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Case Report

Anaphylaxis in a Patient Undergoing FESS for Nasal Polyposis: Revisting Samter's Triad

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ABSTRACT

Patients with nasal polyposis frequently have associated bronchial asthma and hypersensitivity to Non-steroidal anti-inflammatory drugs (NSAIDs). When the three conditions co-exist, it is referred to as the Samter's triad. Patients with Samter's triad are an important subset of those with aspirin-exacerbated respiratory disease (AERD). We present a case of a young female patient undergoing endoscopic sinus surgery for nasal polyps, who although did not show any other features of AERD, went on to develop florid anaphylaxis to diclofenac administration intra-operatively. After adequate resuscitation and intensive care stay, the patient made a complete recovery. NSAIDs must be avoided in patients with nasal polyps, despite showing no other features of this syndrome. Other analgesic agents that can be used include IV paracetamol and opioids like tramadol.

Nasal polyposis is a common manifestation of allergic rhinitis and frequently requires endoscopic sinus surgery (FESS). Patients with nasal polyposis present with a unique set of challenges for the anaesthetist. Although the precise pathogenesis of polyp formation is poorly defined, many clinical associations have been found between nasal polyps, asthma, eosinophilic inflammation of airways and nonsteroidal anti-inflammatory drugs (NSAIDs) hypersensitivity [1-2].

The association of nasal polyps, asthma and aspirin hypersensitivity was first described by Widal et al in 1922 [3]. Samter and Beer popularized the clinical syndrome of nasal polyposis, asthma and aspirin hypersensitivity [4]. This syndrome has been termed "Syndrome de Widal" or "Samter's Triad".

Hypersensitivity to NSAIDs is amongst the most common drug hypersensitivities. It may affect 1-2% of general population and manifest with a whole variety of symptoms involving skin (rash, urticaria and angioedema), respiratory tract (rhinorrhea, nasal congestion and bronchospasm), and in some patients, systemic anaphylaxis may develop [2,5-6]. Among patients with asthma and/or CRS with nasal polyps, the prevalence of NSAIDs hypersensitivity is significantly higher, reaching around 26% [7].

We describe a case of diclofenac anaphylaxis and subsequent management in a patient undergoing FESS for nasal polyps, without any other features of AERD or Samter's triad.

Case Report

A 25-year-old female patient presented to ENT with multiple bilateral nasal polyps and was posted for FESS. The pre-anaesthetic check up did not reveal any medical issues or any drug allergies. The patient was accepted for surgery under ASA 1 and a written consent for general anaesthesia taken. No specific pre-medication was ordered.

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On the day of surgery, she was taken to theatre and all routine monitoring were attached. An 18G intravenous (IV) cannula was secured and ringer lactate started. Anaesthesia was induced with inj. midazolam 2mg, inj. pentazocine 30mg and inj. propofol 150mg, and trachea was intubated with size 7.5mm cuffed oral-tracheal PVC tube after administering inj. vecuronium 6mg. A throat pack was then inserted and a 15-degree head up position established. Anaesthesia was maintained with oxygen, nitrous oxide and isoflurane. Intraoperative monitoring included ECG, NIBP, pulse oximetry, end-tidal CO2, airway pressures and gas analysis.

After around 1 hour of surgery, inj. diclofenac 75mg was administered by diluting with 100ml normal saline, during which the patient suddenly became increasingly tachycardic (HR 140-160/minute) and hypotensive (systolic BP 50mmHg). This was followed by elevated airway pressures, severe bronchospasm on chest auscultation and inability to ventilate. All this culminated in falling oxygen saturation levels despite 100% oxygen. We immediately stopped diclofenac infusion, requested the surgeons to stop the surgery and pack the nose, checked for tube position and patency and called for back up. A provisional diagnosis of anaphylactic reaction was made and inj. adrenaline 50mcg (0.5ml of 1:10000) was administered IV. This was repeated once, after 5 minutes. Inj. hydrocortisone 200mg IV was given, and salbutamol puffs were administered down the endotracheal tube. Another 18G IV cannula was inserted and ringer lactate boluses were infused. An arterial blood gas was drawn at this time, which showed isolated hypoxaemia, but no acid-base disturbance. After about 20 minutes of resuscitation, the patient vitals stabilized (HR 95/min, BP 106/56, SpO2 97% on FiO2 0.5).

