ACUTE PULMONARY EDEMA—CASE REPORT *

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ACUTE pulmonary edema is a dramatic condition occurring as paroxysmal attacks or as a terminal affair. It is always serious and demands immediate and unfaltering action. It is frequently a fatal event in the course of many diseases, both acute and chronic, and is commonly interpreted as the immediate cause of death (1). Regardless of the nature of the disease upon which acute pulmonary edema is interposed, once the activity of the edema reaches a certain critical point, it proceeds very rapidly, and it becomes progressively difficult to reestablish the normal physiologic state (2). When acute edema of the lungs develops, therefore, there can be no delay as to diagnosis and treatment if the therapy is to be effective. It is with this in mind that we wish to review some pertinent facts concerning this condition, and to discuss a fatal obstetric case recently encountered, which offers points of interest and instruction.

The pulmonary or lesser circulation may be looked upon as a counterpart of the greater circulation. The minute vessels of the lung, unlike the vessels of any other area of the body, are surrounded by, and practically in contact with, air. The blood must flow through the alveolar wall, while the respiratory exchange takes place, without change in fluid or solutes. As Weiss (2) contends, it is indeed remarkable that pulmonary edema does not develop under stress more frequently.

Acute pulmonary edema is not infrequently a problem in the obstetrical patient. Some authors state that it occurs in almost one-third of all eclamptic victims, and the proportion is greater in eclampsia which terminates fatally (3, 4). A study of cases reveals that acute pulmonary edema occurs more frequently in eclampsia associated with convulsions. It has been reported in all stages of pregnancy from the second month through to term, during delivery, and in the puerperium (3). Its occurrence cannot always be explained on the basis of left ventricular failure or valvular disease. The strain of labor added to a weakened heart musculature, with or without the presence of a valvular lesion, is, however, a definite factor.

Teel, Reid and Hertig (4) reported six cases of acute edema of the lungs in nonconvulsive eclampsia. None of these patients had findings which warranted a diagnosis of valvular heart disease, but four may

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have had previous hypertension, coronary disease or nephritis. In one case the edema developed postpartum.

ETIOLOGY

Many authors discuss the actual mechanism involved in the production of pulmonary edema, but the pathogenesis is still not completely understood.

In 1876, Welch (5), experimenting on rabbits, explained the clinical symptoms of this condition as a disproportion between the working power of the left and right ventricles, with a consequent back pressure in the blood vessels of the lungs. However, experimental data by other workers during the succeeding years gave evidence that the validity of this theory can no longer be considered as established (6, 7).

A second theory suggests a neurogenic basis as the pathogenic factor in the formation of pulmonary edema. Farber (8), and Weiss (2) believe that the underlying mechanism in neurogenic pulmonary edema is a disturbance in the vasomotor system, either peripheral or central, with secondary changes in the functional capacity of the heart, in the greater and in the pulmonary circulation. In this sense, it is induced indirectly by neurogenic factors. Hess (9) concluded that the cause of edema of the lungs is related to both the vagus and the sympathetic innervation of the lungs.

The classification of the conditions in which pulmonary edema occurs is given by Cecil (10) as mechanical, infectious and toxic, although many cases do not seem to fall within these three groups. The mechanical group comprises those associated with changes in the pulmonary circulation caused by heart failure. Pulmonary edema involved in the infectious group occurs as a terminal complication for many infectious conditions, and diseases associated with retention of fluids in the tissues, such as beriberi, toxemias of pregnancy and glomerulonephritis. The toxic group includes those cases which follow such conditions as poisoning from gases, alcohol, morphine and epinephrine.

Another group described in the literature is associated with diseases of the central nervous system, for example, cerebral trauma, epilepsy and cardiovascular accidents. Still another group includes cases of acute pulmonary edema occurring in angioneurotic edema in which left ventricular failure is not at all implicated; also there are those found where the pathologic change seems much too slight to explain the condition.

