Occupational Asthma in a Beet Sugar Processing Plant*

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A patient with occupational asthma in the beet sugar processing industry is described. Symptomatology, skin testing, immunologic testing, and specific bronchoprovocation testing indicate exposure to moldy sugar beet pulp was the cause of the patient's occupational asthma. Cooperation between the treating physician and public health authorities is encouraged. ***(Chest 1992; 101:1720-22)***

Sugar beets are an important source of sugar in the United States. Nationwide there are approximately 40 processing plants and 15,000 beet sugar mill workers.

Respiratory disease, in particular hypersensitivity pneumonitis, secondary to work in sugar cane processing is well recognized.[1,2] We report here the case of a patient with occupational asthma from exposure in a beet sugar processing mill.

Methods

Michigan physicians are required by law to report known or suspected occupational illness to the State Health Department (Part 56 of Public Act 368 of 1978). The index patient was referred by an occupational physician who was evaluating the patient for a worker compensation claim.

An Occupational Safety and Health Administration (OSHA) enforcement inspection was initiated at the facility in response to the physician's report.

Bronchoprovocation testing was done following a standard protocol.[3] Baseline methacholine testing was done the day preceding the challenge. Exposure to each challenge material was carried out by the patient pouring the material back and forth between two stainless steel pans, first for 30 s, then for 2 min, then for a second 2 min, and finally for 5 min. Spirometry was done 10 min after each period of exposure; after the last 5-min exposure, spirometry was done every 10 min for an hour, then every hour for 7 h and then at 24 h. There was a minimum interval of one week between the challenge with the control dust, the fresh sugar beet pulp, and the moldy sugar beet pulp.

Description of Industrial Process

After harvesting, sugar beets are stored in piles outdoors. The beets are taken from these outdoor piles to a water flume where they are carried through a series of rock catchers and weed and trash separators. The beets are moved by conveyor belts to the slicers, which slice them into the shape of french fried potatoes. The sliced sugar beets are fed in one end of a diffuser and hot water in the other end. The liquid leaving the diffusers is called raw juice and contains 10 to 15 percent sucrose. This raw juice is then purified by multiple liming (addition of calcium oxide) and carbonation steps (addition of carbon dioxide and carbon monoxide). The water is evaporated in vacuum pans (now 50 to 65 percent sucrose) and then seeded to crystallize the sugar. The sugar is then conveyed to large storage silos.

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The plant under investigation processed approximately 1,000,000 tons of sugar beets per year. Approximately 120,000 tons of sugar are produced and 800,000 tons of sugar beet pulp. Wet pulp from the diffuser is first pressed and then dried to reduce moisture to 10 percent by weight. The dried pulp is pelletized and then taken by conveyor to large storage silos. The pulp is used for animal feed.

Operations and housekeeping on the sugar side of the plant are much cleaner as is typical of a foodprocessing area. Special coveralls and hats were worn by employees in the sugar area. Operations in other parts of the plant that involve multiple reaction kettles, kilns, furnaces, produce and steam pipes, are similar to those in a chemical facility. Housekeeping in this part of the plant was poor.

Other ancillary operations include a lime kiln and three large boiler-turbine generators to produce the electricity and steam needed for the process.

The operation is a seasonal operation with a "campaign" lasting four to six months after harvesting. The work force is approximately 300 during the campaign, but is reduced to less than 100 at other times of the year. During the off season, employees work on cleaning, maintenance, and repair.

Three years previously, there had been a fire and explosion in the pelletizing area secondary to pulp dust. Areas around and beneath the conveyor belt had significant accumulation of powdered material. Pulp that had become wet appears moldy and is extremely malodorous. The whole facility, as well as the surrounding community, has a distinctive odor that is present on clothes after leaving the plant.

