SHORT REPORT

Occupational asthma caused by sodium disulphite in Norwegian lobster fishing

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Sulphite is added to various foods due to its antioxidative effect. Sulphite is known to provoke bronchoconstriction in some atopics and individuals with asthma. Some asthmatics develop symptoms after ingestion of sulphite preserved foods.¹

Occupational asthma due to sulphite has been described in the potato, wine, and laundry industries.² ³ An asthma-like syndrome has been described in agricultural workers during apricot sulphurisation.⁴ To our knowledge this is the first reported case of sulphite related occupational asthma in the fishing industry.

CASE REPORT

A 31 year old fisherman with no prior history of asthma or pulmonary symptoms developed a dry cough for the first time around 1 September 2001 while dipping Norwegian lobsters (Nephrops Norvegicus) into a sodium disulphite (SD) solution on board a fishing vessel. A wire basket containing 15 kg lobster was dipped into a barrel containing 100 litres of water with 2 kg SD powder. This process takes place on deck with a roof. This was the only time that the patient himself mixed the solution. Otherwise he was exposed as a “bystander” about 2 metres from the container.

Later in September 2001, while fishing Norwegian lobster with exposure to the sulphite solution, he developed coughing with shortness of breath for the first time. He was successfully treated with bircanyl and budesonide. Later in the autumn he fished cod without symptoms.

While fishing Norwegian lobster again on 10 February 2002 he developed severe shortness of breath. He was treated in hospital; symptoms improved after prednisolone.

After this he started treatment with continuous budesonide and bircanyl. Peak flow monitoring showed variation from 650 l/min to 200–400 l/min.

The patient was studied in our department in July 2002; he had not been exposed to SD since February. He had not taken any asthma medication for two months. Initial lung function tests were: FEV₁ 5.05 l (100%), FVC 7.05 l (115%), and FEV₁/FVC (72%) (S-model spirometry no. 20.600, Vitalograph Ltd, Buckingham, UK). Histamine challenge gave a borderline response with PC₂₀ = 7.8 mg/ml.⁵ His eosinophil count (0.56 x 10⁹/l) and total IgE (717 kU/l) were increased. RAST for shrimp, crab, and lobster were negative. Standard prick tests and SD were negative. A histamine release test for SD was negative (ReferenceLab, Copenhagen).

After obtaining informed consent a non-blinded specific bronchial challenge, replicating the work process, was performed in a hospital setting. A sieve filled with plastic coffee cup holders was used. A baseline FEV₁ using the previously described equipment was 5.25 l. The subject then dipped the sieve in 10 l of tap water for 5 minutes as a placebo. FEV₁ was 5.00 l (95% of the initial value) at the end of the placebo period. Within 2 minutes thereafter the subject dissolved 200 g SD in the 10 l and the provocation was initiated. After 5 minutes FEV₁ fell to 4.65 l (89%). Thereafter he developed severe shortness of breath, wheezing, flushing, and coughing with rhonchi. After 10 minutes, FEV₁ fell to 4.15 l (79%) and the exposure was stopped (see fig 1). After salbutamol treatment symptoms disappeared and the FEV₁ was 4.90 (95%).

DISCUSSION

It is known that sulphite can induce bronchoconstriction in some asthmatics and atopics.⁶ Sulphite induced symptoms were reported in a patient after the oral ingestion of sulphite preserved foods.⁷ Sulphite gives rise to 95% of the total allergy to fish.
bronchoconstriction is apparently not caused by an IgE mediated hypersensitivity reaction, but is possibly mediated via neural pathways. Some diuretics like furosemide inhibit sulphite induced bronchoconstriction.⁷

Exposure to SO₂ is known to cause respiratory symptoms in asthmatics, probably via an irritant mechanism. Sulphite aerosol produces SO₂, but it has been shown that the inhalation of sulphite aerosol causes more bronchoconstriction than the corresponding level of SO₂ alone. Thus sulphite appears to have a bronchoconstrictor effect in itself in addition to an SO₂ effect. It is possible that SO₂ forms sulphite in the airway mucosa.⁸

Anaphylaxis to sodium bisulphite has been reported in an asthmatic patient, suggesting a specific reaction.⁹

The initial drop in FEV₁ after placebo during the sulphite challenge suggests a degree of bronchial hyperreactivity. It is unlikely that the FEV₁ would have dropped further if the placebo period had been extended after the planned 5 minutes. He had neither symptoms during the placebo nor a history of respiratory symptoms when exposed to weather changes, or aerosol when showering.

It is likely that SD was the specific cause of occupational asthma in this case. There were good correlations between occupational exposure and both start of symptoms and repeated asthma episodes. Symptoms diminished during periods when he was not involved in lobster fishing. A specific bronchial provocation test under controlled circumstances was positive. The Danish National Compensation Board has compensated this case.

There was no evidence that the patient had a specific IgE related reaction to sulphite. It is unlikely that SO₂ levels during the specific provocation test were high enough to cause non-specific irritation resulting in asthma, because the attending nurse and physician detected a slight sulphur odour, but experienced no symptoms. However, an irritative response cannot be entirely excluded as no air measurements were taken.

Sulphite is used in food, laundry, and photographic industries. Respiratory morbidity caused by sulphite preserved foods ingested by atopics and asthmatics has been well documented. However, long term effects of sulphite exposure have not been well studied. The present study documents an association between occupational sulphite exposure and asthma.

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