RECURRING INTRACARDIAC CLOTTING ITS ASSOCIATION WITH ACUTE BRONCHIAL ASTHMA

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So little is known of the cause of sudden failure in a damaged heart that the striking features in this case prompt me to record it.

Past History of Case

The patient was a primipara, aged 23, without illness or known infection of any kind until the age of 16, when she had her first attack of acute bronchial asthma following a "chill." Attacks increased in frequency and severity, except during a recent pregnancy. Two and a half years ago a severe attack, typically asthmatic at its inception, rapidly developed into a heart crisis lasting twenty-four hours, when her life was despaired of. Her doctor said that her recovery was wholly unexpected and was almost as sudden as the onset; further, that the most interesting and puzzling feature of this sudden apyrexial illness was that she developed very definite rough valvular murmurs which cleared up in a short time. Soon afterwards she acted upon the suggestion that married life might cure her asthma, and the attacks actually did become less frequent and severe for a time, ceasing altogether during pregnancy. She felt perfectly fit throughout, and apparently had a normal but lengthy labour, lasting twenty-four hours, which resulted in the birth of a healthy live child weighing 8 lb. Thereafter, however, the old attacks recurred with renewed severity.

With great difficulty, and only after most searching questioning of her husband, was it possible, after her death, to elicit a definite history of failing heart, although neither he nor the patient herself had had any suspicion that her symptoms were due to an abnormal heart. Since her confinement she had had occasional stitches, with pain under the heart, attacks of palpitation, and an increasing distaste for effort. She had to "go easy," and often had to sit down and blow out her cheeks. Latterly she was nearly always tired, and for the last two months found stair-climbing almost impossible.

Recent History

One morning she had an attack of acute bronchial asthma. Her doctor gave her an injection, which eased her sufficiently to enable her to undertake a journey of twenty-five miles without undue discomfort. Throughout that day she was quite bright, took her meals, walked around a little, but felt tired. She had a very restless night, due to a return of distress in her breathing.

I saw her the next morning. She was sitting up in bed in the favourite position, leaning forward with arms on knees, wheezing like a typical asthmatic, her face pale and distressed, her lips slightly cyanosed, her pulse rapid and of poor tension. Cardiac dullness was obliterated, there was hyperresonance over both lung areas, inspiration was feeble and expiration prolonged, with the normal characteristics of both obliterated by sibilant and sonorous rales. A hypodermic injection of ephedrine and adrenaline eased the breathing almost immediately. Later in the day the wheezing returned, and a further similar injection with omnopon was given, following which she slept restlessly most of the afternoon. She apparently was dazed and confused on waking, and cried out in her disturbed sleep. These symptoms were put down to the effect of omnopon and exhaustion. She appeared to be very ill in the evening, but her father said that he had seen her worse, that she would be all right in the morning, and that there was no need to send for the doctor. At 9 p.m. she was able to sit up in bed and take a little supper. Later, according to her husband, some froth appeared at the corners of her mouth,

and only then did she say that she felt very ill. Still later the husband was suddenly awakened by a queer gurgling choking noise. The patient looked as if dying; her face was blue, she was gasping for breath, and was wildly throwing her arms about and struggling to get out of bed, saying, "It's wind." She was got back to bed in a convulsed state, with her arms and legs bent and stiff. Her breathing ceased almost immediately.

Post-mortem Findings

Lungs.—The pleurae were normal, while the pleural sacs contained no free fluid. The left lung was frothy and emphysematous throughout. The upper two lobes of the right lung showed a condition similar to that of the left, but the lowest lobe was dark, firm, and hard, and on section was like a recent blood-clot. There was much gelatinous mucus in the larger bronchi and lower trachea.

Heart.—No evidence of pericarditis was seen, but there was an excess of free fluid in the pericardial sac. The heart was pale, flabby, enlarged, and full of ante- and post-mortem clots. Ante-mortem clots were present in both chambers of the right heart; they were non-globular, fleshy, tough, and rope-like. That in the ventricle stretched the whole length of the main stem of the pulmonary artery to act as an embolus causing massive infarction of the right lower lobe. It had a wide extensive base which was firmly moored to the chordae tendineae of the tricuspid valve, and was separated piecemeal from them with some difficulty. The wall of the right ventricle was degenerated and fibrosed, and only a very thin rim of muscle tissue remained, while at the tip the whole wall was little more than one-sixteenth of an inch thick. The endocardium, valves, and blood vessels were healthy.

None of the other organs or the systemic blood vessels showed any pathological change. There was no oedema of the extremities.

Discussion

The course of events would appear to have been as follows. A primipara aged 23 suffered for seven years from recurring attacks of acute bronchial asthma. The subject of serious and unsuspected cardiac damage which gave rise to none of the usual physical signs of heart failure, she survived the incidence of an intracardiac thrombus, and two years later had a normal pregnancy and labour. She died unexpectedly during or following an attack of acute bronchial asthma, her death, which was preceded by an attack of acute suffocative pulmonary oedema, being ultimately precipitated by embolic pulmonary infarction resulting from extensive intracardiac clotting.

The post-mortem findings made it clear that the patient had had in the past, probably in infancy, some acute infection, causing myocarditis (but not endocarditis), which gave rise to no direct physical signs, probably not even a disturbance of rhythm, but which later produced indirect evidence of cardiac damage and of the heart's increasing inefficiency in a recent limited response to effort associated with dyspnoea and palpitation, these urgent symptoms probably being due to increased rate exhausting the contractile force of the heart with diminution of the output. The labouring heart always dilates, dilatation being the physiological answer to an increased call for effort, and, heart failure having set in, the chambers do not completely get rid of their contents, so accumulation and stasis takes place. Any sudden severe strain, such as an acute attack of asthma on a failing heart, like the one under discussion, would greatly aggravate and accelerate this stasis and would produce ideal conditions for intracardiac clotting.

