

Table 2. Patients with positive results to drugs used in anesthesia

Patient no.	Sex/age (years)	PA*	Intraoperative AR† (symptoms/drug)	Drug allergy	Atropine	Positive skin tests	Remarks
1	F/29	1	Bronchospasm/suxamethonium	No	Yes	Suxamethonium (ID‡) Atracurium (ID)	—
2	F/47	2	Disorientation/unknown	No	No	Atracurium (prick)	—
3	F/31	2	None	Yes	No	Suxamethonium (prick) Atracurium (prick)	AR to cosmetics
4	M/73	3	Hypotension/atracurium	No	No	Atracurium (prick)	—
5	M/61	1	None	Yes	No	Atracurium (ID)	AR to eye-drops
6	M/32	0	Anaphylactic shock/atracurium	No	No	Atracurium (prick)	—
7	M/74	1	Anaphylactic shock/cephazolin	No	No	Cephazolin (ID)	—

* Number of previous administrations of anesthesia.

† Adverse reactions.

‡ Intradermal.

to general anesthesia, allergy evaluation with MR should be performed in all patients with a history of anaphylactoid reactions during general anesthesia (9). The two patients with latent sensitization to MR had had adverse reactions to cosmetics and eye-drops containing benzalkonium, respectively, suggesting the possible implication of quaternary ammonium ions. The prevalence of latent sensitization to MR is similar to that of previous reports (9, 10). Therefore, preoperative screening may be advisable in patients with a history of adverse reactions to cosmetics or drug preservatives containing quaternary ammonium.

Key words: latex; muscle relaxant; screening.

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REFERENCES

1. Laxenaire MC, Moneret-Vautrin DA, editors. *Riesgo alérgico en anestesia y reanimación*. Barcelona: Masson, 1993.
2. Moneret-Vautrin DA, Laxenaire MC, Widmer S, Hummer Sigel M. Interêt des prick tests dans le dépistage de l'anaphylaxie aux myorelaxants. *Ann Fr Anesth Reanim* 1987; 6:352-5.
3. Moneret-Vautrin DA, Laxenaire MC. Concept and limitations of predictive tests in anaesthesiology. *Ann Fr Anesth Reanim* 1993;12:212-18.
4. Vervloet D, Charpin D, Pradal M, Lagier F, Birnbaum J. Faut-il faire des tests de dépistage aux myorelaxants? *Rev Fr Allergol* 1990;30:1-5.
5. Porri F, Pradal M, Rud C, et al. Is systematic preoperative

screening for muscle relaxants and latex allergy advisable? *Allergy* 1995;50:374-7.

6. Turjanmaa K. Incidence of immediate allergy to latex gloves in hospital personnel. *Contact Dermatitis* 1987;17:220-5.
7. Slater JE. Rubber specific IgE in children with spina bifida. *J Urol* 1991;146:578-9.
8. Lagier F, Vervloet D, Lhernat I, Poyen D, Charpin D. Prevalence of latex allergy in operating room nurses. *J Allergy Clin Immunol* 1992;90:319-22.
9. Moneret-Vautrin DA, Laxenaire MC. The risk of allergy related to general anaesthesia. *Clin Exp Allergy* 1993;23: 629-33.
10. Abrech C, Widmer S, Beaudoin E, Moneret-Vautrin DA, Laxenaire MD. Prospective study of sensitization to muscle relaxants. *J Allergy Clin Immunol* 1995;95:289.

Ascites in hereditary angioedema

HEREDITARY angioedema (HAE) is a rare hereditary immunodeficiency with decreased levels or function of C1 inhibitor (C1INH). It is characterized by recurrent episodes of soft-tissue swelling, with nonpruritic, nonpitting edema which may occur at any anatomic location.

Severe abdominal pain due to intestinal involvement is frequently seen during an HAE attack, sometimes being the sole manifestation of HAE (1). In these cases, a wrong diagnosis of acute abdomen is frequent, leading to unnecessary surgery (1).

Although rare, ascites may also appear during HAE attacks (2-5) and can constitute another confounding factor if the correct diagnosis is not suspected.

