CHEST Online Supplement

Anaphylaxis

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CHEST 2018; 153(2):528-543

 DOI: 10.1016/j.chest.2017.07.033

e-Table 1. Anaphylaxis – A Historical Perspective

2641 BC	Hieroglyphs show Pharaoh Menes dying from a bee sting reaction
1878	Paul Ehrlich* discovers mast cells
1901	Jokichi Takamine arranges to market "Adrenalin" in USA by Park, Davis and Co.
1902	Paul Portier and Charles Richet* describe anaphylaxis
1903	Nicolas Maurice Arthus describes "Localized anaphylaxis" now known as the Arthus reaction
1906	Clemens Von Pirquet coins the term "Allergy" ("allos" meaning other and "ergos" meaning activity
1907	Alexandre Besredka introduces the term "anaphylactic shock"
1910	Sir Henry Dale* describes role of histamine in allergic reactions
1913	Charles Richet awarded Nobel Prize for discovery of anaphylaxis
1921	Prausnitz and Küstner transfer immediate hypersensitivity via serum From an allergic to a non-allergic individual
1921	Coka and Cooke introduce the term "atopy"
1937	Daniel Bovet* discovers antihistamines
1944-48	Diphenhydramine (Benadryl) and Chlorpheniramine introduced
1948	Cortisone administered to arthritis patients with relief of symptoms
1949	Cortisone introduced for clinical use by Merck and Company
1950	Nobel Prize awarded to Edward Kendall, Tadeus Reichstein and Philip Hench for discoveries related to adrenal corticosteroids
1964	Sir James Black* discovers H2 receptor and cimetidine
1966	Kimishige and Teruko Ishizaka discover immunoglobulin E (IgE)
1982	Bengt Ingemar Samuelsson* awarded Nobel Prize for identifying leukotrienes as the "slow reacting factor of anaphylaxis"
1994	Pork-cat syndrome and anaphylaxis to mammalian meats after cat sensitization reported
2008	Chinese heparin adulterated with over-sulfated chondroitin sulfate resulting in anaphylactic reactions
2009	Delayed anaphylaxis to galactose-1,3-alpha-galactose in mammalian meat discovered

*-seminal discoveries of relevance to anaphylaxis and its management

e-Table 2. Mast Cell Activation Disorders and Idiopathic Anaphylaxis: Diagnostic aspects and differentiation of mast cell disorders from idiopathic anaphylaxis

H=high levels; N=normal levels

FINDINGS	Systemic mastocytosis	Monoclonal mast cell activation syndrome	Mast cell activation syndrome	Idiopathic anaphylaxis
Multifocal aggregates in marrow or ECT[1]	**Yes**	**No**	**No**	No
D816V mutation[2]	**Yes**	Yes	**No**	No
Aberrant CD25 expression on mast cells	**Yes**	Yes	**No**	No
Tryptase baseline	H	H/N	H/N	No
Urine 11β-PGF2α[3]	H	H/N	H/N	N
Cutaneous mastocytosis	Yes	No	No	No
Response to medication	Good	Good	Good	Variable

[1]ECT=extra-cutaneous tissue ; [2]D816V= Point mutation of the KIT oncogene at codon 816 is seen in >90% of systemic mastocytosis (SM) cases; [3]11β-PGF2α is an intermediate metabolite in the arachidonic acid metabolic pathway, and is irreversibly produced from prostaglandin D2
Tissue mast cell aggregates, D816V mutation, urinary prostanoids excretion, tryptase and staining for CD25 allows syndrome differentiation of mast cell disorders and separation from idiopathic anaphylaxis