



**International Journal of Medical and Pharmaceutical
Case Reports**

6(5): 1-5, 2016; Article no.IJMPCR.24677
ISSN: 2394-109X, NLM ID: 101648033



SCIENCEDOMAIN *international*
www.sciencedomain.org

Samter's Triad: Rare or Under-Diagnosed?

Adekunle Olatayo Adeoti^{1*}, Oluwatoba Ezekiel Osasona¹,
Olatunji Bukola Olaoye¹ and Olufemi O. Desalu²

¹Department of Medicine, Ekiti State University Teaching Hospital, Ado-Ekiti, Ekiti State, Nigeria.

²Department of Medicine, University of Ilorin Teaching Hospital, Ilorin, Kwara State, Nigeria.

Authors' contributions

This work was carried out in collaboration between all authors. Author AOA wrote the draft of the manuscript, supervised the work and contributed to the correction of the draft. Author OEO designed the figures and managed the literature searches. Author OBO provided the case and contributed to the correction of the draft. Author OOD managed the literature searches and contributed to the correction of the draft. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJMPCR/2016/24677

Editor(s):

(1) Rafik Karaman, Bioorganic Chemistry, College of Pharmacy, Al-Quds University, Jerusalem, Palestine.

Reviewers:

(1) William Grant, SUNY Upstate Medical University, USA.

(2) Denise Rossato Silva, Federal University of Rio Grande do Sul, Brazil.

Complete Peer review History: <http://sciencedomain.org/review-history/13435>

Case Study

Received 29th January 2016
Accepted 11th February 2016
Published 25th February 2016

ABSTRACT

Samter's triad is an acquired inflammatory disorder characterized by asthma, nasal polyps and aspirin sensitivity. Aspirin sensitivity is the hallmark of this condition. It is a form of pseudo-allergy, as IgE does not play any significant role in its pathophysiology. There is a paucity of reported cases of Samter's triad in Africa.

Our patient is a 24-year old female who presented with worsening of asthmatic symptoms and naso-ocular complaints after thirty minutes of intake of non-steroidal anti-inflammatory drugs (NSAIDs) for dysmenorrhea. We found bilateral nasal polyps on examination of her nostrils and two previous episodes of similar reactions to NSAIDs.

There is a need for increased awareness about this condition among healthcare givers managing patients with bronchial asthma as several cases may be missed in hospital settings. Samter's triad requires detailed drug history of sensitivity to NSAIDs with accompanying naso-ocular manifestations in all asthmatic patients.

*Corresponding author: E-mail: Kadeoti2002@yahoo.com;

Keywords: Samter's triad; aspirin-exacerbated respiratory disease; bronchial asthma; nasal polyp.

1. INTRODUCTION

Samter's triad is a clinical condition that comprises of asthma, nasal polyps and aspirin sensitivity. Aspirin-exacerbated respiratory disease, aspirin-intolerant asthma and aspirin-induced asthma are other names used to describe this condition. It involves reactions to aspirin and other non-steroidal anti-inflammatory drugs with worsening asthma symptoms and naso-ocular manifestations which typically occurs within 30 to 120 minutes of ingestion of the drug [1-3].

It is a pseudo-allergic reaction, as it is not mediated by immunoglobulin E (IgE). This aspirin-induced exacerbation is seen in 2-23% of patients with asthma [4]. Samter's triad is mainly a clinical diagnosis, however, the definitive diagnosis could be established by aspirin challenge test which is rarely necessary for clinical practice [2,5]. The pathophysiology of this condition, though unclear, is said to be due to the overwhelming production of pro-inflammatory arachidonic acid products especially the cysteinyl-leukotrienes leading to chronic airway inflammation. In addition, aspirin blocks the cyclooxygenase-1(COX-1) enzyme promoting dysregulation of the arachidonic acid metabolism and favouring the lipo-oxygenase pathway. Although Samter's triad is believed to be a metabolic disorder, there were convincing evidences that a genetic predisposition to functional polymorphism of cys-leukotriene C4 synthase may be present in some individuals with the triad [2,6].

To the best of our knowledge, there are no reported cases in Africa in the literature. Despite its classic features and well-defined parameters, Samter's triad is often under-diagnosed. The index case was identified after three previous episodes of similar reactions to three different NSAIDs with the classical features of Samter's triad which were missed by the attending General practitioners.

