



This article appeared in a journal published by Elsevier. The attached copy is furnished to the author for internal non-commercial research and education use, including for instruction at the authors institution and sharing with colleagues.

Other uses, including reproduction and distribution, or selling or licensing copies, or posting to personal, institutional or third party websites are prohibited.

In most cases authors are permitted to post their version of the article (e.g. in Word or Tex form) to their personal website or institutional repository. Authors requiring further information regarding Elsevier's archiving and manuscript policies are encouraged to visit:

<http://www.elsevier.com/copyright>

'Lone' atrial fibrillation precipitated by monosodium glutamate and aspartame

Craig G. Burkhardt*

University of Toledo College of Medicine, 5600 Monroe Street, Suite 106B, Sylvania, Ohio 43560, United States

Received 13 January 2009; accepted 15 January 2009

Available online 10 February 2009

Keywords: 'Lone' atrial fibrillation; Atrial fibrillation; Monosodium glutamate; Aspartame; Aspartate; Excitotoxins

'Lone' atrial fibrillation infers absence of hypertension, heart failure, and coronary artery disease as the cause of this tachycardia. Korantzopoulos et al. [1] recently reviewed underlying causes and links associated with this entity, but failed to mention two epidemiological causes that receive considerable press, namely a flavor enhancer, monosodium glutamate (MSG) and an artificial sweetener, aspartame as the culprit of lone atrial fibrillation.

As an aside, Donald Rumsfeld was most significant in the release of aspartame. He was the Chief Executive Officer, President, and Chairman of G.D. Searle & Company, a worldwide pharmaceutical company which sought approval of aspartame for human usage. To be exact, on the second day of Reagan's first term in 1980, he appointed Dr. Arthur Hull Hayes as FDA Commissioner because no FDA Commissioner in 16 years would approve aspartame. Dr. Hayes immediately over-ruled the resistance of the FDA's own scientific panel, the Board of Inquiry, and then Dr. Hayes proceeded to leave the FDA and work for Searle himself.

1. Case report

A healthy 57-year old physician with a normal cardiac exam and echocardiogram, had a four month history of persistent atrial flutter. This was resolved with catheter ablation in 2007 leading to a temporary return to sinus rhyme. Over the last several months he developed paroxysmal atrial fibrillation which was confirmed by outpatient monitoring. Patient was placed on beta blockers and was being considered for a second ablation.

In the interim, the patient eliminated monosodium glutamate and all artificial sweeteners from his diet. Surprisingly, he experienced elimination of occurrences of arrhythmia. To test the validity of this dietary elimination, he performed three separate patient challenges with Chinese food (with MSG),

three glasses of Crystal light (aspartame), and a bag of beef jerky (MSG). All of these challenges resulted in development of atrial fibrillation within a few hours.

2. Discussion

The reaction to MSG and aspartame is to a derivative of the amino acid, respectfully, glutamate and aspartate. Aspartame, for example, releases aspartate during digestion. Free glutamate can occur in food as a consequence of manufacturing. MSG-sensitive persons do not react to protein in which there is bound glutamate and/or bound aspartate, nor are they affected by minute amounts of free glutamic and aspartic acids that might be found in unadulterated, unfermented food.

Both glutamate and aspartate are known neurotransmitters in the brain and central nervous system. Any central nervous system effects appear to occur due to the excessive levels of neurotransmitters which develop in a short time period. Normally glutamate and aspartate are bound within proteins and broken by enzymes in the process of digestion. As this process is gradual, controlled levels of free glutamate and aspartate are achieved in the body. When these amino acids are eaten in the free form with no peptide linkages, glutamate and aspartate enter the bloodstream quickly causing much higher concentration levels of these amino acids. MSG and aspartame have been associated with numerous symptoms including headaches, dizziness, seizures, nausea, numbness, muscle spasms, fatigue, heart palpitations, anxiety attacks, vertigo, and memory loss.

