aggregation.[24] In adults with metabolic syndrome, moderate inhibition of ADP-induced platelet aggregation and decreased collagen-induced aggregation have also been reported with a high dose of ESC.[25] In one study, the mean platelet volume was high in patients with depression and was reduced after treatment with ESC (10–20 mg/day), along with a reduction in the platelet count.[26]

Literature about the correlation between the dose and the duration of SSRI therapy and occurrence of epistaxis is limited. A previous study did not find any correlation between the dose or duration of SSRI intake and increased risk of intracranial bleeding.[27] However, there are a few reports about the resolution of epistaxis with dose reduction. Two case reports described the resolution of epistaxis and haematuria with a decrease in the SSRI dose.[28] One study reported resolution of epistaxis after a decrease in the dose of ESC from 20 mg to 15 mg per day.[29] Yet another case series reported resolution of epistaxis either following discontinuation of the medication or dose reduction in children and adolescents.[30] A previous review found that the correlation between the duration of SSRI intake and bleeding was inconclusive; however, the risk was highest during the first month of treatment.[21]

There are several reports of bleeding following the intake of ESC, including that of inter-menstrual spotting in a woman without any gynaecological disorders, following a daily intake of ESC 10 mg. The spotting disappeared after discontinuing ESC, although it re-appeared on re-initiating ESC 5 mg daily.[31] Some studies have reported upper gastrointestinal bleeding following the intake of ESC.[32] Bleeding (upper gastrointestinal, epistaxis, and anaemia) was also reported in another patient with acute coronary syndrome, who was on ESC.[33] In another report, epistaxis was reported in a male patient taking ESC 20 mg daily; however, it resolved by reducing the daily ESC dose to 15 mg.[29] In an open trial, a patient with night eating syndrome discontinued ESC due to epistaxis.[34] Bleeding from multiple sites has been reported with the intake of ESC, wherein a patient had mild nasal and rectal bleeding following the intake of ESC in combination with mirtazapine and venlafaxine. The bleeding subsided once the doses of all drugs were reduced. However, there was no report of bleeding while the patient was on ESC 20 mg/day monotherapy, which suggested a dose-dependent relationship between ESC and bleeding.[35] A patient on citalopram had epistaxis and subconjunctival haemorrhage that resolved after discontinuation of the medication.[36] Continuation of the same dose of medication was not reported in any of the cases; hence, the effect of duration has not been well researched.

Epistaxis has also been reported with the use of other SSRIs alone or in combination with other medications. Fluoxetine caused hyponatraemia and epistaxis in an elderly woman[37] and fluvoxamine caused sporadic epistaxis in adult male[38]; another elderly woman experienced epistaxis while on paroxetine and limaprost alfadex.[39] In another study, the combination of fluoxetine and risperidone, but not their monotherapies, was shown to cause epistaxis.[40] Epistaxis was also reported with other antidepressants; mirtazapine was reported to cause thrombocytopenia, neutropenic fever, hypo-cellularity of the bone marrow, and epistaxis.[41] Venlafaxine[42] can also cause epistaxis. However, there are some contradictory reports as well. In a patient with a history of recurrent epistaxis, cauterisation was performed to stop bleeding. However, intake of fluoxetine 20 mg led to stopping of epistaxis.[43] In contrast to bleeding, there was a report of venous thromboembolism with ESC.[44] Another study reported bilateral multifocal pulmonary thromboembolism in a 70-year-old diabetic woman on ESC therapy.[45]

The risk of bleeding is seen not only in adult patients; there are reports of bleeding in children as well. ESC therapy is an independent risk factor for bleeding in paediatric patients on warfarin46. A report about epistaxis with sertraline in children and adolescents has also been published.[30]

Limitations

The patient's baseline laboratory values, which included the complete blood count and coagulation profile, were unavailable for comparison. Ideally, the patient should have been evaluated by an otolaryngologist at the time of the bleeding. In addition, the patient had epistaxis at different doses and upon starting; thus, we cannot report a definite dose on which the patient developed epistaxis.

Conclusions

In conclusion, the risk of bleeding with the use of SSRIs has long been known. Our patient, without any of the commonly known risk factors for epistaxis, developed epistaxis following the intake of ESC. Since he was an

occasional drinker and had a history of seasonal allergy, these might have been the probable risk factors for ESC induced epistaxis in this case. Thus, clinicians should be cautious while prescribing ESC to patients with a history of rhinitis.

Recommendation

Further studies are necessary to identify the high-risk groups of patients who might develop epistaxis following the intake of SSRIs for safer administration and prevention and better management of bleeding.

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Conflict of interest

The author has no conflict of interest to declare.

Ethical approval

Informed consent was obtained from the patient for publication of this case report.

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