The surgeons quickly completed the procedure. As the patient remained haemodynamically stable at the end of the surgery, we decided to give her a trial of extubation. Muscle relaxation was reversed with neostigmine and glycopyrrolate and inj. ondansetron 6mg was given. Trachea was extubated after adequate respiratory efforts and eye opening on command. The patient was kept under observation in the recovery area for 2 hours and the shifted to intensive care for overnight observation. She was prescribed oxygen therapy and salbutamol nebulization and was declared allergic to NSAIDs. The patient had an uneventful ICU stay and was shifted to the ward the next day after being informed about the incident and advising a formal intradermal testing.

Discussion

Rozsasi A et al [8], in 2006, conducted a retrospective study on ENT cases from 5 years. They found that although intraoperative bronchospasm was more common in paranasal sinus surgeries, these were not specific to intraoperative use of NSAIDs. This study however, only takes into account bronchospasm and not full-blown anaphylaxis with haemodynamic compromise and respiratory features. We feel that our case report is important because there is a paucity of literature describing anaphylaxis in a patient with nasal polyps, without other obvious features of AERD.

The clinical syndrome of AERD comprises of chronic rhinosinusitis (CRS), nasal polyps, bronchoconstriction in asthmatics, and/or eosinophil inflammation in the upper and lower airways, urticaria, angioedema, and anaphylaxis following the ingestion of NSAIDs blocking the COX-1 enzyme [1]. In this case, NSAIDs are an exacerbating factor rather than a feature of the syndrome. Patients with Samter's triad are an important and challenging subset of those having AERD or aspirin-exacerbated respiratory disease (also called NERD or NSAIDs-exacerbated respiratory disease).

AERD has been estimated to affect 0.3 to 2.5 % of the general population [9]. The symptoms of this syndrome usually start with rhinitis in the third decade of life, along with nasal congestion, hyposmia, chronic rhinorrhea and are usually followed by nasal polyps. Nasal polyps usually follow an aggressive course in the disease, sometimes resulting in facial deformities like midfacial expansion [10].

The diagnosis of AERD presents a major challenge and is usually based on the clinical picture. This may be supported by imaging studies such as computed tomography or endoscopy. Some patients have a definitive history of aspirin sensitivity, but in others an aspirin challenge or a provocation test may need to be performed. Aspirin provocation test can be performed via oral, bronchial inhalation, nasal inhalation or intravenous routes. This test bears the risk of severe exacerbation of asthma and can be life threatening, hence, must be performed by trained personnel in a specialized facility.

The molecular pathogenesis of AERD has never been fully understood, but several theories have been put forward. It may either involve an alteration in arachidonic acid pathway and its receptors/enzymes, or a release of inflammatory mediators and cytokines. Another theory revolves around involvement of microbes [1].

The treatment of AERD depends on the stage and extent of the disease. The various management strategies include:

Aspirin Desensitization: Aspirin desensitization with a maintenance dose of 100 mg daily has a positive impact on nasal polyp relapse and seems to be a safe and suitable therapy to improve clinical complaints and the quality of life of individuals with AERD [11].

Leukotriene Modifier Drugs: These drugs have an established impact in the treatment of asthma and allergic rhinitis. There are two classes of drugs under this group-the cysteinyl leukotriene 1 receptor antagonists (montelukast, zafirlukast) and 5-lipooxygenase inhibitor

(zileuton). These drugs however, do not affect the incidence or prognosis of nasal polyps [12].

Surgery: Sinus surgery is viewed as an adjunct to medical therapy and may also have to be performed when all medical therapy fails. FESS has been reported to improve asthma severity and quality of life in patients presenting with rhinosinusitis with nasal polyposis [13].

Low salicylate diet: The low-salicylate diet may offer a novel treatment adjunct to the current management of AERD. Clinically and statistically significant improvements on both subjective and objective outcome measures were noted for the upper and lower respiratory tracts [14].

Future Attempts: Many future therapies that are in the pipeline include Anti-IgE and anti-IL-5 antibodies and antibodies against Staphylococcus aureus endotoxin [1].

Conclusion

To conclude, AERD is a distinct clinical syndrome characterized by chronic rhinosinusitis, nasal polyposis, asthma, eosinophil inflammation of upper and lower airways and hypersensitivity to NSAIDs. A subset of these patients has Samter's Triad, which is characterized by asthma, nasal polyps and NSAIDs hypersensitivity. Patients undergoing endoscopic sinus surgery for nasal polyps must be assumed to have AERD, even though they show no other features. NSAIDs must be avoided in these patients as they can lead to serious hypersensitivity or anaphylactic reactions and hence, we have established similar guidelines in our department.

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