The seeming lack of diagnostic acumen is easily explained: in the absence of a history of heart infection or even of the more usual physical signs of heart failure in

an unknown patient who had had a normal labour four months previously, one would be most unlikely to suspect, much less be able to assess, the heart's efficiency during an attack of acute bronchial asthma of any severity. It must be noted, too, that the subjective signs of a failing heart, although definite, were elicited only after the patient's death, and even then with the greatest difficulty.

In reaching the conclusion that asthma was the factor which ultimately precipitated death, a few questions presented themselves for satisfactory explanation. (a) Was it possible for the patient to have had a normal labour of twenty-four hours' duration four months previously if her heart was in such a damaged and dangerous condition? (b) If her heart was in so grave a condition, surely there would have been some very definite physical signs? (c) Can ante-mortem clotting take place in the heart in the absence of valvular disease or endocarditis? (d) Were her attacks those of true *bronchial* asthma? (e) What is the evidence that she survived an attack of intracardiac clotting two years previously?

In answer to the first three questions Sir James Mackenzie (1917) said that he repeatedly saw individuals in the greatest danger whose serious condition had never been suspected, because there was present no physical sign indicating heart trouble, but if the patient's reaction to effort, as measured by her own sensations, had been investigated and appreciated, the gravity of the condition would have been recognized; that many cases of sudden death may be due to acute oedema, usually associated with mitral stenosis, but that there may be no valvular lesion; and that he was often unable to account for such a death. Further, he stated that many women with serious organic heart trouble seem little the worse for their pregnancy and that they may bear their confinement well, yet the effect of pregnancy on a diseased heart is that its strength is lessened permanently.

That the patient's attacks of acute asthma were of the bronchial type cannot be doubted, as they responded repeatedly to ordinary anti-asthmatic remedies, were typically bronchial in appearance and behaviour, and were relieved by the endocrine readjustment of pregnancy. In the severe sublethal attack of two years ago, which also began as acute bronchial asthma and which very soon became a cardiac catastrophe, it would appear that the superimposed heart signs and symptoms were due to intracardiac clotting. The heart signs appeared very rapidly, the patient all but died within a few hours, the attack was apyrexial, recovery was very rapid, and following it appeared very definite and rough valvular murmurs which cleared up with incredible speed. It is probable that the murmurs were due to an organized thrombus either in the right auricle or in the right ventricle—more likely the latter—moored to the chordae tendineae of the tricuspid valve (as described in the post-mortem findings), and that the rapid disappearance of the murmurs was the result of the rapid organization and absorption of the thrombus. Moreover, the fatal attack started in an exactly similar way, and the post-mortem findings gave a very positive explanation of the course of events in this final attack.

Summary of Conclusions

A fatal case of intracardiac clotting precipitated by an attack of acute bronchial asthma and followed by massive embolic infarction of the lung in a young primipara is described.

There is evidence that two years previously the patient survived intracardiac clotting which also resulted from bronchial asthma.

Intracardiac clotting may take place in the absence of endocarditis and may give rise to the signs of a gross valvular lesion.

A seriously damaged heart may, even with the strain of pregnancy and a lengthy labour, show no direct physical sign of failure.

REFERENCE

Mackenzie, Sir James (1917). *Principles of Diagnosis and Treatment in Heart Affections*, 1st ed., pp. 50, 122, 184, 212, Oxford Medical Publications.

ACUTE PSYCHOSES FOLLOWING SURGICAL PROCEDURES

BY

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In the *British Medical Journal* of January 28, 1939 (p. 162), Dr. Barber describes a case of acute mental illness from a gangrenous appendix with abscess formation, and refers to such a mental manifestation as being "very rare" in surgical conditions. The case was interesting and instructive, but I believe it would be more instructive still were it realized that such a state is by no means a rarity.

In Erdington House (the Birmingham Mental Observation Wards) we see from time to time acute psychotic patients of this type admitted from surgical wards. There is a history, more or less common to them all, that the patient has had surgical treatment for some acute condition—for example, appendicitis, strangulated hernia, severe wounds—and that in a varying number of days after an apparently satisfactory operation or other procedure there develops an acute mental state characterized by restlessness, confusion, noisiness, impulsive violence, resistance to all nursing attentions, and occasionally by fleeting hallucinations and delusions of a persecutory nature. Removal to the mental observation wards becomes necessary.

On admission the above mental state is easily confirmed. But in addition there is a fairly obvious general physical condition of toxæmia—rapid pulse, slight pyrexia, hot dry skin, malar flush or sallow sunken features, dry brown furred tongue, sordes on teeth, etc. The entire ensemble of symptoms and signs, both physical and mental, gives, in other words, the characteristic picture of a toxic psychosis. With this clinical picture one knows from experience that there will almost invariably be found either a large collection of pus under an apparently healed wound or else that pus already discharging is not gaining a sufficiently free outlet; and that vigorous measures to this end will produce a rapid and satisfactory result. The following four typical cases are illustrative.

Illustrative Cases

Case 1.—A man aged 23 underwent herniotomy on February 25, 1937. On March 3, after apparently normal progress, he became violent, noisy, and difficult, and was transferred to Erdington House the next day. On admission he was dull, morose, suspicious, and restless, and complained of pain in the wound, which was seen to be tense and swollen, the surrounding tissues being inflamed and indurated. With hypertonic fomentations there began on the 8th a most profuse and vigorous discharge of thick, foul greenish-brown pus. By the following day the temperature had subsided; he was quiet and brighter, and expressed great relief. On the 10th he was returned to the surgeon with paralytic ileus, from which he also recovered, and at no time was there any recurrence of mental symptoms.