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A case of occupational asthma from metabisulphite in a fisherman

Richard Pougnet[1], Brice Loddé[1], David Lucas[2], Dominique Jégaden[3], Sally Bell[4], Jean-Dominique Dewitte[5]

[1]Department of Occupational Health, University of Brest, France
[2]Occupational Medical Service of Iroise, Brest, France
[3]Department of Occupational Health, University of Brest, president of the French Society of Maritime Medicine (SFMM), France
[4]Clinical Quality Consultant, 6 Devonshire Terrace, London W2 3DN, UK
[5]Professor of Occupational Medicine, University of Brest, France

ABSTRACT

Background. *The objective was to describe a case of occupational asthma in a fisherman linked to the inhalation of metabisulphites, then to discuss the history and actions for prevention.*

Material and methods. *We report the case of a fisherman fishing for Dublin bay prawn (Nephrops), who suffered from asthma after being exposed to metabisulphites. This case is compared with other cases in the occupational medical literature. He was a 53-year-old male and a former smoker, who presented with dyspnoea and a very productive cough, due to both chronic exposure to metabisulphites and acute exposure to contamination due to the breakdown of the ventilation system in the trawler on which he was fishing. The symptoms appeared to be occupational. Tests carried out in the following months revealed reversible airway obstruction with non-specific bronchial hyperreactivity, while allergy tests were negative. A visit to the trawler allowed us to determine the unusual cause of exposure in the case. Other reported cases concern normal working processes causing reversible airway obstruction.*

Discussion. *Metabisulphites are antioxidants and preservatives used in the food industry which have been identified as the causative agent in occupational asthma since the 1980s. Only two other cases have been previously reported in fishermen, however. The clinical presentation of our subject was notable for the productive nature of his cough. The appearance of symptoms after combined acute and chronic exposure made us consider an irritative pathophysiology in this asthma, as opposed to sensitization. The negative allergy testing in this subject was similar to other cases reported in the literature. Our subject alone was submitted to patch testing, which was also negative.*

Conclusions. *Our case represents occupational asthma caused by metabisulphites with clinical signs notable for the productive nature of the cough. Few cases have been reported amongst fishermen despite the significant use of this substance with irritant and sensitizing characteristics in the workplace.*

Key words: occupational diseases, asthma, sulphites

INTRODUCTION

Metabisulphites, which are used as preservatives and antioxidants in both the food production and laundry industries, have already been described as the cause of occupational asthma for some employees in these sectors [1–3]. They are also implicated

Dr Richard Pougnet, Centre de Consultations de Pathologies Professionnelles et Environnementales, CHU MORVAN, Avenue Foch, 29200 Brest, France

in food intolerance and drug allergies amongst the general population.

Although these compounds are widely used in the fishing industry, notably for the preservation of shellfish [4–6], occupational asthma caused by metabisulphites is poorly described in fishermen [7].

The purpose of this paper is to describe a case of occupational asthma in a fisherman after exposure to metabisulphites, and to discuss the clinical and laboratory characteristics in comparison with two other cases in fishermen already reported in the literature [8, 9].

CASE REPORT

The patient had been a 53-year-old professional Dublin bay prawn fisherman for the previous 22 years. He had no history of atopy or respiratory diseases, specifically no asthma. He had been a non-smoker for the past 5 years and his previous tobacco consumption was estimated at 30 packs per year.

Within his professional duties he spread metabisulphite by hand onto freshly caught Dublin bay prawn. His work involved taking a handful of metabisulphite in the form of a fine white powder, and sprinkling it on the Dublin bay prawn as if sowing seed. This operation lasted two or three minutes, and would be repeated each time the trawl was lifted. This exposure was therefore repeated many times each day during the Dublin bay prawn fishing season (April––October), and had been going on since he had started to work on this trawler six years previously.

His symptoms began in September 2007 after a night when he and his colleagues had detected a strong odour of metabisulphite in the crew quarters. After handling the metabisulphite following the first trawl of the day, he presented with acute expiratory dyspnoea. This dyspnoea was accompanied by a cough with profuse sero-mucous sputum. He did not describe any other symptoms (ENT, ophthalmological, dermatological, etc.). The respiratory symptoms resolved spontaneously after fishing, with the exception of the cough, which continued for the rest of the trip (2 weeks). When he came back onshore (for 3 to 10 days at a time) the cough became dryer and less severe, but it became worse and more productive each time he treated the catch during further trips.

He consulted his general practitioner in early October 2007. Auscultation found rales and rhonchi. The blood count was normal; in particular, there was no eosinophilia. The chest X-ray was normal. His doctor requested a respiratory opinion and prepared an occupational injury certificate.

