

significant difference in the expression of GAPDH mRNA in any of the groups (mean [95% CI]: IgA nephropathy 12 423 (10 938–13 908), other glomerulonephritis 12 533 (10 640–14 425); hypertension 12 590 (11 110–14 070); controls 12 577 (10 790–14 364).

We evaluated the clinical course of 15 patients with IgA nephropathy: endothelin-1 mRNA expression in monocytes was high on admission, but decreased gradually, as did urinary protein excretion, after treatment with prednisolone, anticoagulants, or antiplatelet drugs. By contrast, we could not detect endothelin-3 mRNA expression in monocytes in any patient or control.

The use of cDNA probe for endothelin-1 allowed us to examine the regulation of endothelin-1 mRNA by measuring local endothelin-1 gene expression in monocytes. Specific functions of T cells, B cells, and monocytes have been related to disease activity in IgA nephropathy. Endothelin stimulates mitogenesis in cultured rat mesangial cells, while increasing the expression of proto-oncogenes, which are presumably involved in regulating DNA replication. Proliferating mesangial cells up-regulate extracellular matrix synthesis, which may induce glomerular sclerosis.⁶ Endothelin-1 secreted by circulating monocytes would probably access the glomerulus, interstitium, or blood-vessel wall. Monocytes may be activated during their journey through the glomerular microvascular bed and interstitium to release endothelin-1 locally. Such a mechanism is consistent with our finding of a positive correlation between monocyte endothelin-1 mRNA values and histopathological changes or urinary protein excretion. Also, the negative correlation between endothelin-1 mRNA and creatinine clearance suggests that endothelin reduces glomerular filtration rate. Our findings show that increased endothelin-1 mRNA expression is specific to patients with IgA nephropathy. The low monocyte endothelin-1 mRNA values in patients receiving steroids and antiplatelet or anticoagulant drugs is probably attributable to immune suppression being associated with deactivation of monocytes and, thereby, decreased endothelin-1 mRNA expression.

This work was supported in part by a Grand-in-Aid for the study of Intractable Diseases from the Ministry of Health and Welfare, Japan.

References

- 1 Ehrenreich H, Anderson RW, Fox CH, et al. Endothelins, peptides with potent vasoactive properties are produced by human macrophages. *J Exp Med* 1990; 172: 1741–48.
- 2 Badr KF, Murray JJ, Mreyer MD, et al. Mesangial cell, glomerular and renal vascular responses to endothelin in the rat kidney. *J Clin Invest* 1989; 83: 336–42.
- 3 Nakamura T, Ebihara I, Nagaoka I, Tomino Y, Koide H. Activated peripheral blood mononuclear cells in IgA nephropathy express platelet-derived growth factor B-chain messenger RNA. *J Lab Clin Med* 1992; 120: 212–21.
- 4 Nakamura T, Ebihara I, Nagaoka I, Takahashi T, Tomino Y, Koide H. Abnormal regulation of insulin-like growth factor gene expression in peripheral blood mononuclear cells from patients with IgA nephropathy. *Am J Nephrol* 1992; 12: 292–302.
- 5 Lee SMK, Rao VM, Franklin WA, et al. IgA nephropathy: morphologic predictors of progressive renal disease. *Hum Pathol* 1982; 13: 314–22.
- 6 Ishimura E, Sterzel RB, Budde K, Kashgarian M. Formation of extracellular matrix by cultured rat mesangial cells. *Am J Pathol* 1989; 134: 843–55.

Division of Nephrology, Department of Medicine, Juntendo University School of Medicine, Tokyo 113, Japan (T Nakamura MD, I Ebihara MD, I Shirato MD, Mitsumine Fukui MD, Y Tomino MD, H Koide MD)

Correspondence to: Dr Hikaru Koide

Health risk in hospitals through airborne allergens for patients presensitised to latex

X Baur, J Ammon, Z Chen, U Beckmann, A B Czuppon

Two nurses had to stop practising because of severe work-related allergic respiratory or systemic symptoms. Both developed similar symptoms within a short time when they attended hospitals as outpatients or visitors or consulted a doctor or dentist, even when they had no direct contact with latex articles. The airborne latex allergen concentration in one room that induced symptoms was 128 ng per m³. In skinprick tests both women showed positive reactions to the phosphated cornstarch powder used to prevent latex gloves sticking together, but only when the powder had been in contact with latex. This powder seems a likely vehicle for the airborne latex allergen.

