

Table 1. Airborne endotoxin concentration according to home characteristics

		Endotoxin levels			P-value*
	n	Geometric mean (ng/m ³)	Arithmetic mean (ng/m ³)	95% CI	
Type of building					
Home	47	0.133	0.124	0.039–0.208	0.206
Apartment	53	0.138	0.148	0.075–0.221	
Environmental tobacco smoke					
Smokers					
No	76	0.139	0.205	0.139–0.270	0.537
Yes	24	0.125	0.197	0.097–0.296	
Furry pets in the home					
No	48	0.142	0.194	0.120–0.268	0.538
Yes	52	0.131	0.205	0.123–0.287	
Cat only	21	0.131	0.163	0.113–0.213	0.082
Dog	20	0.094	0.12	0.074–0.166	
Cat + dog	2	0.360	0.630	–0.382 to 1.642	
Other	9	0.213	0.398	0.014–0.782	
Mechanical ventilation					
No	70	0.141	0.207	0.131–0.282	0.274
Yes	30	0.123	0.195	0.140–0.249	
Floor type					
Smooth	30	0.134	0.140	0.095–0.185	0.854
Carpeted	70	0.143	0.221	0.129–0.313	
House plants					
No	28	0.142	0.285	0.118–0.451	0.567
Yes	72	0.134	0.172	0.132–0.211	

*Nonparametric test.

effect on the onset of atopy (4). We found no correlation between the presence of furry pets and either airborne or dust endotoxin levels. This suggests that animals were not responsible for endotoxin levels in either air or dust. This is consistent with the showing by Lieutier-Colas et al. (1) that airborne endotoxin levels were not correlated with the presence of the rats in rat quarters. Michel et al. also reported that the presence of furry pets in homes did not influence the HD endotoxin levels (5). We also found no correlation between dust and airborne endotoxin levels. This suggests that it seems necessary to measure endotoxin in both dust and air to obtain a relevant assessment of exposure.

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Systemic mastocytosis associated with recurrent paroxysmal atrial fibrillation

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Key words: systemic mastocytosis; atrial fibrillation.

Systemic mastocytosis is a rare and chronic disorder characterized by diffuse mast-cell infiltration of multiple organs; it is most commonly seen in the skin, but the skeleton, bone marrow, gastrointestinal tract and central nervous system may also be involved.

The attacks symptoms, like skin involvement lasting 15–30 min associated with headache, dyspnoea, chest pain, nausea, diarrhoea, paraesthesias, lethargy, warmth, light-headedness, palpitations and tachycardia and sometimes anaphylaxis are a consequence of the release of mast cell mediators. This mediators' release may be spontaneous or as a result of heat, exertion, emotional upset, medication (aspirin, opioid analgesics, dextran and radiological contrast dyes, general anaesthesia) or insect sting (bee, wasp, etc.). Treatment of systemic mastocytosis can only relieve symptoms and does not change the course of the disease. Antihistamines (H1 and H2 blockers) are the mainstays of therapy; epinephrine and systemic corticosteroids have to be used in severe systemic reactions (1).

To our knowledge, only one case of myo-pericardial involvement, with atrio-ventricular block and pericardial effusion, has been reported in a patient with systemic mastocytosis (2) as usually symptoms such as palpitations and tachycardia are considered as a consequence of factors associated with anaphylaxis (e.g. hypotension, hypoxia, the administration of drugs for treatment of shock).

We report the case of a 57-year-old man who was referred by the emergency Department to our Division because of flushing with intense hands and feet

Paroxysmal atrial fibrillation can be a manifestation of systemic mastocytosis.

pruritus, dyspnoea, chest pain, diarrhoea. Blood pressure was 120/60 mmHg, while the pulse was arrhythmic as well as the heart beat. Electrocardiogram (ECG) showed atrial fibrillation (average ventricular frequency 120 beats/min). He was treated with 1 g of hydrocortisone i.v. and 10 mg clorpheniramine maleate i.m.; after 10 min, as he did not improve, 250 ml of saline solution with 1 g of methylprednisolone and 50 mg ranitidine i.v. were administered.

