Case Reports

Diaphragmatic Flutter with a Manifestation of High Frequency Ventilation

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A case of diaphragmatic flutter following status asthmaticus is presented. The initial symptom was tachypnea resembling hyperventilation syndrome. When spontaneous ventilation was completely suppressed for 15 minutes with intravenous administration of pethidine, levels of arterial blood gases were not much deviated from normal limits, because of possible high frequency ventilation. Although the medical therapeutics previously reported in literatures, including diphenylhydantoin, were ineffective in this case, butylophenones provided favourable results despite of some complications. The etiology and management of diaphragmatic flutter are discussed.

Key Words: Diaphragmatic flutter, High frequency ventilation, Dirhythmic breathing, Hyper-ventilation, Butylophenone

CASE REPORT

A 15-year-old high school girl was admitted to the Tokai University Hospital on October 13, 1980, because of tachypnea. She had been asthmatic since her childhood. In the end of September 1980, she was admitted to a local hospital with severe status asthmaticus. Although she improved well with a treatment of steroid, she developed intractable tachypnea. Therefore, she was transferred to this hospital.

On admission, this debilitated girl was 161 cm tall and weighed 54 kg. Her physical examination was essentially normal except her breathing which appeared extremely rapid like a “locomotive”. Minute ventilation measured with a water sealed spirometer was 24 liter with a respiratory rate of 82 per minute. Arterial blood gases with room air revealed pH 7.605, PaCO2 18.6 torr, and PaO2 124 torr. Other laboratory studies including chest X-ray were unremarkable.

Her respiratory rate increased gradually and reached maximum 180 per minute during the first one week of hospitalization (Figure 1), though this tachypnea usually disappeared during her sleep. Rebreathing of carbon dioxide, Valsalva maneuver, 10 cm of continuous positive ambient pressure (CPAP) breathing, and an acupuncture which is suggested to treat hyperventilation gave little effects. She complained of dyspnea and numbness on the fingertips due to respiratory alkalosis only when her respiratory rate exceeded 120 per minutes. Administration of sedatives, such as 20 mg diazepam, induced temporary sleep which returned her breathing normal.

When she developed severe respiratory distress with respiratory rate of 180 per minute, two distinct waves were observed on the spirogram (Figure 2). One was larger, slow irregular rate of 10 to 15 breaths per minute with a tidal volume 1500 to 2000 ml (shown “1” on Figure 2-A), and the other was smaller at 180 per minute with 200 ml of tidal volume (shown “s” on Figure 2-A). When 35 mg pethidine was administered intravenously she became apneic. The larger wave disappeared soon and the smaller wave diminished its tidal volume less than 90 ml then. However, she did not become cyanotic (Figure 2-A).
At that time, arterial blood gases revealed pH 7.456, PaCO2 34.6, and PaO2 92 torr.

On 24th October, 1980, she developed severe tachypnea. When 20 mg of diazepam was slowly injected intravenously, she became drowsy and her respiratory rate returned slow (Figure 3). Subsequently, she started to have visual abdominal muscle spasms, mainly located in her epigastrium, which became occasionally strong enough to shake her bed. Therefore, possible diaphragmatic flutter was suspected. Cinefluorography showed typical diaphragmatic flutter with a rate of 240 per minute on both hemidiaphragms while respiratory rate was 120 per minute. Diaphragmatic flutter was observed only when she was awake (Figure 4-A). It usually disappeared with a sleep (4-B), but appeared again with a wake (4-C).

Phrenic nerve block gave an only temporal cessation of the flutter, but the flutter of the intercostal muscles persisted. Halothane inhalation ceased flutter completely, but only during her sleep. Diphenylhydantoin gave also little effects even when its serum level reached 1.9 mg/dl. A bolus of intramuscular injection of 5 mg haloperidol resulted in brephalotremor and subsequent sleep. After seven hours sleep, she noted that she could not look downward because of fixation of her eye balls. However, it was found that her diaphragmatic flutter was completely disappeared. Since then, intramuscular administration of 7.5 mg droperidol three times a day suppressed flutter with minimum side effects.

She was discharged in early January, 1981,
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Fig. 3. On diazepam 20 mg, the patient became drowsy, but relieved from respiratory distress. When she became able to breathe normally strong vibration was felt on the abdomen. Even when she held her breath, the spirometer recorded irregular vibration.

Fig. 4. Overnight monitoring of breathing pattern. A: awake, B: asleep, C: drowsy state in the morning. From the top to the bottom, a: thermistor on the nostril, b and c: strain gauge pneumograph on the chest wall and abdomen respectively, d and e: electric encephalogram, f: electric oculogram, g: electric myogram on the hyoid muscle, and h: ECG.

