Hemomediastinum Induced by Heparinization after Stellate Ganglion Blockada

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(Key words: stellate ganglion blockade, hemomediastinum, heparin, myocardial infarction)

A patient who was treated for sudden deafness by stellate ganglion blockade (SGB) receiving 6 to 8 ml of 0.25% bupivacaine complained of thoracodorsal pain after seven sessions of SGB. Considering that the patient had a prior history of percutaneous transluminal coronary angioplasty (PTCA) for a very recent episode of myocardial infarction (MI), the pain was attributed to restenosis of coronary arteries. This was concidered a case of hemomediastinum induced by heparinization.

Case Report

M. M.: A 64-year-old female

Chief complaints: Deafness in the right ear and tinnitus

Past history: Appendicitis at 33, Diabetic mellitus at 57 (instituted on insulin), MI at 64 (diagnosed and PTCA was performed at 25th June, 1990)

Family history: Many family members and relatives were obese and hypertensive

Present illness: Around 25 July, 1990, she complained pain in the right auricle and noticed deafness in the right ear on July 29 when she could not hear the person on the other end of the telephone line.

She was diagnosed by a neighborhood doctor as having sudden deafness in the right ear and was referred to the Department of Otorhinology of our university hospital on July 31. After referral she was started on various drugs including steroid and vasodilator by intravenous drip infusion and SGB with 6 to 8 ml of 0.25% bupivacaine as well in compliance with request of Otorhinologist. On August 7, she was admitted to the Department of Otorhinology for the purpose of a workup.

As mentioned in the Past History, the patient developed MI in June 25, 1990 and underwent PTCA on same day. Later she was started on ticlopidine hydrochloride and child Bufferin®.

The hearing level in her right ear steadily improved until the sixth session of SGB.

Around 10 o'clock on the morning of August 8, bleeding was noted by the suction test during drug infusion with a 23G needle in the course of 7th session of SGB. The needle was withdrawn after infusion of about 3 ml of 0.25% bupivacaine, and astriction applied. After 30 min. neither bleeding nor swelling was noted at the site of puncture so that the patient was allowed to return to her ward.

About one hour and a half after the 7th sessison of SGB the patient

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J Anesth 6:367–371, 1992

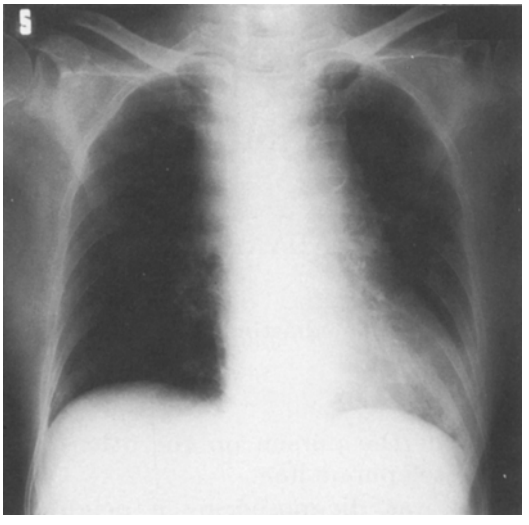

Fig. 1. Chest X-ray: Mediastinal enlargement was recognized about two hours after the heparinization.

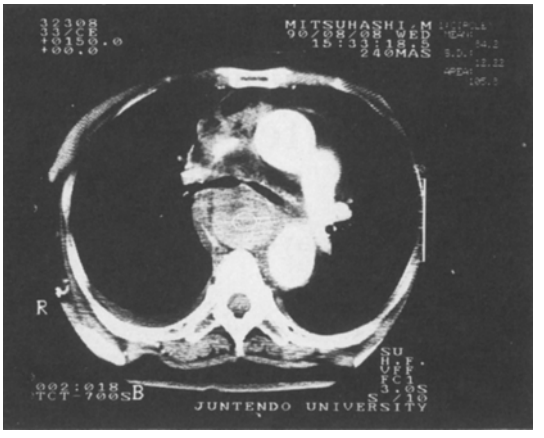

Fig. 2. CT findings: High-density diffusion defect was seen in the mediastinal region about two hours after the heparinization.

