TARDIVE DYSKINESIA WITH HIGH DOSE H1 ANTIHISTAMINE IN A PERSON WITHOUT COMORBIDITIES

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ABSTRACT This case report is about one of the rare side effect of high doses of H1 antihistamine (tardive dyskinesia) in a 37-years-old woman without comorbidity and without another parallel medicine intake.

KEYWORDS TD tardive dyskinesia, Fexofenadine, Citrizin, clemastine, Prednisolon, orobuccolingual dyskinesia, Antihistamine, Comorbidity, Urticaria, OTC (out of the counter), PD Parkinson Disease), HD Huntington Disease

Case report

A thirty-seven-year-old woman without any chronic disease started to take cetirizine (OTC) against generalized skin allergic reaction (urticaria) because of an unknown allergen in the last days of February month 2021 in the capital region in Denmark.

Patient mother and mother side of the family are well own for allergy and asthma.

There were 2 cases of an old age brain tumour on the mother's side of the patient.

She had generalized urticaria and pruritus despite cetirizine 10 mg daily in 3 days.

She did not have other types of allergic symptoms. After three days, it starts to flush in the face, and it itches in the whole of the body and urticaria. After 2 hours starts swelling of tongue and respiration difficulty. After 112 calls, under diagnose of quincke oedema. Receive iv clemastine and corticosteroid and afterwards adrenalin inhalation with a good outcome. Patient discharged from hospital with continuous fexofenadine 360 mgx2 daily and six-day time-limited Prednisolon 25 mg daily tablet. Urticaria disappeared with fexofenadine and Prednisolon treatment. Patient receives fexofenadine from 4 mars to 14 mars against a severe allergic reaction. Patient finished her Prednisolon treatment and continued just fexofenadine with the same doses to prevent allergic reaction according to medical prescription.

Gradually during fexofenadine treatment, pt had uncontrolled movement around the mouth and jaw and tongue progressive with different intervals between hours to minutes, with a duration between half an hour to seconds. Patient was completely conscious and aware of her uncontrolled movement in her face.

Patient never has uncontrolled movement upper part of her face. Patient has fexofenadine daily for five days without any other medication. The last admission in the hospital was because of suspicion about partial epilepsy and therefore made acute EEG (during dyskinesia face movement), and that shows no evidence for epileptic activities.

Patient tried to stop taking fexofenadine after 11 days (total fexofenadine intake) and can feel better and less episode of involuntary movement I her face after 12 hours (of fexofenadin stop) have pt much less movement and after 24 hours after antihistamine stop, was pt completely symptom free.

Patient observation shows when dyskinesia movement appears around the mouth and jaw, it can be stopped with a finger press on the jaw or moving part of the face. (It is a typic and well-known finding in tardive dyskinesia).

Lab test results

No evidence of allergies according to complete allergic follow-up. Normal epilepsy follow-up: EEG normal. MRI cerebrum with contrast normal. Face picture with dyskinesia attack.

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Figure 1: Face picture with dyskinesia attack.

Discussion

About H1-receptor antagonists

The H1 antagonists have two types: first-generation and second-generation. First-generation drugs are more sedative than the other ones.

The first-generation maybe block autonomic receptors. H1 drugs can immediately be absorbed after oral intake, with peak blood concentrations in 1–2 hours. They can be distributed throughout the body, and the first-generation agent can enter the central nervous system. Nearly Most of the agents have an effective duration of action of 4–6 hours following a single dose; second-generation agents are longer-acting, with a duration of action of 12–24 hours.

The second-generation agents are less lipid-soluble than the first-generation drugs and are substrates of the P-glycoprotein transporter in the blood-brain barrier; Therefore they can enter the central nervous system very difficult or not at all.

The active metabolites of hydroxyzine, terfenadine, and loratadine are available as drugs (cetirizine, fexofenadine, and desloratadine, respectively).

Both neutral H1 antagonists and inverse H1 agonists reduce or block the actions of histamine by reversible competitive binding to the H1 receptor. Some agents work as inverse agonists, and it is possible that all act by this mechanism. They have negligible potency at the H2 receptor and little at the H3 receptor.

Abnormal movement

Stereotypic movements definition are repetitive, sometimes rhythmic movements.

The movements can occur related to the syndrome of drug-induced tardive dyskinesia and most often affect the mouth; in orobuccolingual dyskinesia.

For example, constant movements of the jaw, tongue, and lips. (merit textbook of neurology edition 13)

Tardive dyskinesia can occur by neuroleptic drugs and other dopamine receptor blockers, particularly metoclopramide.

Other drugs, for example, dopamine agonists, anticholinergics, and antihistamines can causes dyskinesia.

Multiple infarctions in the basal ganglia and lesions in the cerebellar vermis can be considered as a reason for dyskinesia. (Bradley page 239)

Opposite to the nature of chorea, tardive dyskinesia usually indicates repetitive stereotypical movements, which are in the orolingual region (Mejia and Jankovic, 2010; Waln and Jankovic, 2013).

Movements in tardive dyskinesia located to the lower face, whereas in HD, irregular contractions of the frontalis muscles and associated elevation of the eyebrows are common (Jankovic and Roos, 2014). The gait is normal with tardive dyskinesia, although a bizarre gait can occur sometimes.

TD nearly occurs about six weeks or more of dopamine receptor blockade, but onset as soon as after the first dose can occur.

Risk factors for tardive dyskinesia include old age, female gender, affective disorder, and edentulousness.

The typic manifestation of TD is repetitive stereotypic movements of the mouth, tongue, and lower face. (Waln and Jankovic, 2013)

The mechanisms of TD is partially understood, but the agents that cause this syndrome shows to exhibit potent binding to postsynaptic D2 receptors (Waln and Jankovic, 2013).

Denervation supersensitivity of the postsynaptic dopamine receptor has been considered as a reason.

PET investigation shows upregulation of D2 receptors in neuroleptic-treated patients.

With TD symptoms, Neuroleptics should be stopped if possible.

Tocopherol can treat TD symptoms. Mild TD can be managed with benzodiazepines or baclofen.

Catecholamine-depleting drugs, specially tetrabenazine, can be used in severe Tardive dyskinesia. (bradly page 1450)

Conclusion

Tardive dyskinesia is a known rare side effect for some antihistamines. About fexofenadine with named dose can be considered to need more investigation and future interest to determine the possible unknown origin of such side effect. Fexofenadine is OTC (out of the counter) Medicine. It, therefore, can be more important to be sure about how is prevalence or incidences of neurologic side effect in different doses and possible toxicity risk level with high doses.

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Conflict of interest

There are no conflicts of interest to declare by any of the authors of this study.

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