Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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ADVERSE REACTIONS TO A NEW FOOD INGREDIENT

To the Editor:

Meat substitutes marketed under the brand name Quorn (Quorn Foods, Inc., Riverside, Connecticut) are made with processed *Fusarium venenatum* fungus (1). These products, which have been marketed in the United Kingdom for over 10 years and in the United States since 2002, cause adverse reactions in certain persons (2). The most common reactions are vomiting and diarrhea, but reports to the Center for Science in the Public Interest suggest that other symptoms are involved. A recent study linked a severe hypersensitivity reaction in a patient with asthma to an immunoglobulin E antibody to a specific ribosomal protein in Quorn (3).

The Center for Science in the Public Interest, through a web site (www.quorncomplaints.com) and by mail, has received 597 reports of adverse reactions (Table 1), including 85% from the United Kingdom, 14% from the United States, and 1% from elsewhere in Europe. Some reports were supported by further correspondence or telephone calls.

One report of anaphylaxis concerned a 47-year-old man who experienced vomiting and diarrhea, followed by severe breathlessness, numbness of the mouth and lips, and loss of feeling in arms and legs. He was admitted to a hospital and advised to carry an EpiPen (King Pharmaceuticals, Inc., Bristol, Tennessee) after discharge. He had no other allergies. Another report concerned a 37-year-old woman who had allergies to seafood, poultry, kidney beans, and chickpeas. She was admitted following an anaphylactic reaction attributed to Quorn. Within 30 minutes of ingesting Quorn, she developed facial swelling, urticaria, and wheeziness, and was unable to speak. Her blood pressure was 59/35 mm Hg. She was given adrenaline, hydrocortisone, and chlorpheniramine, and advised to carry an EpiPen.

To ascertain the frequency of reactions to Quorn, as well as to common food allergens, we commissioned a telephone survey of 1004 British consumers aged 16 years or older (Table 2). Respondents were asked if they were allergic to various foods. Those who had consumed Quorn (n = 396) were asked if they experienced certain symptoms after eating Quorn. Although the sample size was limited, sensitivity to Quorn appeared to be at least as common as allergies to the major food allergens. Symptoms attributed to Quorn included stomachache or cramps (3% [n = 12]), diarrhea (1% [n = 4]), vomiting (1% [n = 3]), and indigestion, nausea, hives or rash, headache, and flatulence (<1% each).

Table 1. Symptoms Associated with Consumption of Quorn Mycoprotein*

Symptom	Number (%)
Vomiting	398 (67)
Diarrhea	199 (33)
Urticaria	36 (6)
Fainted, almost fainted	34 (6)
Bloody vomit, burst blood vessels near the eyes, or rectal hemorrhage	16 (3)
Swelling of the face or throat, difficulty breathing	16 (3)
Anaphylaxis	4 (1)

* Based on 546 adverse reaction reports.

Table 2. Frequency of Reactions to Quorn and Other Foods*

Food	% Allergic or Sensitive
Quorn mycoprotein	5%
Shellfish	3%
Milk	2%
Peanuts	2%
Wheat	2%
Gluten	1%
Nuts (not peanuts)	1%
Fish	1%
Eggs	1%
Soya	0.5%
Other	3%

* N = 1004 respondents. Sensitivity to Quorn mycoprotein was based on 396 respondents who had eaten foods containing Quorn. Other percentages are based on the entire sample. Survey conducted in March 2003 by Taylor Nelson Sofres, London, United Kingdom.

We question the wisdom of introducing into the food supply an ingredient that appears to cause severe gastrointestinal reactions, anaphylaxis, and other symptoms.

Michael F. Jacobson, PhD
Center for Science in the Public Interest
Washington, D. C.

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FATAL OUTCOME OF SARS IN A PATIENT WITH REACTIVATION OF CHRONIC HEPATITIS B

To the Editor:

A 44-year-old man with known compensated chronic hepatitis B was admitted on March 5, 2003, with a 1-week history of malaise, anorexia, and tea-colored urine. On examina-

