Continuous education in sedation: Anaphylaxis management during procedural sedation

SADJ September 2016, Vol 71 no 8 p368 - p369

JA Roelofse,[1] C Lapere,[2] A von Backstrom[3]

INTRODUCTION

Anaphylaxis is a medical emergency that requires immediate recognition and intervention. Equipment and medication should be readily available. Early administration of adrenalin is the key to survival. In this paper we discuss the critical aspects of anaphylaxis: the definition, presentation, the pathophysiology, the best emergency management and measures to prevent recurrence.

Keywords: anaphylaxis, adrenaline, laryngeal oedema, emergency management, procedural sedation

DISCUSSION

Anaphylaxis is a severe, life-threatening, systemic hypersensitivity reaction that is immunologically mediated.[1] This unpredictable syndrome involves multiple organs as a result of the sudden systematic release of mast cells and basophil mediators.[2] Nothing should delay the early administration of intra-muscular adrenaline as this is the key to a successful outcome.[1]

Many definitions, criteria and terminology exist for anaphylaxis. Sampson *et al* proposed a consensus that is widely agreed upon.[3] Anaphylaxis is likely when any one of the three following criteria is met:

1. Acute onset of an illness with involvement of skin and/or mucosal tissue (flushing, urticaria, angioedema) with at least one of the following:
   a. Respiratory compromise (e.g. dyspnea, bronchospasm, stridor)
   b. Hypotension or associated symptoms of end-organ dysfunction (e.g. hypotonia, syncope)
2. Two or more of the following rapidly occurring symptoms following exposure to a likely allergen:
   a. Involvement of the skin-mucosal tissue (generalized hives, swollen tongue)
   b. Respiratory compromise (e.g. dyspnea, bronchospasm, stridor)
   c. Hypotension or associated symptoms (e.g. hypotonia, syncope)
   d. Persistent gastro-intestinal symptoms (e.g. abdominal pain, vomiting)
3. Hypotension and exposure to a known allergen for the patient
   a. Infants and children: low systolic blood pressure (age specific) or > 30% decrease in systolic blood pressure
   b. Adults: systolic blood pressure of < 90 mmHg

The syndrome can develop in minutes to hours, therefore the sedation practitioner must be vigilant and able to recognize the development of symptoms suggestive of anaphylaxis. Monitors cannot change the outcome, therefore practitioners need to monitor what they cannot control. We accept that different levels of sedation require different standards of monitoring, yet, any patient who receives sedation of any kind needs some monitoring according to SASA Guidelines. Moderate sedation levels require continuous monitoring that includes pulse oximetry, heart rate, respiratory rate and pattern of breathing, and the recording of blood pressure.[4]

The manifestations of anaphylaxis are seen in the cardiovascular system, upper and lower respiratory tract, gastrointestinal tract and skin. Some or all of the following features can be present: nasal congestion, rhinorrhoea, lacrimation; generalized erythema, pruritus, urticarial, angio-oedema, bronchospasm, laryngeal oedema, nausea, vomiting, hypotension, myocardial ischaemia and cardiac arrhythmias.[5] A feeling of impending doom (*angor animi*) is often the first symptom to occur.

Food most commonly triggers anaphylaxis in children, and drugs in adults. Certain drugs like muscle relaxants, antibiotics and aspirin are more often the culprits.[6]

Local anaesthetics: Anaphylactic reactions to amide and ester type local anaesthetics are extremely rare.[7]

Propofol has a 1:60 000 incidence of anaphlaxis. Initially there was a higher incidence due to the cremophor used as a preservative.[8] There is not an increased incidence in patients allergic to eggs.[7]

Antibiotics: Penicillin is the most common cause of anaphylaxis in the community and may be responsible for up to 75% of deaths due to anaphylaxis.[7] Only a minority of patients who report an allergic reaction to penicillin have a documented allergy on skin testing.[7]

1. **James A Roelofse:** *MB.ChB, MMed, PhD, Dip NDBA (USA).* Professor University of the Western Cape, Visiting Professor, University College London.
2. **Cherese Lapere:** *MB.ChB, DipPEC, DA(SA), PDD.* Sedation Practitioner.
3. **Andre von Backstrom:** *MB.ChB, Dip Sed.* Sedation Practitioner, Sedation Solutions, London.

Corresponding author
James A Roelofse:
Private Bag X1, Tygerberg 7505. Tel: 021 937 3085,
Cell: 083 458 2427. E-mail: jar@sun.ac.za

Certain factors contribute to the severity and the fatality risk of anaphylactic reactions: poorly controlled asthma (particularly in adolescents and young adults), underlying cardiovascular disease and extremities of ages (young children and adults above 55).[9]

Important chemical mediators of anaphylaxis include cytokines, preformed granule-associated substances (histamine, tryptase, chymase) and lipid derived mediators (prostaglandins, leukotrins). These are released due to the degranulation of mast cells and basophiles.[2] Histamine release is the pivotal event that activates H1 and H2 receptors. Activation of H1 receptors causes pruritus, rhinorrhoea, bronchospasm, flushing and tachycardia. H2 receptors mediate increased vascular permeability and hypotension.

These chemical mediators could affect the myocardium directly. H1 receptors mediate coronary artery vasospasm and increase vascular permeability. H2 receptors mediate increased atrial and ventricular contractility, increased atrial rate and coronary artery vasodilatation. Anaphylaxis has been associated clinically with myocardial ischemia, conduction defects, atrial and ventricular arrhythmias and T wave abnormalities.