Case Report

The patient, a 33-year-old white man, was referred to the Michigan Department of Public Health in December 1988. The patient first began experiencing shortness of breath, dry cough, sharp chest pains, sore throat, headache, fatigue, stiffness in his joints and difficulty sleeping in the summer of 1985. He was laid off for two months. He stated that his symptoms returned June 1986 and persisted until told by an allergist to leave work July 1987. He returned to work October 1988 with reoccurrence of his symptoms. His symptoms remitted after he left work February 1989. He has not returned since February 1989. He has had a history of nasal congestion his whole life. There was no family history of allergies. He had never smoked cigarettes.

He first began working at the sugar beet processing facility in 1973. He worked there only during the "campaign" from 1973 to 1985. During the rest of the year, he worked as a truck driver and repairing trucks. His job repairing trucks included welding and spray painting. From 1974 to 1976 he was a tank driver in the Army. Beginning May 1985 he worked full time at the sugar beet facility. He initially became sick while installing new equipment in the beet pulp area.

The patient had a normal chest roentgenogram in November 1986, January 1987, June 1988, and September 1988. He was noted to be wheezing on examination in July 1987. He had been treated with bronchodilators and steroids during the 15 months he was off work: July 1987 to October 1988. In September 1988, his FVC was 4.86 L (91 percent of predicted), FEV_1 was 4.44 L (106 percent of predicted), FEV_1/FVC was 91 percent, and FEF25-75 percent was 6.57 L/s (118 percent of predicted). In December 1988, he had a positive methacholine challenge test with 37 percent decrease in FEV_1 at 14 mg. In July 1987 he was scratch test positive to cat epithelium, birch and willow tree, and timothy and June grass (2+), and alternaria (3+). At this time he had intradermal reaction

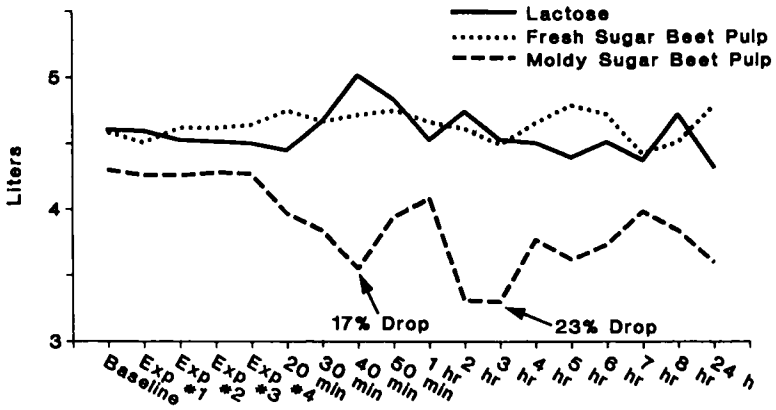

FIGURE 1. Change in FEV_1 after challenge to control lactose, fresh sugar beet pulp and moldy sugar beet pulp.

to mites (2+), mixed weeds (3+), and mixed molds (4+). He also had 3+ reaction to ground up sugar beet pulp pellets. In November 1988, he was skin test positive to house dust (4+), mixed grass (3+), plantain (3+), dog and cat epithelium (2+), and mixed trees (1+). He did not react to fresh or aged pulp, Aspergillus, or alternaria. His serum IgE was elevated September 1988 and November 1988 (533 and 294 IU/mi, normal ≤ 180). In December 1988 he had IgG/hypersensitivity antigen panel performed. He had a negative titer to *Micropolyspora faeni*, all thermoactinomyces, including *Thermoactinomyces sacchari*, and *Aspergillus fumigatus* (type 1, 6, and 1022). He had a positive titer (1:320) to *Aspergillus niger*.

