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lactic shock: mechanisms and treatment

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SUMMARY

This paper reviews the mechanisms of anaphylactic shock in terms of the immunoglobulin and non-immunoglobulin triggering events, and the cellular events based on the rise in intracellular cyclic AMP and calcium that release preformed granule-associated mediators and the rapidly formed, newly synthesized mediators predominantly based on arachidonic acid metabolism. These primary mediators recruit other cells with the release of secondary mediators that either potentiate or ultimately curtail the anaphylactic reaction. The roles of these mediators in the various causes of cardiovascular collapse are examined. The treatment of anaphylactic shock involves oxygen, adrenaline and fluids. The importance and safety of intravenous adrenaline are discussed. Combined H_1 and H_2 blocking antihistamines and steroids have a limited role. Glucagon and other adrenergic drugs are occasionally used, and several new experimental drugs are being developed.

Key words: adrenaline, anaphylactic shock, anaphylaxis, histamine H_1 receptor blockaders, histamine H_2 receptor blockaders

INTRODUCTION

The first case of anaphylaxis was recorded on the tomb of King Menes, an Egyptian Pharaoh who died suddenly in 2640 BC following a wasp sting.[1] The term anaphylaxis is derived from the Greek and means literally 'against protection'.[2] It was introduced in 1902 by Charles Richet and Paul Portier following experiments with unexpected fatal reactions on dogs sensitized to Portuguese man-of-war venom, observed the previous year whilst on board Prince Albert of Monaco's yacht in the Mediterranean. Richet was subsequently awarded the Nobel Prize in Medicine and Physiology in 1913.[3]

At around the same time, in 1906, Von Pirquet introduced the term 'allergy' although he assumed that the major allergic diseases, such as urticaria and asthma, were due to the absence of antibodies.[4] In 1921, Prausnitz and Küstner demonstrated [illegible] formed reagi[illegible] to a non-sens[illegible] until 1967 that [illegible] this reagin a[illegible] known as IgE. [illegible] classified hyp[illegible] immediate hyp[illegible] in practice these types need not necessarily occur in isolation from each other.[5] In immunological terms, anaphylaxis is an example of an immediate, Type-1 hypersensitivity reaction.

DEFINITION

Currently, the term anaphylaxis is best used to describe the rapid, generalized and often unanticipated, immunologically mediated events that occur after exposure to certain foreign substances in previously sensitized persons. Anaphylactoid reactions describe a clinically identical syndrome involving similar mediators but not triggered by IgE antibody and not necessarily requiring previous exposure. Despite important aetiological distinctions, the term anaphylaxis is commonly used to describe both of these clinical syndromes, even when the mechanisms are unknown.[6] The most common life-threatening feature of acute anaphylaxis is cardiovascular collapse or shock. Other life-threatening effects include bronchospasm, angio-oedema and pulmonary oedema.[6,7] This paper will focus on the mechanisms and both general and specific treatment of anaphylactic shock.

MECHANISMS OF ANAPHYLACTIC SHOCK

Mechanisms of anaphylactic shock may be divided

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