Pulmonary function tests (PFT) showed a 20% fall in FEV1 between periods of sick leave and resumption of work with further exposure. Treatment with inhaled budesonide and sick leave improved his symptoms after a few weeks. However, the cough persisted, becoming dry and predominantly nocturnal. During December, bronchoscopy showed a non-specific inflammation of the lining of the trachea and bronchi. The patient continued to use inhaled corticosteroids for six months until the symptoms disappeared and PFT returned to normal in June 2008. The maritime health physician instructed him not to use metabisulphite. He stopped Dublin bay prawn fishing and, therefore, was no longer exposed to metabisulphite.

In late 2008, the patient no longer had any symptoms, with the exception of a recurrence of his cough and shortness of breath during a recent exposure to paint. This exposure was not work related, and it was not possible to retrieve the name or safety data for this product. At this point, the maritime health physician questioned the possibility of a resumption of his previous duties. As a result, he asked the Occupational and Environmental Disease Centre (OEDC) to comment.

Allergy testing and PFT were performed by the OEDC. Skin prick tests for pneumo-allergens, tropomyosin [10], latex, and metabisulphite were negative, as were patch tests with metabisulphite powder. Total serum IgE was normal. There was no evidence of specific serum IgE against tropomyosin or against sodium sulphite. The haemogram was normal. PFT showed a peripheral obstructive disease with reversibility (post bronchodilator test): the MMEF25 was 2.16 L (56% predicted) and increased to 2.82 L (86% predicted) after inhalation. The FEV1 was normal. The bronchial challenge test was positive with 1600 micrograms of methacholine causing a 26% drop in FEV1.

In addition, we visited the trawler on which the subject worked. This enabled us to note that metabisulphite was stored in 5 kg plastic bags. These bags were frequently broken. They were stored on the main deck, near an air vent. This fact explained the presence of the smell of metabisulphite in the crew quarters: the air vent delivered air loaded with metabisulphites to the area. The bed of the patient was close to the air vent area.

A diagnosis of occupational asthma induced by irritants was made. Indeed, the clinical presentation showed an occupational asthma pattern. In addition, laboratory examinations showed bronchial hyperre-

activity, and metabisulphites are already known to be a causative agent of asthma. At present, the fisherman has returned to work on the same trawler, but he no longer uses metabisulphite.

LITERATURE REVIEW

A search of literature revealed two articles about occupational asthma with metabisulphite among fishermen: Steiner et al. in 2008 in the United Kingdom [8] and Madsen et al. in 2004 in Norway [9].

Metabisulphites (S_2O_5) belong to the sulphite family. This group also includes neutral sulphites (SO_3) and bisulphites (HSO_3). The occurrence of occupational asthma due to metabisulphites has been recognised since the 1980s [1]. The pathophysiology is based on different mechanisms: the release of SO_2 [3]; immediate IgE-dependent hypersensitivity [3]; blocking the metabolism of arachidonic acid; increased histamine release due to a lower threshold of nerve fibre excitability causing bronchoconstriction [4]; action of bradykinin B2 receptor [5]; mast cell activation with direct release of mediators [6]; deficiency of sulphite oxidase; and response by cholinergic parasympathetic reflex [2]. Some authors make the assumption of a dual pathogenesis: on the one hand true hypersensitivity, and on the other hand a reaction due solely to the irritant effect of the product [9, 10].

DISCUSSION

The fisherman in our clinical case presented with a productive cough, initially with profuse mucosalivary sputum. This factor differs from cases of asthma previously described in the literature where there was no such sputum [2, 3, 8, 9]. Another unusual fact about the history of our subject was that his symptoms appeared after an unusual exposure: the morning after the sailors noticed metabisulphite in the crew quarters. This observation, however, agrees with those of Madsen et al. on one point: the fisherman in their study developed asthma during a very specific exposure. Whereas he was usually exposed to a solution of 2 kg of metabisulphite in 100 L of water, his illness was triggered after a single exposure to the powdered form of metabisulphite. However, the fisherman described by Steiner et al., who was also exposed to the powder whilst using a similar mixture, reacted to the solution when tested.

His respiratory symptoms had an occupational pattern initially, and were the same as those previously described in occupational asthma with metabisulphites, although some authors have also described the onset of symptoms following the ingestion of sulphites in foods [2].

Our subject continued to have a productive cough whilst at work when fishing. The cough was exacerbated by the manipulation of metabisulphite and was then accompanied by dyspnoea. This dyspnoea has never been of sufficient severity to necessitate an emergency consultation. On the other hand, the fisherman described by Madsen et al. presented severe asthma attacks necessitating hospitalization. These asthma attacks occurred immediately after exposure to metabisulphite and consisted of dyspnoea and dry cough. As well as periods of work and contact with metabisulphite, our subject also had a nocturnal cough that persisted for several months. This observation was comparable with that of a wine factory worker [2] and with that of a fisherman described by Steiner. Moreover, the latter continued to show symptoms for more than two years. Furthermore, our patient showed a recurrence of his cough when exposed to respiratory irritants which did not contain metabisulphites. The recurrence due to exposure to paint was long after the original exposure.