The treatment of anaphylaxis should begin with a rapid assessment, maintenance of airway, breathing and circulation and recognition of the presenting alarm symptoms.[3] Help should be called immediately and the practitioner should initiate emergency treatment.[3] Of great importance is the discontinuation of the exposure to the allergen (if known). When the criteria for the diagnosis are fulfilled, adrenalin should be given without delay.[3] The sedation practitioner needs to be able to identify these clinical features in correlation with the diagnostic criteria to prevent the delay of life-saving treatment.

A prominent risk factor for fatal anaphylaxis is the delay in the administration of adrenaline. Early administration of adrenaline is defined as the correct dose given within 30 minutes of exposure to the allergen: 0,01mg/kg (0.5mg maximum dose) administered intramuscularly (anterolateral thigh), repeated every 5-15 minutes.[3,10] Intravenous dose is dependent on severity: 10µg-1mg boluses, with subsequent infusions as needed.

Further management includes patient positioning by elevating the legs in the supine position[3], and rapid intravenous fluids via large bore cannulas, concurrently giving high flow of oxygen.[1] H1 antagonists: e.g. Diphenhydramine/Promethazine 25-50mg given intramuscularly or slow intravenous administration of H2 antagonists: Ranitidine 1mg/kg or cimetidine 4mg/kg. Treat bronchospasm as per usual protocol.

Should cardiac arrest occur, good quality CPR should be initiated immediately as per current resuscitation protocols, including intravenous adrenalin.[10]

Patients taking β-adrenergic antagonists may be more likely to experience severe reactions characterized by a slow pulse, severe hypotension and bronchospasm. Glucagon can be given to these patients.[11]

The major causes of death due to anaphylaxis is listed as asphyxia, shock, disseminated intravascular coagulation and adrenalin overdose.

Recurrent or biphasic anaphylaxis may occur in up to 20% of patients[3], usually within 8-12 hours. Corticosteroids should be given to prevent the incidence of recurrence, Hydrocortisone up to 2g or Methylprednisolone as an alternative, can be given intravenously.

The patient should be referred to an Emergency unit for further management.

After an anaphylactic event, the patient should be referred to an allergist for testing. Skin-prick, intradermal or serological testing are the usual modalities used. Skin prick testing has a high predictive value in the setting of a history of anaphylaxis. Intradermal testing is used for local anaesthetics, propofol and muscle relaxants. Skin testing should be performed 4-6 weeks after an anaphylactic reaction. Risk of triggering anaphylaxis is small (<0,1%), but resuscitative equipment must be available.

Measures to reduce the incidence of anaphylaxis and anaphylactic deaths include appropriate training and counseling on the use of auto-injectors for the patient and their families.[1] Patients should also wear bracelets or warning identification. Patients and families need to be taught to use the adrenaline auto-injector and cautioned to keep the adrenaline kit with them. Patients need to be able to identify the allergen to which they are allergic and know how to avoid it.[1] All anaphylactic drug reactions should be reported.

Sedation practitioners always need to obtain a thorough history for drug allergy.

CONCLUSION

Anaphylaxis is a severe, life-threatening, systemic reaction that can occur within minutes after exposure to an allergen. Sedation practitioners need to know the clinical presentation as well as subtle signs and symptoms suggestive of the diagnosis. Early adrenalin administration is the key to the survival of patients with anaphylaxis.

References

1. Tse Y, Rylance G, Infirmary RV. Corrections. Arch Dis Child [Internet]. 2010;95(12):1071–1071. Available from: http://adc.bmj.com/cgi/doi/10.1136/adc.2007.120378
2. Kalesnikoff J, Galli SJ. Anaphylaxis: Mechanisms of mast cell activation. Chemical Immunology and Allergy. 2010;95:45–66.
3. Sampson HA, Muñoz-Furlong A, Campbell RL, Adkinson NF, Allan Bock S, Branum A, *et al*. Second Symposium on the Definition and Management of Anaphylaxis: Summary report. Second National Institute of Allergy and Infectious Disease/ Food Allergy and Anaphylaxis Network Symposium. Ann Emerg Med. 2006;47(4):373–80.
4. Society of South African Society of Anaesthesiologists Sedation Guidelines 2015 Guidelines for the safe use of procedural sedation and analgesia for diagnostic and therapeutic procedures in adults. 2015;21(2):1–38.
5. Evans C, Tippins E. Emergency treatment of anaphylaxis. Accid Emerg Nurs. 2005;13(4):232–7.
6. Soar J, Pumphrey R, Cant A, Clarke S, Corbett A, Dawson P, *et al*. Emergency treatment of anaphylactic reactions-Guidelines for healthcare providers. Resuscitation. 2008;77(2):157–69.
7. Dippenaar J, Naidoo S. Allergic reactions and anaphylaxis during anaesthesia. Curr Allergy Clin Immunol.2015; 28(1):16-20
8. Koul A, Jain R, Sood J. A critical incident report: Propofol-triggered anaphylaxis. Indian J Anaesth. 2011;55(5):530–3.
9. Worm M, Babina M, Hompes S. Causes and risk factors for anaphylaxis. J Dtsch Dermatol Ges [Internet]. 2013;11(1):44–50. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23181736
10. Truhlář A, Deakin CD, Soar J, Khalifa GEA, Alfonzo A, Bierens JJLM, *et al*. European Resuscitation Council Guidelines for Resuscitation 2015. Section 4. Cardiac arrest in special circumstances. Resuscitation. 2015;95:148–201.
11. Mertes PM, Tajima K, Regnier-Kimmoun MA, Lambert M, Iohom G, Gueant-Rodriguez RM, *et al*. Perioperative anaphylaxis. [Review] [155 refs]. Med Clin North Am [Internet]. 2010;94(4):761–89. Available from: 20609862