ACUTE ŒDEMA OF THE LUNGS.

BY
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Acute œdema of the lungs is a rare disease, first described by Laennec in 1819 as a fatal complication of measles in children. It is, however, of importance as it constitutes one of the emergencies of medical practice.

The clinical picture of the disease is striking, yet owing to the comparative infrequency with which it is encountered a correct diagnosis is not always made at first. In a typical case the patient who has previously been in good health is suddenly seized with dyspnoea, and within a short time becomes semi-unconscious or completely so. A sense of terror, pallor of the skin, and sweating are often noted in the early stages. There may be some preliminary warning just before the attack begins, such as pain in the head or neck, or a sense of faintness. The patient may then become unconscious, and the only noticeable features are the dyspnoea, slight cyanosis, and pallor. The cardiac action is usually regular, and the pulse of good volume and tension, but in some cases it is frequent and feeble. In a typical case, after a variable period of a few minutes to half an hour or so, a certain amount of frothy fluid runs up into the mouth from the lungs. This fluid is often stained pink, and continues to well up from the lungs into the mouth and nose as long as the attack persists.

Examination of the lungs in the early stages of the attack will give the clue to the correct diagnosis, for they will be found to be full of bubbling râles which extend from the base to the apex. These are caused by the air passing in and out of the alveoli and finer bronchi which are flooded by the exudation. The duration of an attack is very variable, death may occur within 10 to 15 minutes, or the patient may succumb after lying unconscious for several hours; in other cases recovery takes place after a variable period, and in some instances repeated attacks may occur.

Very little is known about the aetiology of edema of the lungs, and there are probably several contributory causes. Thus it may be associated with aortic disease, arterio-sclerosis, myocarditis, or chronic nephritis, and it may be met with in pregnancy, in diabetes, or in certain nervous conditions. In other instances it is thought to be a manifestation of angioneurotic œdema, or to result from such operations as paracentesis of the thorax or abdomen. Acute œdema of the lungs has also been recorded as a complication of acute infective diseases, such as typhoid fever, measles, influenza, pneumonia, and rheumatic fever, or it may occur as a terminal phase in pulmonary tuberculosis.

A Group of Theories.

The various theories propounded to account for the occurrence of acute œdema of the lung are mainly speculative and to a certain degree lacking in support. They may be grouped under certain headings applicable to the various clinical manifestations of the condition mentioned above.

The Cardiac Theory.—There is thought to be a disproportion between the working power of the left and right ventricle, so that the left expels a little less blood in a given time than does the right. The result is that stagnation occurs in the left auricle and the pressure in the pulmonary vessels is raised. This theory is based on the experimental work of Prof. W. H. Welch, who produced œdema of the lungs in rabbits by squeezing the left ventricle between the finger and thumb and partially paralysing it.

The Periaortitis Theory.—The inflammatory process is thought to spread from the aorta to the cardipulmonary plexus, with resulting increase of tension in the pulmonary vessels, and failure of the right ventricle.

The Toxic Theory.—This is advanced to explain those cases which are associated with chronic nephritis, pregnancy, and infectious diseases in which there is no cardiovascular lesion.

The Angioneurotic Theory.—The rapid onset of an attack in a patient who is apparently in good health, and in some cases the simultaneous appearance of œdema of the face, supports the theory that the pulmonary œdema may be in certain instances a manifestation of angioneurotic œdema.

Three Cases Described.

Three cases of acute œdema of the lungs have occurred in my practice during the last few months, and in view of the rarity of the condition a brief description may not be out of place. All were of the female sex.

Case 1 was a single woman aged 42, who had previously enjoyed good health. For a few months before the onset of her fatal illness it had, however, been noticed that she was somewhat irritable. Apart from this she was apparently in perfect health and, while having tea, she complained of a severe headache and in a few minutes became unconscious. Her breathing was embarrassed and was soon accompanied by audible bubbling sounds. After about an hour fluid began to well up into the mouth and trickled slowly from the nose and mouth. The fluid was stained pink. Examination of the chest showed that the lungs were full of bubbling râles. Her pulse was regular, 120 per minute, but there was no indication of a cardiac lesion, and the sounds at the apex were quite strong. The blood pressure was 120 mm. systolic. No specimen of urine was available for examination. The patient died three and a half hours from the onset without recovering consciousness, the lungs becoming completely waterlogged. Injections of morphine and atropine were of no avail.

Case 2 was that of a woman aged 37, who, on the day of her fatal illness, had just returned from a holiday, and
was, apparently, in good health. When sitting down to
dinner in the evening she complained of a tight feeling
across the forehead and neck; she immediately vomited
and lost consciousness within a few minutes. She did not
recover consciousness and died four hours from the onset.
In this case also the frothy fluid did not appear in the
mouth until one hour after the beginning of the attack,
the lungs became gradually waterlogged, the lower lung
showing more numerous râles than the upper when the
patient was lying on her side. The blood pressure was
slightly raised, being 140 mm. systolic, and there was
definitely hypertrophied. In this case also a specimen of
urine was not available, as the patient had passed it
involuntarily at the onset of the attack. When her rela-
tives arrived it was ascertained that she had had scarlet
fever three years ago and had been subsequently put on a
diet from which meat was excepted owing to high blood
pressure. The fluid continued to well up into the mouth
and nose as long as respiratory movements were made.
Morphine and atropane injections did not benefit her.