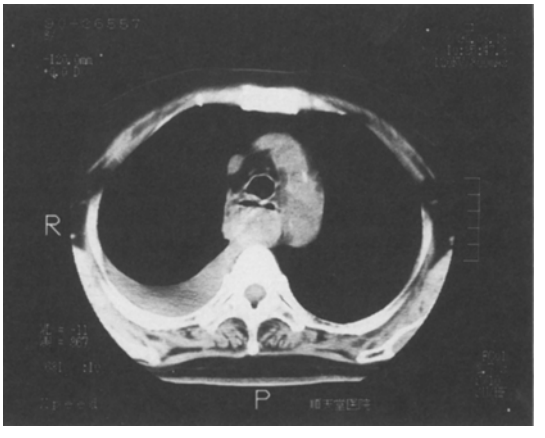

Fig. 3. CT findings: The absorption of about half the thoracic hematoma was seen at 10 days later. No paticular problem was noted at this time.

complained of pain spreading from the site of puncture over the back.

Because of a prior history of MI she was suspected of having angina pectoris. Sublingual nitroglycerin tablets were administered and isosorbide dinitrate tape applied at 11:20 and 12:00 AM, but the symptom was not modified. On consultation, a cardioangiologist entertained a suspicion that the patient had post-PTCA restenosis of coronary arteries. Thereupon therapy was instituted, administering 5,000 units of heparin and 10 mg of morphine hydrochloride at 1:00 PM.

The pain was not modified at all, however. On the contrary, sudden swelling appeared in the neck after heparinization, and the patient complained of shortness of breath. When contacted after the occurrence of these event, the anesthesiologist informed the carrying physician that the patient bled during SGB. She was then given 3 ml of protamine at 2:30 PM.

Pulse rate and blood pressure were normal. ST-segment depression in leads V_5 and V_6 was observed on the electrocardiogram, but this change was so light that its relationship to the pain was precluded. The possibility of angina pectoris was also ruled out based on the results of hemological tests.

Since mediastinal enlargement was seen on chest X-ray at 2:00 PM (fig. 1), CT examination was made at 3:20 PM. Judging from the high-density diffusion defect on the CT scan in the mediastinal region ranging from the retroesophageal space at the level of

the ligual bone through the trachea and dorsal side of bronchi to the dorsal side of the left atrium and ventricle, a clinical suspicion of hemomediastinum was entertained (fig. 2).

Arterial blood gas tension was not remarkable, but the bronchial bifurcation was deformed presumably under the pressure of hematoma, as manifest on the CT scan. Just in case of dyspnea respiration was managed thereafter through endotracheal intubation. The patient had clear consciousness, but with her informed concent a sedative was administered to induce sleep, and she was placed on controlled respiration overnight.

The loss of blood was estimated at about 1,000 ml from the CT findings, but Hb was 11.2 $g \cdot dl^{-1}$ and the subsequent change was so light that blood transfusion was dispenced with.

In order to avoid adverse effects of medications on cardiac function, hemostasis was achieved with carbozochrome alone, and antibiotic was administered to prevent infection.

When a CT scan was done the day after onset of hemomediastinum, that is, August 9, the respiratory tract was under reduced pressure from the hematoma, and the thoracic pool was found to have moved to the posterior region of the right thoracic cavity.

Spontaneous respiration was sufficient, and no particular problem was noted. On the evening of the same day the endotracheal tube was removed, and oxygen was administered through nasal cannulas. Arterial blood gas tension was normal after removal of the endotracheal tube.

The subsequent course was favorable in all respects, and absorption of about half the thoracic hematoma was seen on the CT scan done 10 days later (fig. 3). Hearing had made a sufficient recovery. Systemic conditions including blood sugar were all brought into control so that the patient was discharged on September 2.