The patient after 5 min improved and presented only a mild erythema of the face and chest. Another ECG was carried out and showed sinus rhythm (78 beats/min.) so that no antiarrhythmogenic drugs had to be administered.

Echocardiography showed no ventricular kinetic disorder, normal cardiac size, normal valvular flow, ejection fraction 60%.

Blood tests were normal except for erythrocytation rate (ESR) (25 mm), C reactive protein (CRP) (19.50 mg/l), gammaglobulins (22.5%), glycemia (171 mg/dl), uric acid (9.40 mg/dl), alkaline phosphatase (353 U/l), neutrophilia (76%), monocytes (3%).

The patient reported that he had been under treatment for over 25 years with histamine antagonists (H1 and H2) associated, more recently, with anti-leucotrienes because of a systemic mastocytosis diagnosed elsewhere after skin, bone and bone marrow biopsy. He reported frequent episodes of flushing with intense pruritus, dyspnoea, diarrhoea and severe hypotension; during the last two attacks, about 1 year and 6 months before, treated elsewhere, atrial fibrillation had been registered.

In literature, it has been reported that cardiac involvement may occur during systemic anaphylaxis (3); in fact it has been shown that mastocytes are present in human heart (4), that during anaphylaxis there is a release of mediators from cardiac mastocytes (5) and that histamine causes an increased frequency and ejection fraction (6). This may explain the cardiac involvement in the case of systemic mastocytosis above reported as the patient had a negative history of cardiac disease and there were no predisposing factors for arrhythmia (alcohol, coffee, cocaine, hyperthyroidism, drugs).

We considered it of interest to report this case as it suggests that cardiac

anaphylaxis may occur during a systemic mastocytosis attack even if further studies on the cardiac involvement in systemic mastocytosis are needed.

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Isolated urticaria to ondansetron and successful treatment with granisetron

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Key words: drug allergy; ondansetron; urticaria.

The 5-Hydroxytryptamine₃ (5-HT₃) receptor antagonists (ondansetron, granisetron, dolasetron and tropisetron) are

powerful, well tolerated antiemetic drugs for which very few hypersensitivity reactions have been described (1–6).

In October and November 2003, a 42-year-old man presented two episodes of isolated and generalized urticaria a few hours after chemotherapy for pancreatic adenocarcinoma, consisting of gemcitabine, oxaliplatin, ondansetron, alizapride chlorhydrate and methylprednisolone. The first episode appeared 12 h after the first course and the second 30 min after the second ondansetron injection (other drugs had not yet been given). For the third course of chemotherapy, ondansetron was replaced by metopimazine and a high dose of methylprednisolone. Hypersensitivity reaction did not occur following this replacement, although nausea and vomiting were severe. Before these episodes, the patient had never been given ondansetron. The patient had no other medical history, in particular other drug allergy, apart from an intermittent allergic rhinitis (because of plane tree and birch) for about 8 years. Since Levine demonstrated for penicillins that IgE-dependant reactions could occur up to the 72th hour after drug administration (7), an IgE-dependant mechanism is always suspected when a typical clinical pictures of drug hypersensitivity occurs within the first 24 h after drug intake.

To confirm the presence of IgE antibodies specific for ondansetron, skin test was performed with this drug and two other 5-HT₃ receptor antagonists. Prick tests to ondansetron (pure concentration: 2 mg/ml), dolasetron (20 mg/ml) and granisetron (1 mg/ml) were all negative. Intradermal tests (IDT) to ondansetron proved positive at 0.2 mg/ml, with a wheal at 30 min of 2.5 times that of the initial papule. The IDTs tested negative to dolasetron and granisetron at 1/10 and at full concentrations. As these drugs are injected in short infusion, we choose to test up to pure concentration. Seven normal controls were tested and proved negative for IDTs to ondansetron at 0.2 mg/ml (wheals under 1.1 times that of the initial wheal); one emerged as positive

We describe a unique case of urticaria due to a selective allergy to ondansetron.