■ A 17-year-old white boy was admitted to our hospital for severe abdominal pain. This patient had already been diagnosed as having HAE, which

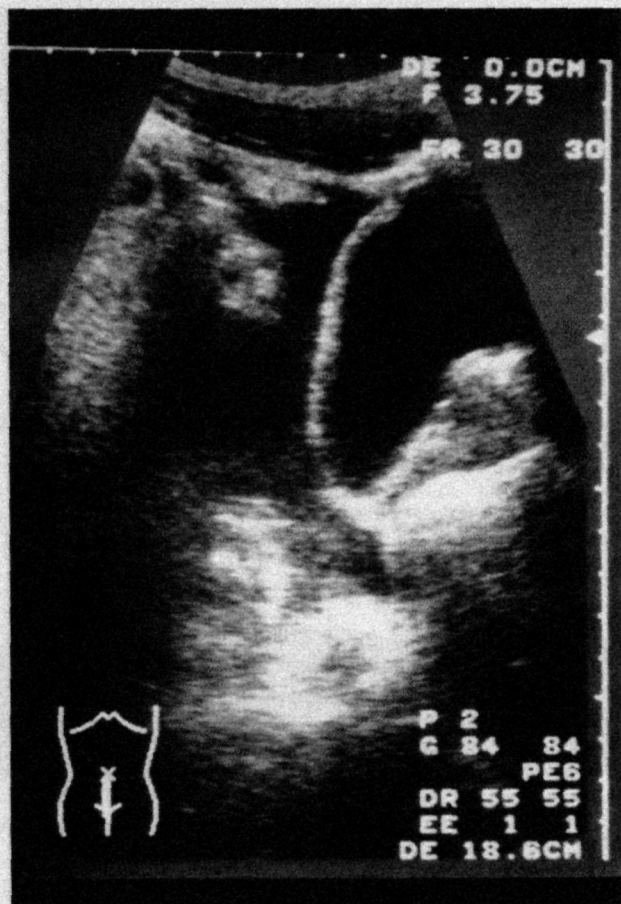


FIGURE 1 Abdominal ultrasonography showing moderate ascites.

was treated with danazol 100 mg/day. He had 3–4 episodes/year of angioedema of the face and limbs and abdominal pain. His mother had a similar clinical picture and also low C1INH levels. His grandmother and great-grandfather had had a history of recurrent swelling episodes.

Three days before admission, he began to have generalized severe abdominal pain, aggravated by ingestion of food and liquids, without vomiting, diarrhea, or noticeable angioedema.

On admission, his temperature was 37.2°C (axilar), heart rate 92 beats/min, respiratory rate 16 breaths/min, and blood pressure 120/60 mmHg. The results of the examination of head, eyes, ears, nose, throat, heart, and lungs were unremarkable. The abdomen showed diffuse tenderness, more pronounced in the lower left quadrant. Bowel sounds were present. Rectal examination was unremarkable.

Blood analysis revealed normal total and differential leukocyte counts, normal red cell parameters, and normal platelet counts. The biochemical emergency work-up revealed only a slight increase in

liver enzymes (serum glutamate oxaloacetate transaminase 58 IU/l – normal 1–30, serum glutamate pyruvate transaminase 43 IU/l – normal 1–30) with normal renal function, creatinine kinase, lactic dehydrogenase (LDH), and serum and urinary amylase levels. Urinalysis was unremarkable. The results of chest and abdominal radiographs were negative. Abdominal ultrasonography showed the presence of moderate ascites, visualized in all quadrants (Fig. 1). Paracentesis revealed an exudate (proteins 0.56 g/l; protein ascites/serum ratio of 0.8; LDH in ascitic fluid under 200 IU/l but LDH ascites/serum ratio over 0.6) without remarkable alterations in the cytologic examination of ascitic fluid.

The abdominal computed tomography (CT) scan showed slight thickening of the small bowel in the left lower quadrant. Complement measurements showed depressed C4 (0.03 g/l – normal 0.10–0.34) and C1INH serum levels (0.06 g/l – normal 0.22–0.34), with normal C3 and C1q.

We maintained close medical surveillance, therapy with intravenous fluids, intravenous ϵ -amino-

We report an HAE

patient with moderate

exudative ascites,

documented during an

abdominal crisis, which

was attributable to

HAE alone.

caproic acid, and danazol 200 mg/day p.o. The clinical picture resolved completely in less than 48 h. Four days after admission, abdominal ultrasonography was normal. Liver enzymes were also normal. C4 and C1 INH serum levels were 0.11 and 0.18 g/l, respectively. The

patient was discharged on danazol 200 mg/day.

The presence of intraperitoneal fluid with characteristics of an exudate in a patient with severe abdominal pain and tenderness is a finding that frequently leads to an exploratory laparotomy if HAE is not considered in the differential diagnosis.

Ascites in HAE is caused by extravasation of intravascular fluid into the peritoneal cavity due to edema of the intestinal wall (2, 4). Although peritoneal fluid with the biochemical characteristics of an exudate has already been reported (2), this is a rare finding in HAE, easily misleading doctors and surgeons into an exploratory laparotomy.