2. CASE REPORT

A 24-year old female was referred from the accident and emergency to the respiratory clinic (follow-up) following treatment for an acute severe asthma that developed 30 minutes after the ingestion of aspirin. She presented at the emergency room with cough, breathlessness,

wheeze and chest tightness. The patient was managed with nebulized salbutamol 5 mg repeated every fifteen minutes for the first hour, 40-60% intranasal oxygen and hydrocortisone 200mg iv given 4 hourly for 24 hours. She was subsequently discharged to the respiratory clinic for follow up after being stable on discharge medications for 24 hours.

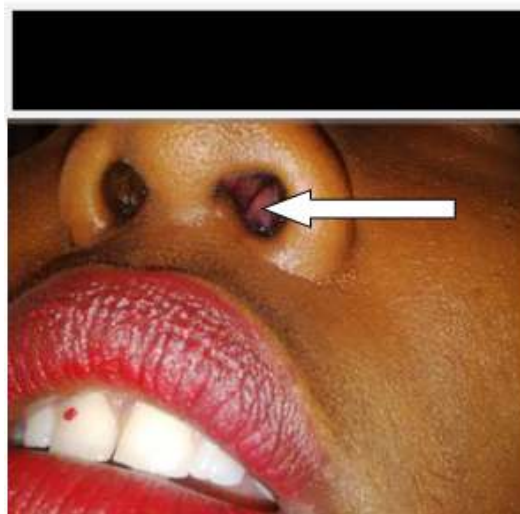


Fig. 1. Shows the nasal polyps indicated with the white arrow

At review, she complained of recurrent sneezing, nasal congestion, nasal discharge, hyposmia and conjunctival tearing with accompanying urticarial rashes mainly on the flexural surfaces of the forearm and neck region. Previous episodes of similar reactions had occurred following ingestion of ibuprofen and naproxen for dysmenorrhea during her menstrual cycle. Such episodes were managed as acute severe asthma with no emphasis placed on the drug history neither were her nostrils inspected. There is family history of allergy although none had similar reactions to NSAIDs. History of recurrent nasal discharge started in adolescence and it preceded the onset of bronchial asthma.

Her cardiovascular and chest examinations were normal. However, bilateral nasal polyps measuring about 0.5 cm by 1 cm were noticed. They were hyperemic with no ulceration and no visible bleeding observed. The patient had no difficulty with breathing and had no sensation of a mass in her nostrils. Chest X-Ray and lung function test were both normal.

Table 1. Shows the spirometric indices of the patient

	Measured	Predicted	% predicted
FEV1 (L)	3.20	2.86	112
FVC (L)	3.66	3.28	112
FEV1/FVC	87.4	84.4	104
PEF (L/min)	7.40	7.22	102

**Fig. 2. Shows the x-ray of the paranasal sinuses (Anteroposterior and lateral view)**

An assessment of Samter's triad was made and she was placed on inhaled salbutamol 200 mcg PRN, oral prednisolone 30 mg daily for 7 days and intranasal fluticasone 50mcg in each nostril once daily. The patient was counseled about the nature of the illness, strongly advised against NSAIDs use and referred to see the Otorhinolaryngologist. Following two weeks of use of the above medications, the nasal polyps regressed significantly as the X-ray of the paranasal sinuses showed well aerated sinuses with left maxillary antral mucosa thicken and enlarged turbinates. The patient continued the use of intranasal fluticasone for another two weeks.

3. DISCUSSION

Our index patient being female in her third decade supports the preponderance of Samter's triad in this gender and age group [2,7]. Similarly, history of recurrent nasal discharge preceded asthma symptoms and nasal polyposis which was in agreement with a previous study that suggested a natural history of progression from the initial chronic upper airway inflammation manifested as rhinosinusitis which later progressed to lower airway inflammation over the years with asthma symptoms and then the

emergence of nasal polyps. This upper and lower respiratory airway link is proposed to be due to the effects of nasobronchial reflex; effects of nasal breathing on the lungs; and the similarity in pathobiological responses such as eosinophilia [8].

In addition to the bilateral nasal polyps, nasocular symptoms and hyposmia seen in the index case are common findings in patients with Samter's triad which might be due to the chronic inflammatory process and a conductive deficit of smell from the obstruction of the olfactory epithelium in the nostrils [2]. Other extrabronchial symptom aside excessive lacrimation and redness of the eye were the cutaneous findings of urticarial rash seen in our patient. These features usually accompany the typical triad in patients with Samter's [3,5].