CASE 3 was a woman aged 62, who was suddenly taken
ill during the night after a journey to London. She
became very dyspeptic, cold and cyanosed, with a feeble
and rapid pulse, and the lungs were full of bubbling râles.
In this case consciousness was not lost and the pulmonary
cedema was not so severe as frothy fluid did not well up
into the mouth. Injection of morphine gr. ¼ and atropane
gr. 1/100 was followed by a wonderful result, the dyspepsia
and bubbling in the lungs being immediately relieved.
The next morning the lungs were clear, except for some
edema of the left lower lobe. Definite signs of cardio-
vascular degeneration were present, the heart being
enlarged, the arteries thickened, the systolic blood pressure
170 mm., and the urine contained much albumin. The
patient gave a history of an attack of acute nephritis
following a chill 18 years previously, and had suffered
from a septic throat five years before this. No difficulty
in swallowing had been noted until the last nine months,
during which the patient had had several minor attacks
of nocturnal dyspepsia.

The subsequent convalescence was notable for the
rapidity with which the albuminuria vanished. A small
pleural effusion developed on the left side, which was
absorbed without aspiration. The patient proved to be
an example of the recurrent or paroxysmal type of acute
pulmonary edema, which has been described by several
authors. Thus attacks have been known to extend over
a period of 10 years, and as many as 72 seizures during a
space of two years have been recorded. It has also been
noted that this recurrent variety is nearly always asso-
ciated with cardiovascular disease or with nephritis.
Almost a year later this patient had a second attack,
the lungs becoming flooded with fluid and the pulse-rate
rising to 132. The attack again occurred at about 2 A.M.,
and was aborted by an injection of morphine gr. ¼ and
atropine gr. 1/100. The urine was once more loaded with
albumin, whereas only a week previously it had been
examined and found free from protein. Vomiting occurred
several times during the following morning, and a small
pleural effusion again appeared on the left side with some
residual edema at the right base and a little dry pleurisy.

A week later the patient had a third attack which was
less severe than the previous ones, and again responded
well to morphine and atropane injections.

DIAGNOSIS AND TREATMENT.

At the onset of the attack the question of a
cerebral vascular lesion or coma from some other
cause naturally arises. As soon, however, as the
edema of the lungs appears and the fluid wells up
into the mouth there is little doubt as to the
correct diagnosis. In less severe cases in which
there is not such a copious exudation the presence of
bubbling râles in the lungs may suggest the
diagnosis of acute suffocative catarrh as opposed
to acute suffocative pulmonary edema. The
former is, however, an infective process, known
also as acute purulent bronchitis, and associated
with purulent expectoration and pyrexia.

There is no doubt that the most efficacious
treatment consists in the immediate injection of
morphine in doses up to gr. ¼. This is strongly in
favour of the disease having a cardiac origin.
Atropane gr. 1/100 is usually given in conjunction
with the morphine, although its use is probably
not essential. In cases which are likely to recover
the injection usually affords very rapid relief.
Other methods which are recommended are vane-
section, inhalation of amyl nitrite, and dry cupping.
It is very doubtful, however, if any form of treat-
ment will succeed if an injection of morphine and
atropine fail.

ABDOMINAL TUBERCULOSIS IN CHILDREN.*

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INFECTION of the abdominal cavity and its
contents by tubercle is apt to assume forms of
striking severity in the child in contrast to their
comparative rarity in the adult. In the latter
abdominal tuberculosis in a severe form is usually
a late event in the course of tuberculosis elsewhere;
in the child it is far more often the major or only
disability. Not only is this the case but it is also
true that in the child who manifests signs of tuber-
culosis elsewhere the oldest lesion is to be found
in the majority of cases in the abdomen. Experience
in the post-mortem room shows clearly that when
tuberculous bronchial glands or tuberculous invasion
of the lung are found in a child some involvement
of the abdominal contents is almost invariably
present. To find this it may be necessary to
examine closely any enlarged glands in the mesen-
tery and to explore the thoracic duct, in
such glands or in the lumen of this duct are often
to be found evidences of tubercle which remain
unrevealed to a hurried and casual inspection.

THE ROUTE OF INFECTION.

Whatever views may be held as to the route of
tuberculous infection as met with in the adult, there
can be no question that in the child the alimentary
tract is the common portal of entry. Apart
altogether from the danger of tubercle-infected
milk (a danger which is yearly becoming smaller
owing to quickening of the health-conscience
of the community), the floor-loving and toy-sucking
propensities of the young child expose it to the risk
of swallowing tubercle-infected dust at all hours of
the day.

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