A Case Report of Hypereosinophilic syndrome after Anaphylaxis presenting for Thoracic Endovascular Aortic Repair (TEVAR) for Type B Aortic Dissection

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ABSTRACT/INTRODUCTION

Currently there is no international consensus or guidelines on management of patients with hypereosinophilic syndromes undergoing anesthesia. Perioperative anesthesia management measures came from a mixture of case reports, review of management guidelines on eosinophilic disorders. This case report describes a case of hypereosinophilic syndrome undergoing thoracic endovascular aortic repair for type B aortic dissection with monitored anesthetic care after anaphylaxis with rocuronium for previous surgery.

Keywords: Hypereosinophilic syndrome; Anaphylaxis; Anesthesia; thoracic endovascular aortic repair

CASE REPORT

A 57-year-old male, weighted 57kg, of south east asian descent was scheduled to undergo an elective parathyroidectomy for primary hyperparathyroidism under general anesthesia. He was a chronic smoker. Other past medical history include hypertension with Type B aortic dissection, adrenal incidentaloma with unremarkable biochemical workup. After preoxygenation, the patient was induced with 100mcg fentanyl, 130mg propofol and 50mg rocuronium. 3 minutes into induction, he developed life threatening cardiovascular collapse with bronchospasm. Physical examination revealed bilateral expiratory wheeze and truncal rash. Anaphylaxis was suspected. Ventolin puff, hydrocortisone 100mg bolus and adrenaline 0.1mg boluses were given. Noradrenaline and adrenaline infusion commenced. Transthoracic echocardiogram (TTE) revealed normal left ventricular ejection fraction and no pericardial effusion. Operation was aborted. Arterial blood gas was grossly normal. Transesophageal echocardiogram (TEE) showed a small intramural hematoma distal to the aortic arch and no Type A aortic dissection. The patient was kept intubated to the intensive care unit on adrenaline infusion.

Subsequently the patient had an uneventful recovery and was discharged. He was referred to an immunology clinic for work up of anaphylaxis. Skin prick test immediate readings for latex, fentanyl, propofol, rocuronium and chlorhexidine were negative. Skin tests for neuromuscular blocking agents including atracurium, cisatracurium and suxamethonium were negative. Although the workup was negative, among the exposed items,

neuromuscular blocking agents related allergic reaction were most likely. Previous literature has reported that rocuronium may cause non-IgE mediated allergic reactions, which skin tests have limited sensitivity in non-IgE reaction detection. The conclusion by immunologists was still anaphylaxis considering the overall picture. He was thus labelled allergic to rocuronium.

2 months after the anaphylaxis incident, the patient presented to the Accident and Emergency unit for chest pain with radiation to jaw and back with profuse sweating. Electrocardiogram (ECG) was normal sinus rhythm. Urgent contrast CT thorax showed resolved Stanford type B aortic intramural hematoma. Similar saccular thoracic aortic aneurysm at mid descending thoracic aorta at T6-9 levels measuring up to 3.8cm in diameter. There was no active contrast extravasation or mediastinal hematoma. Incidentally, there was enlargement of bilateral axillary lymph nodes up to 1.2 cm with indeterminate nature. Blood tests revealed normal cardiac enzymes, severe hypereosinophilia >60% and the absolute eosinophil count was 13.2 x 10^9/L. Cardiology was consulted to rule out eosinophilic myocarditis. CT coronaries were normal. Echocardiogram showed ejection fraction of 60%, normal biventricular size and function, no gross valvular abnormality, no regional wall motion abnormality, no pericardial effusion. There was no evidence of eosinophilic myocarditis based on ECG, cardiac enzymes and echocardiogram results.

He later developed extensive pruritic erythematous papules with crusting and scaling over limbs, abdomen, chest wall, back and neck. Painful ulcerative lesions at finger tips were also present. Bloods revealed normal C3 and C4, ANA, anti ENA were negative. ANCA was weakly positive.Skin biopsy was done showing superficial perivascular lymphoplasmacytic infiltrate with occasional eosinophils. Bone marrow aspirate demonstrated megakaryocytic and granulocytic hyperplasia with eosinophilia. Cytogenetic analysis showed a normal male karyotype and fluorescence in situ hybridization (FISH) was negative for *PDGFRA*, *PDGFRB*, FGFR1, and JAK rearrangements.

