

Hepatitis C and urticaria: cause and effect?

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Key words: chronic urticaria; hepatitis C infection.

Urticaria is a common disorder that affects as many as 20% of the population at some point during their lifetime. Factors believed to cause or aggravate urticaria include drugs, foods, additives, connective tissue disorders and infections. It is well established that hepatitis B virus causes urticaria (1). Whether hepatitis C infection causes urticaria or not is still debated. There have been conflicting reports both in favour of (2,3) and against (4,5) hepatitis C virus (HCV) causing urticaria. Kanazawa and colleagues (2) reported a positive relationship between hepatitis C and urticaria. Nineteen of 79 patients tested were found to be positive for HCV. Subsequent studies however did not confirm such an association (4,5).

We describe here a case of chronic intermittent urticaria caused by HCV.

A 49-year-old Caucasian man presented with a typical history of recurrent urticaria of several months duration. There were no clues in the history apart from promiscuity, and all routine screening investigations were negative apart from an approximately three times raised alanine aminotransferase (ALT). In view of a history of multiple sexual partners and raised ALT, hepatitis serology was requested. Hepatitis C antibody was reported to be positive by both enzyme-linked immunosorbent assay (ELISA) and microparticle enzyme immune assay.

Infective causes including viral hepatitis may be a cause of chronic urticaria.

Results of ELISA for hepatitis B were negative. We conclude that HCV infection may have caused urticaria in this patient.

There does not appear to be any clear-cut evidence to affirm or refute a direct link between chronic urticaria and HCV infection in the literature.

We suggest that HCV status should be checked in patients presenting with urticaria in areas with a high prevalence. It may not be cost-effective to perform routine screening for HCV where the prevalence of this infection is low, but should be considered where the history or laboratory tests suggest this possibility. Further studies are needed to establish any definite association, possible aetiopathogenic role and the cost implications of screening and therapy of HCV in chronic urticaria.

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References

1. VAIDA GA, GOLDMAN MA, BLOCH KJ. Testing for hepatitis B virus in patients with chronic urticaria and angioedema. *J Allergy Clin Immunol* 1983;**72**:193-198.
2. REICHEL M, MAURO TM. Urticaria and hepatitis C. *Lancet* 1990;**336**:822-823.
3. KANAZAWA K, YAOITA H, TSUDA F, OKAMOTO H. Hepatitis C virus infection in patients with urticaria. *J Am Acad Dermatol* 1996;**35**:195-198.
4. SMITH R, CAUL EO, BURTON JL. Urticaria and hepatitis C. *Br J Dermatol* 1997;**136**:980.
5. LLANOS F, RAISON-PEYRON N, MEUNIER L, DUCOS J, MEYNADIER J. Hepatitis C virus infection in patients with urticaria. *J Am Acad Dermatol* 1998;**38**:646.

Fatal allergy as a possible consequence of long-term elimination diet

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Key words: anaphylaxis; elimination diet; loss of tolerance.

We report the case of an adolescent girl died from anaphylaxis to milk proteins presumably due to a loss of tolerance after an exclusion diet.

At 2 months of life she presented with eczema and, from age 3, developed asthma showing positive prick test results for milk, egg and Dermatophagoides. She had never been on a restriction diet and eczema and asthma were

A girl with a previously asymptomatic sensitization to milk presented a fatal loss of tolerance after an exclusion diet.

controlled by periodic topical steroid treatment. A subcutaneous immunotherapy with a house dust mite extract was performed from age 6 to 11 with good asthma control.

At the age 11 she referred to a physician for the persistence of moderate eczema. So far she had neither experienced a systemic reaction, nor asthma to foods. Prick test (5/22 mm) and allergen-specific IgE measurement (18 kU_A/l) were still positive for casein, and a milk-free diet was emphasized. The girl and her parents strictly complied with the dietary recommendation. Six months later the eczema had not improved and symptoms of immediate hypersensitivity (asthma, angioedema, hurticaria) appeared whenever she was exposed to milk forcing the girl to continue the diet. In the following years her allergic symptoms worsened with severe reactions, requiring adrenaline autoinjector and

admissions to the emergency department, even after exposure to minute quantities of allergen by skin contact or inhalation.

At the age of 18 she inadvertently entered a dairy shop and inhaled milk proteins experiencing a fatal reaction (asthma, horticaria, angioedema).