Diaphragmatic Flutter and has been followed at the outpatient clinic. Although she has had occasional flutter lasting for a few to 30 minutes, she appeared to tolerate to regular activity with an oral dose of 2 mg haloperidol three times a day.

DISCUSSION

Three types of breathing patterns have been reported in the cases of diaphragmatic flutter\(^1\)\(^-\)\(^3\). The first is tachypnea. The second is dirhythmic breathing superimposed with high frequency waves\(^3\),\(^4\). The third is diaphragmatic flutter associated with apneustic respiration\(^6\).

All of these patterns were demonstrated in this case. Diaphragmatic flutter began to develop gradually soon after she was awake in the morning (Figure 4-C). It was dirhythmic breathing. As the flutter became larger, the original rhythm was replaced by that of diaphragmatic flutter. Since flutter was not so large in the morning, the respiratory rate was relatively slow in this period. But it became more than 180 per minute by the evening. Although no clear apneustic period was demonstrated, the original breathing rhythm seemed to have lost its regularity when diaphragmatic flutter became larger (Figure 2-A). It is speculated that repetitive stimulation of vagal stretch receptors or chest wall proprioceptor by diaphragmatic flutter suppresses the reflexogenic spontaneous respiratory rhythm, and the respiratory muscles start to synchronize with diaphragmatic flutter\(^8\). As shown on the cinefluorogram, the rate of respiration is not necessarily the same with that of the flutter. The respiratory muscles synchronize one half or one third of the diaphragmatic flutter depending on the intensity of the flutter.

It was of interest that the patient could keep normal blood gas level for 15 minutes despite her spontaneous ventilation was completely suppressed after administration of 30 mg pethidine. Diaphragmatic flutter created 16.2 liter per minute ventilation with 3 Hz and 90 ml of tidal volume which was less than her anatomical dead space of 140 ml. Therefore, it can be postulated that her exchange of alveolar gas might be due to accelerated diffusion of gases under high frequency ventilation\(^9\).
It is generally accepted that a diaphragmatic flutter derives from the disorder, either in central nervous system or at peripheral phrenic nerve. Since bilateral diaphragm and other respiratory muscles were involved, the origin of this case was most likely central. The two major disorders in central nervous system are explained as a cause of this disease. One is organic disorder, such as encephalitis of viral origin[4,14], and the other is psychogenic factors such as hysteria[12]. Flemister reported as case of dirhythmic breathing pattern associated with chronic obstructive lung disease and acute respiratory failure[6]. That case could be a result of the impaired ventilatory function, hypoxia and hypercapnia. Also, myoclonus due to brain hypoxia are known as Lance-Adams syndrome[10]. In severe respiratory failure, respiratory center receives impulses which induce an increased respiratory rate. These impulses arise from central or peripheral chemoreceptors as a result of abnormal blood gas levels. They may arise from mechanical receptors in the lungs or chest wall, or overworked respiratory muscles working under disadvantageous length-tension conditions, either via mechanoreceptors or via chemoreceptors. If the respiratory center simultaneously receives two independent impulses which demand high and normal rates of breathing, the center may conceivably respond to these independent stimuli by generating two distinct respiratory rhythms simultaneously.

This patient had no history of encephalitis or other disorders to induce organic lesion in central nervous system. Severe status asthmaticus preceded diaphragmatic flutter in this case. Furthermore, she was psychologically very immature, especially dependent on her mother. Therefore, isolation from her mother during long hospitalization can be assumed as a factor of her disease. Hypoxia, hypercapnia and obstructive ventilatory impairment could have existed at that time. It can be assumed that these factors appear to be responsible for this case.

The management of this patient was very difficult even after a diagnosis was made. Diphenylhydantoin is one of choice in this disorder[5]. However, dosage of 300 mg per day (serum level of 1.9 mg per 100 ml) for 10 days was ineffective. In order to suppress the respiration, several sedatives, such as nitrazepam, diazepam, and chlorpromazine, were administered without desired effect. These sedatives, however, induced temporary drowsiness with slight improvement of symptoms. Dissociation of the rhythms between diaphragmatic flutter and respiratory muscles by administration of 20 mg diazepam might be due to the decrease of conscious level (Figure 2).

Butylophenone is thought to have similar pharmacological actions with chlorpromazine and stronger respiratory depressant. Therefore, this has been used to treat myoclonus as well. We hoped this drug to reduce respiratory rate. However, an intramuscular administration of this drug 5 mg ceased diaphragmatic flutter completely though patient had some unfavourable side effects. Droperidol can be given by parentally, but has less side effects than haloperidol which can be given in a tablet. Initially, we could not use haloperidol because of side effects as described, but gradually these side effects disappeared by reducing its dose and adding of 100 mg promethazine hydrochloride.

As a conclusion, we suggest that butylophenone might be worth trial before surgical intervention of the phrenic nerve.

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REFERENCES
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