Lancet 1993; **342**: 1148–49

The most common clinical manifestation of type 1 hypersensitivity to latex is contact urticaria.^{1–5} In our experience, at least 30% of affected medical staff also suffer from conjunctivitis, rhinitis, or bronchial asthma when they or colleagues wear latex gloves.⁴ However, to our knowledge, such symptoms have not previously been reported in a non-occupational setting.

Between 1971 and 1992, a 27-year-old nurse underwent surgery 15 times for various diseases. She had worked predominantly in surgery wards since 1983 and began to report work-related urticaria in 1987, conjunctivitis in 1988, facial erythema and rhinitis in 1991, and asthmatic symptoms in 1992. She noticed that her respiratory and conjunctival symptoms started or worsened when she or somebody nearby changed latex gloves. She had a local hypersensitivity reaction when a bladder catheter was inserted after caesarean section. Hypersensitivity reactions to latex occurred also in everyday life (eg, touching the car window rubber seal, a balloon bursting) and when she consulted a doctor or dentist. She had a history of intolerance to bananas, iodine-containing radiographic contrast solutions, and Dolantin (pethidine HCl).

When she came to our lung function department as an outpatient, she initially showed no symptoms. Within a few minutes of entering she developed rhinoconjunctivitis, photophobia, cough, shortness of breath, and facial erythema even though she had touched no latex articles. Plethysmography about 10 min after she entered the room showed moderate bronchial obstruction. Latex gloves are put on or removed about three times a day in this room, but this was not done while the patient was there. 15 min later, increasing bronchial obstruction developed, with airway resistance rising from 0.495 to 0.751 kPa L⁻¹ s (normal <0.3 kPa L⁻¹ s).

In skinprick tests the patient had a 3–4-fold reaction⁶ to latex allergens (3 mg/L) and a 3-fold reaction to banana. There was no response to a panel of twenty common allergens. She gave a positive response to an extract of the phosphated cornstarch used to cover latex gloves (collected with a dust sampler). However, she showed no reaction to the cornstarch when it had never been in contact with latex.

Specific IgE concentration was estimated at 30 U/mL with commercial (Pharmacia) and our own latex allergens,⁴

but total IgE was normal (78 U/mL). Air dust was collected from the lung function room (90 m³) with a sampling device (Gravikon VC25, Stroehlein, Kaarst, Germany) equipped with a membrane filter (cut-off particle size >0.4 µm; Sartorius, Goettingen, Germany). A solid-phase competitive immunoinhibition assay with natural latex as the target allergen⁷ found a concentration of latex allergen in our lung function room of 128 ng per m³ of room air.

The second patient was a 33-year-old midwife who developed work-related contact urticaria, rhinitis, and conjunctivitis after practising for 7 years. She wore latex gloves for about 2 h per day and changed them ten to twelve times per shift. 8 years later she developed asthmatic symptoms in the ward and during exercise. Contact with a balloon and with her dentist's gloves caused strong local wheal and flare reactions and in the latter case also an asthma attack. 2 years later she developed general urticaria and anaphylactic shock during a routine gynaecological examination. 9 months ago she gave up working. However, when visiting a relative in hospital, she had rhinitis, conjunctivitis, and shortness of breath without direct contact with latex gloves.

This patient stayed in our hospital for diagnostic reasons for 3 days. At first she showed no symptoms. However, after coming for a few hours to the lung function department and staying in ordinary ward of the internal medicine clinic she developed persisting rhinoconjunctivitis, without direct contact with latex articles. Airway resistance was normal initially but moderately high the next day (0.49 kPa L⁻¹ s).

Total IgE was slightly high (146 U/mL). Routine skin testing with twenty common allergens gave negative results. However, the skinprick test was 3-fold positive⁶ for latex and the latex-specific IgE concentration was 4.86 U/mL. When she handled five pairs of latex gloves for 30 min while wearing synthetic gloves to avoid direct skin contact she developed conjunctivitis, facial erythema, and rhinitis.

These two case reports suggest that the handling of powdered latex gloves a few times per day produces concentrations of airborne latex allergen high enough to cause respiratory and conjunctival reactions in highly presensitised individuals. Swanson and colleagues⁸ have reported airborne latex allergen concentrations of up to 208 ng/m³ in various US hospital units but no cases of indirect hypersensitive reactions. We have found that the major latex allergen that binds 80–100% of IgE antibodies to sensitised patients is a 14.5 kDa protein, identified as the rubber elongation factor of the rubber tree *Hevea brasiliensis*.⁷ It adheres to the powder of latex gloves and can thus be transported to the respiratory tract. The airborne carrier consists of phosphated corn starch used by manufacturers to prevent the gloves sticking together.