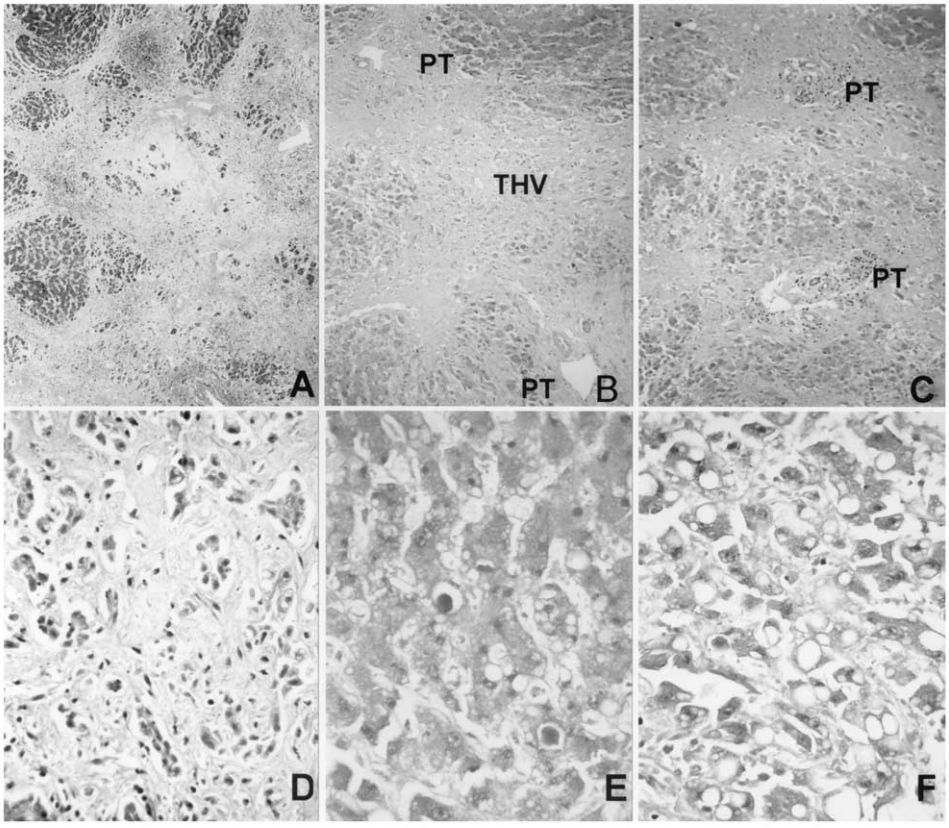

Figure. Photomicrographs of the liver necropsy specimen (A: Masson trichrome, ×15; B to F: hematoxylin-eosin, B: ×15, C: ×60, D: ×120, E and F: ×240). PT = portal tract; THV = terminal hepatic vein.

tion, he had jaundice but no other signs of chronic liver disease. He was afebrile and his vital signs were normal. He had thrombocytopenia (platelet count, 124×10^9/L) and a deranged coagulation profile (international normalized ratio: 1.82). Liver function was grossly abnormal (total bilirubin, 6.7 mg/dL; albumin, 3.2 g/dL; alkaline phosphatase, 132 U/L; alanine transaminase, 2090 U/L). Serological tests confirmed hepatitis B surface antigen (HBsAg) positivity. He was hepatitis B e antigen (HBeAg) negative and anti-HBeAg positive. Both anti-hepatitis C virus and immunoglobulin M anti-hepatitis A virus antibodies were negative. His serum HBV-DNA was 5.1 MEq/mL.

The diagnosis was acute reactivation of chronic hepatitis B. Treatment with lamivudine (100 mg daily) was started. He remained clinically stable with biochemical improvement until day 4, when he developed a fever of 39°C. No focal source of infection was found and his chest radiograph was normal. Despite empirical intravenous cefotaxime and levofloxacin, his fever persisted. A chest radiograph on day 7 showed bilateral air space consolidation. His condition deteriorated the next day, involving respiratory failure and shock, and he was admitted to the intensive care unit. By this time it was apparent that he had contracted severe acute respiratory syndrome (SARS) during the hospital outbreak of the disease (1,2). This was subsequently confirmed when SARS-coronavirus (SARS-CoV) was isolated from his nasopharyngeal aspirate. Despite mechanical ventilation and therapeutic intervention that included broad-spectrum antibiotics, ribavirin, and pulse methylprednisolone, his condition worsened and led to multiple system organ dysfunction. The patient died on day 16.

Postmortem examination revealed extensive consolidation in the lungs with diffuse alveolar damage and hyaline membrane, features commonly found in patients who had died of SARS. His liver was cirrhotic and showed severe hepatocyte dropout with small islands of hepatocytes left in the parenchyma (Figure, A). Most of the portal tracts had minimal-to-mild chronic inflammatory cell infiltration with some portal tracts showing lymphocyte depletion (Figure, B and C). There was moderate periportal cholangiolar transformation (Figure, D). Furthermore, severe cholestasis was demonstrated with large bile plugs observed within the bile canaliculi (Figure, E). There was also mild macrovesicular steatosis without accompanying hyaline change (Figure, F). SARS-CoV could not be isolated from the liver tissue.

Peiris et al (3) found a higher percentage of patients with HBsAg posi-

tivity among SARS patients who developed acute respiratory distress syndrome. However, whether the worse pulmonary outcome was directly related to chronic hepatitis B is not known. In this patient, reactivation of chronic hepatitis B was probably not the major cause of death. The patient was improving until he developed symptoms of SARS. With lamivudine treatment and without SARS, he most likely would have recovered (4). Conversely, his impaired hepatic function might have aggravated the course of SARS and contributed to his death. The interval between the onset of fever and death was much shorter (16 days) than the mean admission-to-death time of 36 days in other SARS patients who had died (2).

The postmortem finding sheds light on the effect of SARS on the patient's hepatitis. Lymphocyte depletion in the portal tracts was unusual because submassive necrosis of liver due to exacerbation of chronic hepatitis B is often associated with prominent lymphocytic infiltration (5,6). Peripheral lymphopenia is an important feature in SARS and appeared to have affected the liver even in the face of hepatitis. Interestingly, lymphocyte depletion in this patient is reminiscent of hepatic histology in patients with full blown AIDS (7). This may reflect the overwhelming effect of SARS on the immune system. As no SARS-CoV could be isolated from the liver tissue, a direct cytopathic effect of SARS-CoV on the liver was unlikely.

Cytokine dysregulation probably is important in the pathogenesis of SARS, and hence steroid or other immunosuppressive agents have been used as first-line therapy (1,8). Because immunosuppression may predispose to flare up of chronic hepatitis B, we propose the use of prophylactic antiviral therapy, such as lamivudine, before beginning immunosuppressive treatment in SARS patients with chronic hepatitis B (9). By decreasing the viral load, the likelihood of reactivation of hepatitis B may be reduced. Meanwhile, further studies are warranted to investigate whether chronic hepatitis B has any effect on the prognosis of SARS.

Alex Y. Hui, MD
Henry L. Y. Chan, MD
Choong-Tsek Liew, MD
Paul K. S. Chan, MD
Ka-Fai To, MD
Chin-Pang Chan, MD
Joseph J. Y. Sung, MD, PhD
Prince of Wales Hospital
Chinese University of Hong Kong
Hong Kong, China

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