RESULTS

Bronchoprovocation Testing

A methacholine challenge test on February 28, 1989 was normal, with a maximum drop in FEV_1 of 2 percent and FEF25-75 of 18 percent. On March 1, 1989, he was exposed to a control irritant dust, lactose. His maximum drop in FEV_1 was 4 percent and FEF25-75 was 11 percent at 7 h. On March 7, 1989, a methacholine challenge test was normal with all post challenges values greater than baseline. He was exposed to fresh sugar beet pulp on March 8, 1989. His maximum drop in FEV_1 was 3 percent and FEF25-75 was 7.5 percent at 7 h postexposure. A methacholine challenge on March 14, 1989 was normal with a maximum drop in FEV_1 of 3 percent and FEF25-75 of 10 percent. Exposure to clumps of moldy sugar beet pulp on March 15, 1989 showed a maximum drop of 9 percent for FEV_1 and 28 percent for FEF25-75 at 7 h. A methacholine challenge test on March 28, 1989 was normal with a drop of 7 percent in FEV_1 and 28 percent in FEF25-75. Exposure to ground-up moldy sugar beet pulp on March 29, 1989 showed a maximum drop of 23 percent in FEV_1 and 78 percent for FEF25-75 at 3 h. The patient also had a drop of 17 percent in FEV_1 and a drop of 42 percent for FEF25-75 at 40 min. The patient complained of chest tightness after this challenge that lasted through the night. Figure 1 shows the FEV_1 results of the lactose, fresh, and ground-up moldy sugar beet pulp challenge tests.

DISCUSSION

To our knowledge, this is the first report of occupational asthma in the beet sugar processing industry. The combination of the temporal association of the patient's symptoms with work, positive methacholine testing, positive skin test with moldy beet sugar pulp, positive IgG titer for *A niger*, and positive bronchoprovocation testing to moldy but not fresh beet sugar pulp support the causal association.

Previous investigators have found specific serum IgG to beet extracts in seven of 13 and specific serum IgG to bacterial extracts in five of 13 beet sugar workers.[4] In that investigation, fresh sugar beets were taken from the beet slicing area (fresh sugar beet) to prepare the antigen for testing. In contrast, we were unable to demonstrate specific IgG or IgE antibodies to fresh sugar beet pulp. Our patient reacted to moldy sugar beet pulp (skin test), had specific IgG to *A niger*, and reacted on challenge testing to the moldy and not the fresh sugar beet pulp. Additionally, aspergillus antigen was found in the rotted pulp. Accordingly, we believe that the causal agent for our patient was a fungus or fungal product in the moldy sugar. Occupational asthma has been reported previously with exposure to *A niger*. Exposure occurred at a biotechnology plant producing citric acid by fermentation of molasses with a strain of *A niger*,[5] a pharmaceutical company making cellulase from *Trichoderma viride* and *A niger* cultures,[6] pharmacy workers handling flaviatase, a proteolytic extract of *A niger*[7] and in bakers using the powder enzyme additives, glucoamylase and hemicellulase, both of which are made from *A niger*.[8] It is possible, however, that something in the sugar beet extract as tested by Forster et al[4] could also cause allergic symptoms.

Although the index patient had some systemic symptoms (*ie*, headache, fatigue, joint stiffness) there was no evidence of bagassosis (hypersensitivity pneumonitis). His roentgenogram was repeatedly normal, there was no documentation of rales or decreased FVC, either in his medical record or after bronchoprovocation challenge, and he tested negative for specific IgG to *Thermoactinomyces sacchari*, the etiologic agent for bagassosis. Hypersensitivity pneumonitis has not been reported in the sugar beet industry but only in sugar cane processing.

The potential for growth of thermoactinomyces and/or a specific fungus is probably related to geographic and temperature factors. In a study of workers from sugar cane processing facilities in India, 11 percent had skin test reactions to *Aspergillus fumigatis*, 5 percent to *Aspergillus flavus*, 4 percent to *A niger*, and 1 percent each to *Aspergillus nidulans* and *Aspergillus terreus*. Fungal spore counts varied between sugar processing mills.[9]

Given the large amount of organic material processed at beet sugar facilities, there is the potential for respiratory disease. Measurement of serial IgG extracts to sugar beet and/or bacterial contaminants has been shown to decrease

with improvement in ventilation and dust control at the processing facility.[4]

The particular clinician who saw this patient participated in the Sentinel Event Notification for Occupational Risks (SENSOR) program[10] and received repeated encouragement to report patients with occupational asthma to the State Health Department. Similar programs exist in nine other states. Public health follow-up at the facility was important in the identification of the cause and management of this patient's asthma as well as in providing recommendations to the company on ways to improve ventilation controls and housekeeping practices. We would encourage other physicians to inform state health departments when they diagnose occupational disease.