Discussion

Cervical and mediastinal hematomas as complications in SGB are very rare, and a search of literature reveals only a paucity of information concerning this subject[1,2]. On the other hand, nerve blockade in patients on anticoagulation therapy has been surrounded by controversy because of a risk of hematoma as a complication, but no definite conclusion has been reached yet. The fact is that nerve blockade is practiced on patients with a hemorrhagic tendency, if absolutely necessary, exercising extreme caution and carefully observing them[3–5].

Reported complications of hematoma imputable to heparinization are divisible into spontaneous events[6–15] and disorders arising from nerve blockade[16–18]. The incidence of hematoma is frequent in elderly patients as can be inferred from their peculiar condition indicating heparin therapy. Hematoma due to heparin therapy have occurred in patients averaging 68.1 years in age, with a range of 50 to 86, except one 21-year-old patient[7].

Considering that our patient was diabetic and received ticlopidine hydrochloride and child Bufferin® after PTCA, the possibility cannot be ruled out that she had brittle blood wall and diminished platelet function.

Her bleeding time, thrombotest, activated partial thromboplastin time, and prothrombin time were all normal after the development of hemomediastinum, but her record contained no information about the hemorrhagic tendency at the time of hematoma development.

The patient had received six sessions of SGB before the onset of an episode. By the account of her family she often complained of pain in the neck and back after returning home. The

thoracodorsal pain that triggered the episode appeared to be imputable to the stimulation by SGB itself, but the possibility could not be precluded that more or less bleeding occurred, and it was obvious that the blood vesel was punctured.

Besides being bullnecked, the patient had physical changes peculiar to persons on a impatient diet (e.g., body weight decrease from 70 kg to 54 kg within 3 month), so her neck looked flabby and hard to search cervical transverse process by loosing of the tissue, and her chart indicated difficulty encountered in SGB. These findings, when considered together, suggest the possibility that SGB gave rise to complications such as vascular puncture. The injured vessel seems likely to be the vertebral artery. When a vessel is injured in SGB, hematoma generally occurs in the neck and is accompanied mainly with shortness of breath[1,2]. It appears uncommon that hematoma develops in the direction of the mediastinum.

As a result, the symptom was complained of as thoracodorsal pain, and as the patient unfortunately happened to have undergone PTCA, the anesthesiologist was not contacted promptly. This led to the mistaken suspicion that the patient had angina pectoris so that heparin was administered.

The dose of heparin used in our case is by no means high, compared to reported doses that gave rise to hematoma[6–18]. It seems a fact, however, that a blood vessel was injured to some extent. Furthermore, the patient also received an antiplatelet agent and had brittle vessel wall. It appears obvious from the course of the symptom as well as from these facts that heparinization prompted the growth of hematoma.

Shortness of breath was not obvious in this case, and the main symptom was pain. In case of the risk involved respiratory depression due to the continued growth of the hematoma such preventive measures as endotracheal intubation and controlled respiration should be taken. Judging, however, from Hb and the CT scan done the next day, the patient seemed unlikely to have dyspnea so that the endotracheal tube was removed in about 24 hours.

The movement of the thoracic pool to the posterior region of the thoracic cavity that was seen on the CT scan seemed to have occurred by gravity. It appears that this change reduced the pressure on the trachea and prompted the absorption of the hematoma.

Despite this accident, the hearing level which tended to improve in the early course of treatment made a recovery without aggravating. Ironically enough, this result may be attributable to the administration of ticlopidine hydrochloride.

In summary, a case of hemomediastinum induced by heparinization after SGB was reported. It is not uncommon that the patient treated by SGB experience cervical or thoracodorsal pain. Our patient had complained also of pain in the neck and back after sessions of SGB. It is to be regretted that the thoracodorsal pain could have been controlled without resorting to heparin if the caring physician had contacted the anesthesiologist sooner.

(Received May 30, 1991, accepted for publication Jan. 8, 1992)

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