It is in the older literature especially that instances of pulmonary edema associated with distention of some abdominal organ are mentioned (11). Hochrein (12) and Vigi (13) have more recently reported cases accompanying dilatation of the stomach and the esophagus. Bowers (14) presented a series of patients with acute gastric dilatation and stated that one later died of acute pulmonary edema. Luisada (11), in an extensive survey of the pathogeneses of pulmonary edema, main-

tained that rapid distention of a hollow viscus does not in itself cause edema of the lungs, though the importance of nervous reflexes suggests itself.

DIAGNOSIS

The first sign of approaching edema of the lungs is frequently a cough. There is a marked bronchorrhea with an abundant or scanty yield. The patient appears anxious, dyspneic, and cyanosis may develop early. The dyspnea increases to orthopnea, and the persistent cough produces frothy and often pinkish sputum. Signs of asphyxia may predominate with sudden hypertension. The pulse will weaken and unconsciousness with death ensue unless adequate therapeutic measures are instituted.

Chest examination reveals fine, subcrepitant, or bubbling rales with some impaired resonance. Roentgenograms will confirm the diagnosis.

The pulmonary edema caused by infections, chemical agents or nervous mechanisms is a primary intra-alveolar edema, and extensive bubbling rales are audible. The exudate in this case is high in protein. As a rule, pulmonary edema of cardiac origin is an interstitial or pericapillary edema, and there may be no rales associated with it. The fluid is transudate and low in protein (2).

TREATMENT

As soon as edema of the lungs develops, certain measures can be immediately instituted. Large doses of morphine and atropine have been recommended by hypodermic injection, and repeated as needed (15). Luisada (25) advocated the intravenous administration of sedatives and narcotics which depress the central nervous system, anatomic system and the smooth muscle fibers of the vessels (morphine, phenobarbital, chloretone, chloral, atropine and papaverine). Early venesection may be helpful. This blood will be replaced by tissue fluids in order to maintain a certain volume of circulating blood, and the right heart will be relieved (16). Tourniquets, applied to the extremities, can be used prior to or instead of venesection. Oxygen and helium should be given with positive pressure (17). The fluid accumulating in the respiratory tree must be removed lest the patient "drown" in his own secretions. Tracheal aspiration can be done blindly or by direct vision. If sufficient fluid cannot be removed in this fashion, bronchoscopic suction is many times a life saving measure (18).

CASE REPORT

A thirty-five year old unipara, gravida IV, entered the hospital in labor of four hours' duration, and with the membranes ruptured. The blood pressure at the time of admission was 112 mm. systolic and 76 mm. diastolic. The pulse and respiration were normal. The first stage of labor lasted nine and one-half hours. The fetal position was right occiput anterior. A sample of the urine on admis-

sion was grossly contaminated so that the report received several hours later showed red, cloudy specimen, with 3000 mg. per cent of albumin, many red blood cells per high powered field, 30 to 40 white blood cells per high powered field and no casts.

Medication during the first stage included one ounce of castor oil, administered one hour and forty minutes after admission, and seconal, 3 grains (0.2 Gm.) five hours after admission. Four hours after admission, the head was engaged, and there was 70 per cent effacement of the cervix, with 5 to 6 cm. dilatation. The pains were strong, occurring every three to four minutes. The patient made unusually strenuous expulsive efforts with each pain, even before complete dilatation. Five hours and forty minutes after admission, a rectal examination showed complete dilatation and effacement of the cervix, and the patient was taken to the delivery room. Analgesia was started at 12:15 p.m. (five hours and fifty-five minutes after admission), using cyclopropane, 10 per cent, and oxygen, 90 per cent, by means of the closed system.