Of note, the brain is not the only tissue having glutamate receptors. Numerous glutamate receptors have been found both within the skin epidermis, heart's electrical conduction system, and the heart itself [2–4]. Thus, MSG and aspartame are both excitotoxins of cardiac tissue as well. The AFIB Report found that 10% of patients with atrial fibrillation found MSG and 4% listed aspartame as triggers for their attacks. In a study of 200 cardiac patients, 16% experienced detectable changes in their heart rate or rhythm after consuming aspartame [5].

* Tel.: +1 419 885 3403; fax: +419 885 3401.

E-mail address: cgbakb@aol.com.

In short, this case report merely adds more credence that MSG and aspartame elimination diets may be beneficial for some patients with atrial fibrillation in clinical practice.

Acknowledgement

The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [6].

References

- [1] Korantzopoulos P, Liu T, Milionis HJ, Li G, Goudevenos JA. 'Lone' atrial fibrillation: hunting for the underlying causes and links. *Int J Cardiol* 2009;131:180–5.
- [2] Morhenn VB, Waleh NS, Mansbridge JN. Evidence for an MND A receptor subunit in human keratinocyte and rat cardiocytes. *Eur J Pharmacol* 1994;268:409–14.
- [3] Morhenn VB, Murakami M, O'Grady T. Characterization of the expression and function of N-methyl-D-aspartate receptor in keratinocytes. *Exp Dermatol* 2004;13(50):5–11.
- [4] Gill SS, Veinot J, Kavanagh M, Pulido O. Human heart glutamate receptors — implications for toxicology, food safety, and discovery. *Toxicol Pathol* 2007;35:411–7.
- [5] Roberts HJ. Aspartame Disease: An Ignored Epidemic. Waterloo, Ontario: Sunshine Sentinel Press; 2003.
- [6] Coats AJ. Ethical authorship and publishing. *Int J Cardiol* 2009;131:149–50.

0167-5273/\$ - see front matter © 2009 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.ijcard.2009.01.028

Coronary slow flow: Description of a new “cardiac Y” syndrome

Massimo Fineschi^{a,*}, Tommaso Gori^b

^a *U.O. Emodinamica, University Hospital of Siena, Italy*

^b *Department of Cardiology, Uniklinikum Mainz, Germany*

Received 15 March 2008; accepted 29 May 2008

Available online 13 August 2008

Abstract

Angina in the presence of intact coronary arteries remains a relatively frequent clinical issue. It is now well understood that an impaired capacity to modulate coronary vascular resistances upon exercise (i.e. an impaired coronary flow reserve) may lead to ischemia and angina. We describe a case of coronary slow flow, i.e. a condition in which resting (micro) vascular resistances are inappropriately high, causing unstable angina. We propose that this novel syndrome should be called coronary syndrome Y to distinguish it from the better understood coronary syndrome X.

© 2008 Elsevier Ireland Ltd. All rights reserved.

Keywords: Coronary slow flow; Coronary microvasculature; Angiography

A 71-year old male was admitted for angina that had occurred mostly at rest, at near-daily intervals, for approximately two weeks. The patient's history included a prior (one year) lateral myocardial infarction treated with primary percutaneous intervention and bare metal stent placement. He was a smoker, but reported no history of diabetes, dyslipidemia, hypertension or other cardiovascular history. He had been taking acetylsalicylic acid 100 mg/day and a statin for one

year. Upon admission, transthoracic echocardiography showed normal size of all heart chambers, normal wall thickness and normal global ejection fraction with mild hypokinesia of the lateral wall. Heart rate (65 beats/min), blood pressure (107/65 mmHg) and serum cardiac biomarkers were normal. His ECG (in the absence of pain) was unremarkable.

Coronary angiography showed patency of the stent implanted in a marginal branch of the circumflex coronary; a moderate (40%) lesion was present in the mid left anterior descending coronary. A remarkable delay in the opacification of both the left (corrected TIMI frame count = 56, normal value = 21 ± 2 [1]) and the right coronary (TIMI frame

* Corresponding author. Department of Cardiology, University Hospital of Siena, Italy.

E-mail addresses: orfine@tin.it (M.Fineschi), tomgori@hotmail.com (T. Gori).