Unlike other subjects, our fisherman has shown no extra-pulmonary signs: urticarial-type rash, watery nasal discharge with sneezing, conjunctivitis, etc. This led us to discuss an irritative mechanism.

Given the potential risks, our team has not made a specific challenge test, especially since, as pointed out by the team of Malo et al. [11], metabisulphites are known to cause non-specific bronchospasm, particularly in patients with bronchial hyperreactivity to methacholine [12]. Similarly, an oral test has not been proposed [13], given the absence of respiratory or ENT reaction in our subject during the ingestion of food [14] rich in metabisulphites (preservatives E220, E222). Indeed, we found that we had sufficient evidence to make the diagnosis of occupational asthma: the reversible obstructive disease persisting after one year of evolution and non-specific bronchial hyperreactivity, associated with an occupational pattern.

Regarding allergy testing, the tests performed on our patient were negative. In other cases in the literature, prick tests to metabisulphite have never been positive, so one might question the relevance of such a test. For the fisherman described by Steiner et al., tests were also negative although he presented with signs of skin allergy. However, the fisherman described by Madsen et al. had eosinophilia and increased total IgE whereas specific IgE against seafood was negative, suggesting that the increase in

total IgE was due to specific IgE against metabisulphites. No team reported measurement of specific IgE against that substance previously. Furthermore, we were the only ones to measure specific IgE against sulphites. Normal levels are thus not an exclusion criterion for the diagnosis of metabisulphite asthma. This could be explained by the time elapsed since the exposure, the lack of cross reaction between sulphite and metabisulphites, or by the fact that the pathophysiology was not IgE-mediated in our subject.

Only our fisherman underwent bronchoscopy, which demonstrated a non-specific inflammatory aspect. The chest X-rays were all interpreted as normal.

The case reported here brings together several of the criteria of reactive airway dysfunction syndrome or Brooks' syndrome [15]: the absence of previous respiratory symptoms or asthma, obstructive respiratory disease, positive methacholine test, and the absence of other respiratory diseases. As for the concentration in breathed air, the smell experienced by sailors on the night preceding the onset of symptoms favours a concentration of at least 1–5 ppm in the air in the crew quarters, which is the threshold noted in the chemical information of this product. It was a potentially high exposure since our subject alleged to have noticed a strong smell, well above the threshold of smell. As a corollary, these respiratory events could correspond to a Reactive Airway Dysfunction Syndrome (RADS). Only one case was previously described by Steiner et al. [8].

A review of the history and a visit to the trawler allowed us to reconstruct the facts. The metabisulphites were stored near an air vent and polluted the ventilation for the crew quarters. The bunk of our subject was the closest to the air vent, so he had to breathe the contaminated fumes. The symptoms appeared to have started following exposure to high concentrations of metabisulphite in the crew quarters.

Because of memory bias, however, we cannot be sure that the symptoms described appeared within 24 hours of the onset of the metabisulphite odour near his bunk. As to the precise concentration of product in the atmosphere, the retrospective nature of our investigation places this criterion outside the scope of our investigation, so we could not confirm a diagnosis of RADS for this subject, especially given that even if the exposure had been at high concentrations on that day, and the fact that he had been exposed chronically in the 6 preceeding years points to an immuno-allergic mechanism.

It was possible to return to work without further exposure. This measure was successful despite the reorganization of work required for a vessel with 5 fishermen.

It is suggested that it was not easy for the fisherman in our study to wear personal protective equipment because of the lack of supervision of compliance in wearing masks, the discomfort it brings, and the lack of trust in its effectiveness. Indeed, in three published cases, this solution has been adopted with different results: an employee of a winery [2] in the 1980s continued to work for several years by wearing a simple surgical mask, a prawn packer [8] also successfully used a mask, but another patient employed in a prawn-processing factory [8] presented, despite the masks, with two episodes of acute decompensation.

CONCLUSION

The profuse bronchorrhea which initially dominated the clinical picture in our subject is a variant not previously encountered. He additionally developed a reaction to irritants.

Although occupational asthma with metabisulphites has already been described in different sectors, it is still an infrequently reported aetiology in the fishing industry. The result of the lack of description in the medical literature of this kind of disease is a lack of specific preventative measures, particularly the lack of information for employees handling these preservatives and antioxidants, who should be encouraged to be more vigilant in the storage of these preparations. As for owners, their responsibility to protect the health of their employees should encourage them to replace or improve preventative measures, including effective storage.

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