Furthermore, NSAIDs with dominant preferential activity towards COX-1 like indomethacin, naproxen, diclofenac, or ibuprofen are known to precipitate severe adverse symptoms in a significant proportion of aspirin-sensitive asthmatic patients. This cross-reactivity was observed in our patient who, on different occasions, reacted to three NSAIDs (aspirin, ibuprofen and naproxen). Selective COX-2

inhibitors and NSAIDs with weaker inhibitory potency towards prostaglandin synthase are alternatives that are well tolerated by such patients [3].

Premenstrual asthma is a very close differential diagnosis in our patient as her symptoms occurred during the menstrual cycle. Contrary to the typical presentation in patients with premenstrual asthma who experience peak worsening of asthma symptoms about 2-3 days before menstruation starts, our patient had symptoms during her cycle which was precipitated by the ingestion of NSAIDs. This period in patients with premenstrual asthma correlates with the late luteal phase of the ovarian cycle when oestrogen and progesterone are at their lowest [9].

Essentially, the chest examination and spirometry were normal as the patient was referred in a stable state after the initial emergency care. Aspirin challenge and bronchoprovocation methacholine challenge tests which are done in well-equipped centers may be needed to confirm the diagnosis, however, it must be emphasized that Samter's triad is essentially a clinical diagnosis [5]. Both medical and surgical treatment options have been shown to be useful. However, steroid use has been found to have a significant effect on disease manifestation as it inhibits the formation of arachidonic acid by blocking phospholipase enzyme [10]. Other treatment options include avoidance of NSAIDs which blocks COX-1 receptors, aspirin desensitization, use of leukotriene receptor antagonist, 5-lipoxygenase inhibitors, inhaled PGE2, antihistamines and surgical treatment mainly functional endoscopic sinus surgery [3].

4. CONCLUSION

Its recognition and appropriate management are crucial to effectively reduce morbidity and improve outcome as patients with Samter's triad generally tend to have more severe symptoms of asthma and nasal polyposis. Samter's triad is under-reported hence, there is a need for renewed interest in improving our diagnosis of this condition in our environment.

CONSENT

All authors declare that written informed consent was obtained from the patient for publication of this paper and accompanying images.

ETHICAL APPROVAL

All authors hereby declare that the study was approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Douglas GC, Karkos PD, Swift AC. Aspirin sensitivity and the nose. *Br J Hosp Med (Lond)*. 2010;71(8):442-5.
2. Spies JW, Valera FPC, Cordeiro DL, de Mendonça TN, Leite MGJ, Tamashiro E, et al. The role of aspirin desensitization in patients with aspirin-exacerbated respiratory disease (AERD). *Braz J Otorhinolaryngol*; 2015. pii: S1808-8694(15)00158-5. DOI: 10.1016.
3. Kowalski M, Makowska J, Blanca M, Bavbek S, Bochenek G, Bousquet J, et al. Hypersensitivity to nonsteroidal anti-inflammatory drugs (NSAIDs)—classification, diagnosis and management: review of the EAACI/ENDA# and GA2LEN/HANNA*. *Allergy*. 2011;66(7):818-29.
4. Obase Y, Matsuse H, Shimoda T, Haahtela T, Kohno S. Pathogenesis and management of aspirin-intolerant asthma. *Treat Respir Med*. 2005;4(5):325-36.
5. Zeitz HJ. Bronchial asthma, nasal polyps, and aspirin sensitivity: Samter's syndrome. *Clin Chest Med*. 1988;9(4):567-76.
6. Laidlaw TM, Boyce JA. Pathogenesis of aspirin-exacerbated respiratory disease and reactions. *Immunology and allergy clinics of North America*. 2013;33(2): 195-210.
7. Berges-Gimeno MP, Simon RA, Stevenson DD. The natural history and clinical characteristics of aspirin-exacerbated respiratory disease. *Annals of Allergy, Asthma & Immunology*. 2002;89(5):474-8.
8. Ferguson B, Powell-Davis A. The link between upper and lower respiratory disease. *Current Opinion in Otolaryngology & Head and Neck Surgery*. 2003;11(3): 192-5.

9. Partridge MR. Asthma: Clinical features, diagnosis and treatment. In: Albert RK, Spiro SG, Jett JR editors. Clinical respiratory medicine. 3rd ed. Philadelphia: Mosby Elsevier; 2008.
10. Mrówka-Kata K, Czecior E, Kata D, Namysłowski G, Dziechciarz-Werbowska J, Sowa P. Current view on nasal polyps management in Samter's triad patients. Otolaryngologia Polska. 2012;66(6):373-8.

© 2016 Adeoti et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
<http://sciencedomain.org/review-history/13435>