On further physical examination, generalized lymphadenopathy was found and subsequently whole body PET CT was performed which showed generalized lymphadenopathy. Groin lymph node excisional biopsy showed dermatopathic lymph node. Pulmonary function tests were normal. He was diagnosed to have hypereosinophilic syndrome.

He was subsequently treated with prednisolone 1mg/kg/day for 2 weeks, stepped up to a maximum dose 75mg prednisolone daily and was gradually tapered down to a maintenance dose of 10mg prednisolone daily with significantly improved white cell count. He was also put on cyclophosphamide 50mg weekly and cyclosporin A 25mg daily.

3 months later, he was scheduled for TEVAR. Hematologist managing his hypereosinophilic syndrome commented he was medically optimized. After multidisciplinary discussion with cardiothoracic surgeons, taking into account the surgical complexity and anticipated duration of operation, anaesthesia plan was monitored anesthetic care with general anesthesia as back up.

The operation was performed at a hybrid theater with a built-in fluoroscopy in the main theater. Premedication with 4 puffs of salbutamol was given prior to induction. Standard monitoring as per American Society of Anesthesiologist (ASA) Standard II guidelines, with pulse oximetry for oxygenation, end tidal capnography, noninvasive blood pressure was placed on the left arm, arterial line 20G was placed at right radial. A 16 gauge IV access was set at the right hand. Stress dose hydrocortisone 200mg IV was given. Nasal oxygen supplement at 2L/min was started. 0.5mcg/kg dexmedetomidine loading was given and maintained with infusion at 0.5mcg/kg/hr. Concomitant target-controlled infusion propofol was started at 0.5ug/ml and gradually stepped up to 2.5ug/ml and maintained throughout surgery. Local anesthetic mixture of 0.25% levobupivacaine with 1% lignocaine with adrenaline at 2.5mcg/ml was locally infiltrated by the surgeon. Bolus of fentanyl 30ug was given just before femoral puncture. The patient was ventilated spontaneously with natural airway, and maintained hemodynamically stable throughout surgery vasopressor free. Nicardipine was given at 0.1mg to 0.2mg bolus to maintain systolic blood pressure at around 90-100mmHg near stent deployment to prevent stent migration. A Zone IV TEVAR was performed successfully. Post operatively he was monitored in a high dependency unit and was subsequently discharged on postoperative day 3.

DISCUSSION

Incidence of hypereosinophilic syndrome

According to the World Health Organization and International Consensus Classification of eosinophilic disorders 2024 update, the age-adjusted incidence rate for hypereosinophilic syndrome was approximately 0.4 cases per 1000000. It is a rare condition, with a reported annual incidence of 0.04 to 0.17 per 100,000 person-years in a UK based real world study.

Definition and diagnosis of hypereosinophilic syndrome

Hypereosinophilic syndrome (HES) is a rare disorder with variable organ involvement, requiring individualized anesthetic management. According to the consensus statements from International Cooperative Working Group on Eosinophil Disorders (ICOG-EO) in 2021,[1,2,3] HES is defined as hypereosinophilia accompanied by organ dysfunction attributable to eosinophil infiltration, after excluding other alternative explanations. Hypereosinophilia is defined as an absolute eosinophil count (AEC) ≥1500/microL in peripheral blood on two occasions ≥2 weeks apart. The severity of eosinophilia was further rated into mild (AEC above upper limit of normal to 1.5x 10^9/L), moderate (AEC 1.5-5 x10^9/L) and severe (AEC > 5 x10^9/L).[1] Our patient had severe and persistent hypereosinophilia on serial blood tests.

Clinical presentation

HES is a highly heterogeneous condition, ranging from asymptomatic to medical emergencies that require urgent treatment of end organ damage. Cutaneous and respiratory manifestations are the most common presentations of HES.[4,5]

Common dermatological symptoms of HES include pruritic papules, eczema, urticaria, angioedema and erythroderma.[6] The cutaneous involvement is demonstrated by prominent eosinophilic infiltration in a skin biopsy.

Pulmonary involvement often manifests as dyspnea, cough, and wheezing.[7] Investigations would include high-resolution CT, bronchoscopy and pulmonary function tests to exclude other etiologies of pulmonary infiltrate with eosinophilia.