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Direct Extension of Bronchogenic Carcinoma Through Pulmonary Veins into the Left Atrium Mimicking Left Atrial Myxoma*

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Direct extention of bronchogenic carcinoma via pulmonary veins into the left atrium is rare. We describe two such cases, one which presented as a left atrial mass with pulmonary edema, and another which was detected at autopsy. ***(Chest 1992; 101:1722-23)***

Secondary involvement of the heart is relatively common at autopsy in patients with malignant tumors.[1,2] These lesions seldom manifest, are less frequently clinically significant, and very rarely are the presenting feature.[3,4] We present a case of bronchogenic carcinoma with direct extention into the left atrium via pulmonary veins, whose presentation mimicked that of a left atrial myxoma. On going through our autopsy records, a similar case, which was not diagnosed clinically, was encountered.

Case Reports

Case 1

A 30-year-old woman presented with a 15-day history of NYHA class 2 dyspnea, low-grade fever and cough. Her symptoms rapidly progressed to class 4 during over the next 12 days. She gave no history suggestive of acute rheumatic fever in the past.

Examination revealed a sick-looking, tachypneic patient with a pulse rate of 130 beats per minute and blood pressure of 110/70 mm Hg with prominent *a* waves in the jugular venous pulse. Auscultation of the chest revealed bilateral extensive crepitations and rhonchi, a loud first heart sound and a short grade 1/6 mid-diastolic murmur at the mitral area. The electrocardiogram disclosed no abnormalities except for a sinus tachycardia of 130 per min. The chest roentgenogram showed a cardiothoracic ratio of 50 percent with evidence of severe pulmonary edema (Fig 1). Two dimensional echocardiography revealed a mobile mass 5 cm in diameter in the left atrium prolapsing across the mitral valve during diastole. The

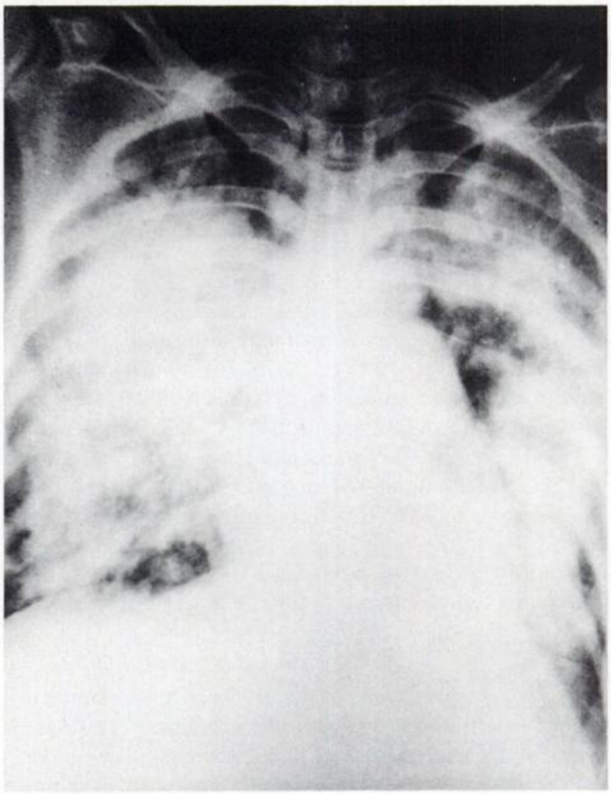

Figure 1. Portable chest roentgenogram, at the time of admission, showing severe bilateral pulmonary edema.