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Bronchial asthma-like attack after celiac plexus alcohol block

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Introduction

Major complications are infrequent during celiac plexus alcohol block and there are no reports of associated bronchial asthma in the literature. We have recently observed an asthma attack with acetaldehyde syndrome in a patient who underwent a celiac plexus block with ethyl alcohol.

Case report

A 65-year-old woman weighing 35 kg was scheduled for left celiac plexus alcohol block to reduce intractable abdominal and back pain from the recurrent esophageal cancer. She had been surgically treated with colostomy for intestinal obstructive symptoms, and received epidural morphine (5 mg/day) for about 1 month for pain relief, postoperatively. She did not receive any antibiotics or anticancer drugs. Preoperative laboratory data showed slight anemia, hypoproteinemia, and liver dysfunction. There was no history of either bronchial asthma or alcohol use. She did not receive epidural morphine on the day of the block. Under an image intensifier, two 14-cm 22-gauge needles were placed in the preaorta area at the T12 and L1 levels. Mepivacaine (2%) 5 ml and iopamiron 5 ml were injected, and the patient obtained satisfactory pain relief without signs of acute complications. Thirty minutes later, 20 ml of 99.5% ethyl alcohol was injected after a negative aspiration test. Five minutes after the administration, she complained of difficulty in speaking. Within 15 min, she experienced severe dyspnea and was given 100% oxygen under a mask in supine position. She developed severe expiratory wheeze and peripheral oxygen saturation (Spo_2) declined to 86%. At the same time, she complained of flushing throughout her body accompanied by hypotension [blood pressure (BP) 70/30 mmHg] and tachycardia [heart rate (HR) 140 bpm]. Aminophylline 250 mg and methyprednisolone 1000 mg were administered by bolus intravenous injection. About 10 min after this injection, the dyspnea improved, the wheeze disappeared, and Spo_2 increased to 96%. Lactated Ringer's solution 1000 ml was rapidly given with dopamine 10 μg/kg/min. A chest X-ray showed no sign of pulmonary congestion. An additional dose of aminophylline 500 mg was injected and she returned to the ward 2 h after the block. Hypotension and tachycardia lasted for 6 h in spite of intravenous fluid and dopamine infusion, and then she followed a satisfactory course without sequleae. (Three months after the block, the patient died due to respiratory failure from diaphragmatic invasion of the cancer.)

After the block, venous blood was sampled several times, and the concentration of acetaldehyde was measured. The acetaldehyde concentrations were (μM): 227, 294, 114, and 54 at 2, 4, 6, and 8 h after the injection of ethanol, respectively.

Discussion

The complications with celiac plexus alcohol block involve vascular injury, renal injury, paraplegia, local anesthetic toxicity, hypotension, and acetaldehyde syndrome (disulfiram-like reaction) [1–4]. The symptoms seen in this case were similar to the typical disulfiram-like reaction, which is characterized by facial or whole-

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body flushing, palpitations, diaphoresis, hypotension, tachycardia, vomiting, and dizziness [4].

The specific diagnosis is made by clinical signs and extremely elevated plasma acetaldehyde concentrations for about 6 h after the block [5]. Disulfiram-like reaction is known to occur also in subjects taking cephalosporine-type antibiotics, chloramphenicol, isoniazid, tolazoline, chlorpropamide, and carmofur [6–9]. This patient did not take such drugs. The most likely cause of the disulfiram-like reaction in this case was the relatively excessive dose of ethyl alcohol in a patient in poor physical condition. Additionally, she may have been aldehyde dehydrogenase (ALDH) I-deficient [10], a condition that exists in about one-third of all Japanese and shows a low Km value for acetaldehyde [11]. Unfortunately, we could not obtain hair roots from her head to examine the ALDH phenotype.

Asai and Watanabe [12] reported ethanol-induced bronchoconstriction, in which asthmatic symptoms deteriorated after intake of alcoholic beverages in Japanese asthmatic patients. Watanabe [13] showed that acetaldehyde (2 μM–100 μM) caused dose-dependent histamine release in leukocyte histamine assay in asthmatic patients; this suggested that histamine released from mast cells or basophils caused by acetaldehyde may play an important role in ethanol-induced bronchoconstriction in Japanese asthmatic patients. There was no history of bronchial asthma in the present case, but blood histamine levels might have been high when acetaldehyde concentrations were elevated after the block.

In summary, we experienced a patient who developed bronchial asthma probably due to elevated acetaldehyde levels after celiac alcohol block. The elevated blood acetaldehyde levels must have caused an asthma-like attack in this patient by a mechanism similar to that of ethanol-induced bronchoconstriction in Japanese asthmatic patients.

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