Atropine, 1/150 grain (0.0004 Gm.), was given by hypodermic injection at 12:40 p.m., and the patient was anesthetized with cyclopropane and oxygen. Mid-forceps were applied, and a female baby delivered at 12:43 p.m. The baby cried spontaneously. A large emesis occurred at about the time of the application of the forceps. The vomitus was described as dark brown in color and semiliquid in consistency. It filled three large emesis basins. The usual treatment, suction and Trendelenburg position, was instigated. The delivery of the baby and the repair of a small, first degree laceration were performed without further anesthesia. Ergotrate, 1 cc., was administered following the delivery of the placenta. The patient awakened, had good color, and responded intelligently as soon as the baby was born. After one hour in the delivery room, she was removed to her bed. During this entire period, her condition was considered satisfactory.

The patient coughed several times at 1:30 p.m., and her respirations increased. At 2:00 p.m. she became suddenly cyanotic. Chest examination revealed moist rales throughout, more pronounced in the left lower lobe. The cyanosis increased; she was dyspneic and coughed up pink frothy sputum. Her pulse increased in rate, but was regular.

Because of the profuse emesis while in the delivery room, an aspiration of vomitus was feared, although there had been no evidence of it at the time. Tracheal suction was immediately instigated by means of a rubber catheter, with several ounces of pink, frothy drainage. Nasal oxygen at 12 liters flow was started. The extreme cyanosis persisted and the patient had severe chills. The blood pressure at this time was 170 mm. systolic and 76 mm. diastolic, and the pulse was 140 per minute and bounding. Preparations were made for bronchoscopy because of the possibility of a massive atelectasis. Morphine, 1/6 grain (0.01 Gm.), and digifoline, 2 cc., were administered at 3:00 p.m. A roentgenogram showed the presence of edema of both lungs and a markedly dilated stomach (fig. 1). The heart was of normal size, shape and position. At about 3:30 p.m., the patient's respiration started to fail and the pulse was irregular.

A 7 mm. orotracheal tube was inserted by direct laryngoscopy and about 300 cc. of frothy, bloody fluid aspirated. Within twenty minutes, respirations and cardiac activity ceased. Intracardiac injections of adrenalin and alphalobeline were given; artificial respiration with oxygen and helium under pressure were continued until the patient was pronounced dead at 4:15 p.m.

ANATOMICAL DIAGNOSIS AT AUTOPSY

Autopsy was performed by Dr. E. R. Strauser, three hours after the patient's death.

Each pleural cavity contained 200 to 300 cc. of clear fluid. Aside from this, the lungs completely filled the pleural cavity. They were heavy and edematous. They floated even with the surface of the water. Microscopic examination showed marked congestion; the bronchial mucosa was pale; the bronchi contained no mucous plugs nor any foreign material, except watery, blood-stained fluid.

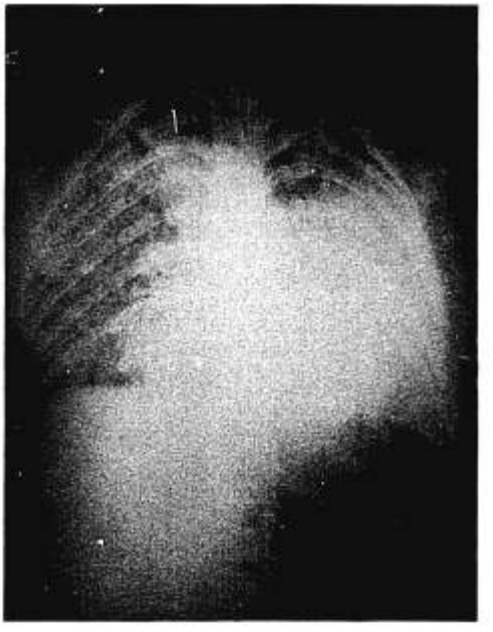

FIG. 1. A portable flat plate of the chest showing marked pulmonary edema and acute dilatation of the stomach.