The airborne dispersion of glove powder should be avoided since it is associated with significant and long-lasting contamination of the atmosphere by latex allergens. Either a more inert material could be used or glove powder omitted altogether.

References

- 1 Axelsson JGK, Johansson SGO, Wrangsjö K. IgE-mediated anaphylactoid reactions to rubber. *Allergy* 1987; **42**: 46–50.
- 2 Turjanmaa K. Incidence of immediate allergy to latex gloves in hospital personnel. *Contact Dermat* 1987; **17**: 270–75.
- 3 Slater JE, Mostello LA, Shaer C, Honsinger RW. Type I hypersensitivity to rubber. *Ann Allergy* 1990; **65**: 411–14.
- 4 Jaeger D, Czuppon A, Baur X. IgE-mediated allergy to latex proteins:

clinical relevance, diagnostic procedure and therapeutic aspects. *J Allergy Clin Immunol* 1991; **87**: 268.

- 5 Levy DA, Charpin D, Pecquet C, Leynadier F, Vervloet D. Allergy to latex. *Allergy* 1992; **47**: 579–87.
- 6 Aas K, Belin L. Standardisation of diagnostic work in allergy. *Acta Allergol* 1972; **27**: 439–68.
- 7 Czuppon AB, Chen Z, Rennert S, et al. The rubber elongation factor of rubber trees (*Hevea brasiliensis*) is the major allergen in latex. *J Allergy Clin Immunol* (in press).
- 8 Swanson MC, Yunginger JW, Warner MA, Hunt LW, Reed CE, Rochester MN. Quantitation of latex aeroallergens in operating suites (OR). *J Allergy Clin Immunol* 1993; **91**: 385 (abstr).

Professional Associations' Institute of Occupational Medicine Institute, Ruhr-University of Bochum (Prof X Baur MD, J Ammon, Z Chen PhD, A B Czuppon PhD), **and Research Institute for Hazardous Substances** (U Beckmann PhD), **Bochum, Germany**

Correspondence to: Prof X Baur, Gilsingstrasse 14, 44789 Bochum, Germany

Recurrence of Rift Valley fever in Egypt

Ray R Arthur, M Said El-Sharkawy, Stanton E Cope, Boulos A Botros, Said Oun, John C Morrill, Robert E Shope, Richard G Hibbs, Medhat A Darwish, Imam Z E Imam

Rift Valley fever (RVF) has been recorded in man and in domestic animals in Egypt after a 12-year absence. Human infections were first noted in the Aswan Governorate in late May, 1993. Only cases of ocular disease, an infrequent and late manifestation, were reported. Of 41 cases, 35 were tested serologically and 27 (77%) had RVF virus-specific IgM antibodies. An estimated 600–1500 infections occurred in the region. Abortions in cattle and buffalo were seen concurrently and antibodies to RVFV were present in 39% of domestic livestock, presumably unvaccinated. RVFV was isolated from an aborted water buffalo fetus.

Lancet 1993; **342**: 1149–50

Rift Valley fever (RVF) was first reported in Egypt in 1977, and in that year and the following one more than 20 000 human infections were recorded, with an estimated 600 deaths. The deaths of sheep and cattle constituted a major economic loss. RVF virus infection was last documented in Egypt in early 1981.¹

In the first week of July, 1993, health officials in the Kom Ombo district of the Aswan Governorate, 800 km south of Cairo, noted an increase in people seeking medical care for impaired vision. A presumptive case definition of visual impairment preceded by febrile illness was established. 41 cases meeting that definition were recorded from July 2 to July 25. Interviews indicated that the earliest appearance of ocular disease had been on May 25. There were 23 males and 18 females and all but 1 were adults. Cases were recorded in all five districts of the Aswan Governorate, but over 70% originated on the east bank of the Nile in Kom Ombo. However, cases occurred as far apart as Aswan in the south and Idfu, which are 100 km or so from each other, and these patients said that they had not travelled to the Kom Ombo area or obtained animals or meat from that area.

Patients complained of acute fever, with severe headache, retro-orbital pain, myalgia, and back pain. A few had