Other potential organ damages include eosinophilic myocarditis, thromboembolic events, gastrointestinal symptoms and neurological complications.[8]

Medical management of HES

Categorising the underlying cause is important as it would affect management directions. The underlying pathology may reflect either a primary process (ie, clonal/neoplastic eosinophils) or a secondary process (reactive eosinophils); If no apparent cause is found, it will be classified as idiopathic HES.[3]

Patients with known *PDGFRA*-positive myeloid variants of HES will likely respond to imatinib mesylate.[9] For others, corticosteroids are typically the first line treatment.[10] In steroid refractory cases, additional agents such as mepolizumab,[11] hydroxyurea and interferon-alpha may be chosen based on toxicity profile and co-morbidities.

Thromboembolism is the leading cause of morbidity among patients with HES.[12] However, there is currently no clear evidence to support the prophylactic use of anticoagulants in the absence of documented thrombus.[13]

Anesthesia considerations of hypereosinophilic disorders

Anesthesiologists should be aware of hypereosinophilia because eosinophils mediate release of vasoactive cytokines which act on multiple organs systems with variable damage.

These patients are at elevated risk of developing life-threatening bronchospasm and acute respiratory distress syndrome following GA. Adequate depth of anesthesia must be maintained intraoperatively. Drugs that lead to histamine release for example, morphine, atracurium and cisatracurium, should be used with caution. Resuscitation drugs for bronchospasm and anaphylaxis should be readily available. In patients with acute asthmatic exacerbation or with signs of active respiratory infection, defer of elective surgery should be considered. Anesthetic agents that have bronchodilatory properties should be favored and desflurane should be avoided due to risks of precipitating bronchospasms. Neostigmine should be avoided as a reversal agent due to risks of increasing cholinergic tone triggering bronchoconstriction. Sugammadex should be used for reversal of rocuronium or vecuronium.

Cardiovascular collapse and thromboembolic events are potential complications, particularly in those with known cardiovascular or neurological involvement. Preoperative anesthetic assessment of each susceptible organ system is essential to assess for disease severity and the degree of organ damage. Baseline CXR, pulmonary function test, neurologic examination and cardiac functional assessment by ECG and TTE should be done for all HES patients.

In our patient, since he has new onset symptoms of chest pain with profuse sweating despite CT thorax showing a static Stanford type B aortic intramural hematoma. It is well justified to have thorough cardiac investigations to rule out underlying eosinophilic myocarditis.[14] Assessment of myocardial function such as TTE should be performed, particularly to look for any formation of mural thrombi affecting both ventricles. Although uncommon, myocardial infarction can occur due to embolic events from thrombus in left ventricular outflow tract. Heart failure symptoms such as shortness of breath, reduced exercise tolerance, ankle edema, paroxysmal nocturnal dyspnea, should be actively seeked for and assessed.

In patients with eosinophilic myocarditis, eosinophils infiltrate the myocardium and can progress through stages of necrosis, thrombosis and fibrosis, leading to heart failure, arrhythmias and sudden mortality. In patients with heart failure, myocardial suppressants should be avoided and invasive monitoring such as invasive arterial line and central venous line should be considered. Inotropic medications should be readily available. Patients with intracardiac thrombus on anticoagulation, perioperative anticoagulation plan would require cardiology and hematology inputs. Intraoperative continuous neurological and perfusion trend monitoring by near-infrared spectroscopy (NIRS) and heightened vigilance for perioperative stroke should be exercised.

In this case, we had a joint discussion with the patient and surgeons regarding the mode of anesthesia of monitored anesthetic care (MAC). MAC had the advantage of avoiding the use of histamine releasing drugs, avoiding the need of invasive mechanical ventilation, reducing the risks of respiratory complications like bronchospasm and was readily able to assess the patient's neurological status intraoperatively with conscious sedation.

For those who are on long term corticosteroid therapy, perioperative stress dose steroid would be detrimental to prevent the catastrophic cardiovascular collapse due to adrenal insufficiency. Interleukin-5 directed therapy such as mepolizumab should be continued perioperatively. Strict aseptic techniques should be excised in patients taking immunosuppressants.

Perioperative anti-coagulation may be considered in those with established or elevated risks of thrombosis. In cases where regional anesthesia was suitable, it should be considered as part of the anesthetic plan or as sole mode of anesthesia.

CONCLUSION

Hypereosinophilic syndrome is a rare medical disease with multiorgan manifestations and complications. Further research, guidelines and case reports would be required to guide anesthesia perioperative management. In this case, we demonstrated a safe approach to provide monitored anesthetic care for a major thoracic endovascular surgery in a patient with hypereosinophilic syndrome.

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