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Short Communication

Types and Implications of Angioedema

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DESCRIPTION

Angioedema is characterised by a localised extravasation of plasma and a fast increase in permeability of post-capillary venules and submucosal or subcutaneous capillaries. Histamine or bradykinin release is necessary for the majority of angioedema causes, while other vasoactive chemicals might also play a role. Prostaglandins, leukotrienes, enzymes like tryptase, cytokines, and chemokines, on the other hand, lack solid data. Naturally, leukotrienes are suspected whenever angioedema is brought on by COX-1 inhibitors [1].

An abrupt, nonpitting swelling of the skin, mucous membranes including the upper respiratory and gastrointestinal tracts are referred to as angioedema. It normally lasts from few hours to three days, the affected tissues then go back to being normal. Sites of preference include the genitalia, hands, feet, and face. Swelling around the lips and eyes (periorbital) is most frequent. Problematic swelling can occur in the larynx, pharynx, and tongue [2]. Laryngeal edoema can be fatal, but pharyngeal edoema and tongue enlargement can also be deadly if they are severe.

Drug induced allergic angioedema

Histamine is the mediator of type I hypersensitivity, which is what causes drug-induced allergic angioedema. In type I hypersensitivity, the drug forms a cross-link with the Immunoglobulin E (IgE) antibody attached to mast cell surfaces, causing histamine to be released. Clinically, drug-induced allergic angioedema manifests as an abrupt onset of mucosal and submucosal tissue swelling. Additionally, the patient will exhibit a characteristic urticarial rash [3]. Treatment with antihistamines, epinephrine, and corticosteroids will have a fast effect on the symptoms.

Drug-induced non-allergic angioedema

Bradykinin mediates drug-induced non-allergic angioedema. Drug-induced non-allergic angioedema does not have an urticarial rash. In addition, compared to histamine-mediated angioedema, the onset is more progressive. The onset of symptoms may last three to five days. Drug-induced non-allergic angioedema causes symptoms that are resistant to antihistamine and steroid treatment, and the condition only goes away when the drug is stopped.

Hereditary angioedema

Hereditary angioedema is a rare genetic condition marked by recurring episodes of fluid accumulation outside of blood vessels, obstructing normal blood or lymphatic fluid flow and resulting in rapid swelling of tissues in the hands, feet, limbs, face, digestive system, or airway. Unlike an allergic reaction, which may be accompanied by itching, this swelling typically does not have that. Cramping results from gastrointestinal tract swelling. An obstruction caused by the airway swelling has the potential to be a very dangerous consequence [4]. These symptoms arise from a lack of or dysfunction of specific proteins that support the maintenance of the regular flow of fluid through very small blood vessels (capillaries). Fluid may occasionally build up in other internal organs. The seriousness of the disease varies greatly among affected individuals.

Hereditary angioedema is characterised by recurrent episodes of swelling in the affected areas brought on by the buildup of excessive bodily fluid (edema). The hands, feet, eyelids, lips, and/or genitalia are the body parts most frequently impacted. In addition to the mucous membranes that line the digestive and respiratory systems, hereditary angioedema patients are more likely to experience angioedema than those who have other types of angioedema (i.e., acquired or traumatic). People who have this condition frequently experience firm, severe swelling instead of red, itching spots (pruritic), rarely is a skin rash (urticaria) evident [5].

CONCLUSION

In the emergency room, angioedema is a frequent occurrence. When the airways are affected, it may be fatal. Many doctors treated it as though it were an allergic reaction. Angioedema management differs from that of allergic angioedema, hence there

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is a need to raise awareness of the condition.

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