The heart weighed 280 Gm. The valves were normal in size and appearance. The musculature of the left ventricle measured from 18 to 20 mm. in thickness, and that of the right, 4 mm. There was no apparent dilatation of the heart and there was no increase of pericardial fluid. The microscopic sections showed considerable edema, but no fibrosis. The cardiac muscle showed parenchymatous degeneration, and in portions, congestive edema.

The stomach was markedly dilated and filled about one-half of the abdomen. The diaphragm on the left had been pushed to the upper border of the fourth rib anteriorly. There was surprisingly little hemorrhage of the mucosa as a result of the distention. This possibly indicates a rapid dilatation which drove the blood out of the blood vessels before rupture could occur. There were no ulcerations.

Each kidney weighed 165 Gm. The epithelium of the convoluted tubules was swollen and showed parenchymatous degeneration. There were no significant changes in the glomeruli except for the congestion.

The liver weighed 1700 Gm. There was extreme parenchymatous degeneration. Swelling of the cells was so extensive that the sinusoids were nearly closed. The central canals and veins showed no important changes.

The mucosa of the duodenum was slightly engorged, but the bowel as a whole was normal. The mesenteric lymph nodes were not enlarged.

The postpartum uterus was contracted, and together with the dilated stomach appeared to fill the peritoneal cavity.

Summary of the Autopsy: Marked bilateral edema of the lungs; extreme dilatation of stomach; parenchymatous degeneration of the liver and kidneys; postpartum uterus; slight bilateral pleural effusion.

DISCUSSION

The cause of the acute pulmonary edema in the above case is not easily explained. Several factors must be considered.

There is the possibility of an irritation due to the aspiration of fluid vomitus. As stated in the case report, there was no evidence of this at the time of the emesis, and the onset of symptoms was one and one-half hours later. The autopsy examination revealed nothing to substantiate such a possibility.

Cyclopropane, the anesthetic agent in this case, has precipitated asthmatic attacks, and been the cause of acute pulmonary edema (19). These reactions, insofar as we know, have always been evident during the anesthesia—never one to two hours later. Cyclopropane is rapidly absorbed and eliminated. At the time of a patient's emergence from anesthesia, the concentration of cyclopropane in the expired air will not exceed 1 per cent (20). The patient in this case had only twenty-five minutes of analgesia with her pains, and two to three minutes during which she experienced a loss of consciousness. She was awake immediately following. The time element is contradictory to a cyclopropane allergy.

The marked albuminuria of the admission specimen, the significance of which is debatable because of the gross contamination with blood, was the only evidence of toxemia. All previous urinalyses had been negative. The autopsy report, showing parenchymatous degeneration of the liver, kidneys, and heart, also points to a toxemia of pregnancy. Terminal acute anoxia to the extent evidenced in this patient may, however, produce such changes.

The startling finding at the autopsy was the marked dilatation of the

stomach. By the process of elimination, we are faced with the probability that this was the primary factor in causing the acute pulmonary edema and death. That this can happen by a neurogenic mechanism has been mentioned. Reflex coronary vasoconstriction may be initiated by vagal irritation in the gastro-intestinal tract, thus causing a relative weakening of the left ventricle (21). In this case, the dilatation was so extreme as to cause, in addition, a mechanical embarrassment.

When did the dilatation of the stomach occur? The illustration indicates that it was present at the time the roentgenogram was taken two hours before death. Large quantities of dark fluid "poured from the patient's mouth" during delivery, showing that the dilatation developed before that time.

Experimental evidence shows that labor can occasion marked inhibition of gastric tonus through reflex inhibition of the vagus and stimulation of sympathetic nerves to the stomach, and it is not unusual for acute gastric dilatation to occur during delivery (14, 22, 23, 24).

Thus it seems reasonable to assume from the facts evolved in this case that the marked dilatation of the stomach played an important role in the sequence of events that followed.

SUMMARY

Acute pulmonary edema is reviewed with special emphasis on the etiologic factors. A fatal case, with autopsy findings and a discussion, is reported.

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