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doi:10.3748/wjg.14.7141

World J Gastroenterol 2008 December 14; 14(46): 7141-7142
World Journal of Gastroenterology ISSN 1007-9327

Mesalamine hypersensitivity and Kounis syndrome in a pediatric ulcerative colitis patient

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Received: October 2, 2008 Revised: November 1, 2008
Accepted: November 8, 2008
Published online: December 14, 2008

Abstract

5-aminosalicylic acid (mesalamine) rarely induces hypersensitivity reactions. If chest pain associated with atypical electrocardiographic changes are seen during its administration, one should always bear in mind type I variant of Kounis syndrome. This variant includes patients, of any age, with normal coronary arteries, without predisposing factors for coronary artery disease, in whom the acute release of inflammatory mediators from mast cells can induce either sudden coronary artery narrowing, without increase of cardiac enzymes and troponins, or coronary artery spasm that progresses to acute myocardial infarction, with elevated cardiac enzymes and troponins.

Key words: Drug hypersensitivity; Kounis syndrome; Mesalamine; Salicylates

Peer reviewer: Hitoshi Asakura, Director, Emeritus Professor, International Medical Information Center, Shinanomachi Renga Bldg.35, Shinanomachi, Shinjukuku, Tokyo 160-0016, Japan

Kounis GN, Kouni SA, Hahalis G, Kounis NG. Mesalamine hypersensitivity and Kounis syndrome in a pediatric ulcerative colitis patient. *World J Gastroenterol* 2008; 14(46): 7141-7142 Available from: URL: http://www.wjgnet.com/1007-9327/14/7141.asp DOI: http://dx.doi.org/10.3748/wjg.14.7141

TO THE EDITOR

We have read with interest the article published recently in this journal[1] concerning a 12-year-old boy who developed severe chest pain after oral administration of 5-aminosalicylic acid (5-ASA, mesalamine). Electrocardiographic changes during the pain were not specific but inverted T-waves were present in the lateral chest leads. Additionally, cardiac enzymes, troponins, C-reactive protein and brain natriuretic peptide were normal. The authors concluded that this patient had suffered a rare hypersensitivity reaction to mesalamine.

It is known that mesalamine can, rarely, induce hypersensitivity reactions such as hypersensitivity myocarditis, hypersensitivity pneumonitis, angioedema, pericarditis, erythroderma, toxic epidermal necrolysis, palmar-plantar erythrodysesthesia, skin rashes and hypereosinophilia.

This case seems to be a characteristic case of type I variant of Kounis syndrome[2,3], the youngest reported so far. Another case of Kounis syndrome secondary to amoxicillin/clavulanic acid use has been recently reported in a 13-year-old child[4]. On the other hand, salicylate products such as aspirin are known to induce hypersensitivity reactions, and aspirin has been reported to induce Kounis syndrome[5].

Pathophysiologically, symptoms of salicylate hypersensitivity can be explained by over-production of leukotriene metabolites, since salicylate allergic patients who have come in contact with salicylates show a marked inhibition of cyclooxygenase (COX), which is continuously expressed in the human body. This leads to a diminished production of COX products such as prostacyclin and thromboxane, and can accelerate the metabolism of arachidonic acid towards lipoxygenase products such as leukotrienes. It is known that leukotrienes are powerful coronary arterial vasoconstrictors, and their biosynthesis is enhanced in the acute phase of unstable angina[2]. Atay *et al*[1] have correctly stated that arachidonic acid metabolites generated through both COX and lipoxygenase pathways are thought to be increased in patients with ulcerative colitis. Ideally, in this case, allergic screening with measurement of mast cell mediators such as histamine and arachidonic acid metabolites should have been carried out. However, this study shows that Kounis syndrome is not a very rare disease but a 'very rarely diagnosed' disease.

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