We believe that an inappropriate exclusion diet had caused a loss of tolerance. Some reasons do not support the possibility that this case could simply reflect the natural course of severe food hypersensitivity. The occurrence of immediate-type symptoms to previously tolerated food has been reported in adults, but usually in these patients there is no overt evidence of previous asymptomatic sensitization. In the usual pattern of allergy children might experience symptoms after few exposures. In our case the temporal link between the diet and the development of symptoms is suggestive for a causal relationship between the two. The immunologic risk of elimination diets has been reported in the literature.

We found three papers involving 13 children with documented IgE hypersensitivity who could all previously tolerate the offending food before the elimination diet induced the subsequent development of immediate type symptoms (1–3). Evidence suggests that exposure to large doses of antigen might produce a suppression of the specific IgE response, a model likely to explain oral desensitization to food (4). Therefore, the induction of a loss of tolerance by interruption of exposure to large doses of antigen is conceivable in children with preexisting subclinical sensitization. This is the first report of fatal loss of tolerance after an exclusion diet and the presence of asthma, a risk factor for severe food allergic reactions, probably played a role. Incidence and hospital admissions for acute anaphylaxis seem to increase (5). Our hypothesis is that this increase might be due, at least partially, to the widespread and uncontrolled recommendation of elimination diets.

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References

- MARTIN ESTEBAN M, PASCUAL C, FLANDOR A, OJIEDA JA. A possible consequence of long term elimination diet in IgE mediated subclinical food hypersensitivity. *Allerg Immunol (Paris)* 1988;**20**:55–56.
- LARRAMENDI CH, MARTIN ESTEBAN M, PASCUAL M, FIANDOR A, DIAZ PENA JM. Possible consequences of elimination diets in asymptomatic immediate hypersensitivity to fish. *Allergy* 1992;**47**:490–494.
- DAVID TJ. Anaphylactic shock during elimination diets for severe atopic eczema. *Arch Dis Child* 1984;**59**:983–986.
- PATRIARCA G, NUCERA E, RONCALLO C, POLLASTRINI E, BARTOLOZZI F, DE PASQUALE T et al. Oral desensitizing treatment in food allergy: clinical and immunological results. *Aliment Pharmacol Ther* 2003;**17**:459–465.
- CLARK AT, EWAN PW. Food allergy in childhood. *Arch Dis Child* 2003;**88**:79–81.

Euro coins and contact dermatitis

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Key words: contact dermatitis, Euro, nickel.

Starting from January 2002, the Euro has become the official monetary unit of the European Union and has replaced the various national currencies. It has been recently demon-

strated that the 1 and 2 euro coins release very high amounts of nickel, at variance with the 50 and 10 cent coins. This has been attributed to the peculiar bimetallic structure of the 1 and 2 euro coins (that are composed by a ring and a central plate of different cupro-nickel alloys): the bimetallic structure generates a galvanic potential that is responsible for corrosion and nickel release in contact with human sweat (1). The release of nickel has been found to exceed by about 300-fold the limits recommended by the European Union. Thus, it has been claimed that handling Euro coins can lead to an increase of the prevalence and severity of contact dermatitis caused by nickel.

We attempted to test the hypothesis that the current handling of Euro coins has increased the rate of nickel sensitization. This was performed by studying two large groups of patients referring for suspected contact dermatitis. The first group was studied before the introduction of the Euro (i.e. before January 2002), and the second group from January 2002, when the new currency was introduced.

More than 1000 consecutive outpatients suspected of contact dermatitis underwent a patch test with the European Standard Series (2). All the patients had an eczema strictly confined to both hands. Patients pierced at any body site were excluded from this analysis in order to avoid a heavy confounding factor, as it has been shown that the presence and number of piercing increases the rate of contact allergy (3). Demographic data, working habits and the history of allergic diseases were carefully recorded in all patients. In particular, the patients were screened by clinical history for the presence of one or more of the following: hayfever, allergic asthma, atopic

Sensitization to nickel did not increase after the introduction of Euro coins.

Table 1. Characteristics of the patients patch-tested before and after the introduction of euros

	Before Euro	After Euro	P (χ^2)
Patients	753	292	
Sex (M/F)	273/480	76/216	NS
Mean age	43	42.5	NS
Age range	15–81	14–82	NS
History of allergic diseases	122 (16.2%)	50 (17.1%)	NS
Patch-test nickel positivity	202 (27%)	78 (26%)	NS
Age range	15–76	14–72	NS
History of allergic diseases	39 (19.2%)	15 (19.6%)	NS