Our case report clearly shows that the cause of both the abdominal pain and exudative ascites was the HAE attack, as proved by the clinical evolution and by the very low levels of C1INH and C4 and normal C3 and C1q. This has also been reported by other authors (2–5), who additionally have

stressed the diagnostic value of ultrasonography (3–5). Indeed, in some patients with recurrent abdominal complaints and without known HAE, it was the ultrasonographic finding of ascites that led to complement measurements, allowing the correct diagnosis of HAE (5).

Acute pancreatitis may also occur during an HAE attack and may cause ascites. However, in our patient, serum and urinary amylase levels and pancreatic imaging (ultrasonography and CT scan) were normal. Our patient also had slight and reversible increases of serum liver transaminases during the HAE attack, a feature which has already been described (3).

We stress that HAE should always be present in the differential diagnosis of unexplained recurrent abdominal pain, in order to avoid unnecessary laparotomy. The presence of ascites in these patients, even if the peritoneal fluid is an exudate, makes the diagnosis of HAE more likely and should not confuse the clinician.

Key words: C1 inhibitor; hereditary angioedema; lactic dehydrogenase.

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REFERENCES

1. Weinstock LB, Kothari T, Sharma RN, Rosenfeld SI. Recurrent abdominal pain as the sole manifestation of hereditary angioedema in multiple family members. *Gastroenterology* 1987;93:1116–18.
2. Talavera A, Larraona JL, Ramos JL, et al. Hereditary angioedema: an infrequent cause of abdominal pain with ascites. *Am J Gastroenterol* 1995;90:471–4.
3. Shah TJ, Knowles WO, McGeedy SJ. Hereditary angioedema with recurrent abdominal pain and ascites. *J Allergy Clin Immunol* 1995;96:259–61.
4. Burghardt W, Wernze H. Ultrasonography for early diagnosis of hereditary angioneurotic oedema. *Lancet* 1987;2:165.
5. Laurent J, Toulet R, Lagrue G. Ultrasonography in the diagnosis of hereditary angioneurotic oedema. *Lancet* 1988;1:761.

Loratadine reduces the expression of ICAM-1

THE adhesion molecule ICAM-1 is the natural ligand of the integrin LFA-1 (CD11a CD18-Mac 1), which is expressed on leukocytes. Thus, we

can explain, at least in part, the mucosal infiltration by inflammatory cells during the allergic reaction (1). Loratadine is a new-generation antihistamine and exerts antiallergic effects, including the modulation of ICAM-1 expression on nasal epithelial cells of rhinitics during the pollen season (2). We aimed to confirm this report by evaluating the inhibiting effects of loratadine on conjunctival ICAM-1 expression upon the allergen challenge.

The study was a randomized, double-blind, two-arm, parallel-group design, comparing loratadine with placebo. The treatment period was 1 week. Twenty symptom-free subjects (seven women, and 13 men aged 18–50 years), with a previously documented diagnosis of allergic rhinoconjunctivitis to *Parietaria judaica*, and without ocular or nasal nonallergic diseases, were randomized. The study was performed outside the pollen season. No topical or systemic drugs were allowed for 1 month before and during the study period. All subjects gave their informed consent and the trial was approved by our department's ethical committee.

Allergen-specific conjunctival challenge (ASCC) was performed as previously described (1). Clinical conjunctival evaluation was performed at baseline and 30 min after challenge. Conjunctival hyperemia, lacrimation, itching-burning, and swelling of eyelids were scored on an arbitrary four-point scale, from 0 to 3 (0=absent, 1=mild, 2=moderate, 3=severe), and a total symptom score was calculated as the sum of individual symptom scores (1).

A sensitive immunoenzymatic alkaline phosphatase–monoclonal antialkaline phosphatase-complex procedure was employed to detect ICAM-1 expression on conjunctival epithelium (1). Details of the procedure have been reported elsewhere (1).

Results are reported as medians with ranges and as percentage frequencies. Differences were considered significant for *P* values less than 0.05 with a two-tailed test. All statistical calculations were done with the rank and GLM procedures of the SAS system.

All subjects showed an immediate clinical reaction within 5 min after challenge, at concentrations ranging from 50 to 600 AUR/ml. All subjects showed a remarkable ICAM-1 expression on conjunctival epithelial cells compared to baseline and the contralateral eye. Total symptom

Loratadine can modulate the ICAM-1 expression on conjunctival epithelial cells induced by allergen-specific challenge.

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