it is very useful in those cases which show signs of vagus hyperactivity, but it is a remedy against the promiscuous use of which ship surgeons should be on their guard.

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ACUTE PHLEGMONOUS GASTRITIS IN PREGNANCY

BY

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Acute phlegmonous gastritis, also known as suppurative linitis or as submucous gastritis, is a rare disease, of which only some 200 cases are recorded in the literature, and none of these in association with pregnancy. The following case is reported because of the rareness of the occurrence of the condition in pregnancy and because of the difficulty in diagnosis to which the combination may give rise.

Case History

Mrs. A., a 3-para aged 32, had always been in good health, except for occasional attacks of erache, sometimes accompanied by discharge. She had had two previous pregnancies, with normal spontaneous deliveries of live children, weights unknown. There had been no miscarriages. She did not attend any ante-natal clinic.

The patient was admitted on January 27, 1939, with a history of persistent vomiting for the past week. Temperature 97°; pulse 128; blood pressure 120/80. There were signs of extreme dehydration. Her tongue was dry, brown, and furred, and she appeared to have lost weight. The urine had a specific gravity of 1022, was acid, and showed a slight trace of albumin. Acetone and diacetic acid were present, but no blood or bile. The heart and lungs were clinically clear. The abdomen was enlarged to about thirty-two weeks' size, without rigidity or tenderness. The pelvis was not examined. The case was diagnosed as pernicious vomiting of pregnancy.

For the first twenty-four hours she was given plentiful alkaline fluids with glucose by mouth, but no diet. During this time she vomited several times, mostly clear bile-stained fluid. Next day she was much improved: temperature 98°; pulse 110; blood pressure 120/80. A little light diet was ordered, but she did not have any appetite. She vomited once or twice. On January 31 she was still vomiting, and there had been no improvement. She was therefore put on a continuous rectal saline, containing 10 per cent. glucose and in addition pot. brom. 30 grains to each pint. She was also allowed sips of water by mouth. The vomiting became less,

and she slept well throughout the night. On February 1 salines were continued. There was no vomit, and her condition had improved. Blood pressure 120/70; pulse 90. The following day a profuse purulent odourless discharge came from the right ear. The patient was seen by the otologist, who diagnosed a chronic streptococcal otitis media. She was therefore immediately transferred to a surgical ward. Her general condition improved whilst away, but she continued to vomit at times. The ear condition cleared up, and she was sent home on February 24.

On February 27 she was readmitted at 10.30 a.m. She was then vomiting copiously, was collapsed, and looked very ill, haunted, and anxious. Her tongue was dry and furred, with sordes on lips and teeth; she had sunken eyes, and was restless, and complained of severe right upper abdominal pain. A slight aural discharge occurred from which a pure culture of a short-chained streptococcus was grown. The vomit was clear bile-stained fluid with no smell. Temperature 101.2°; pulse 140; blood pressure 105/65. On examination she was very tender over the right hypochondrium, especially the gall-bladder area. There was some guarding and rigidity of the upper right rectus muscle. No mass was palpable. At times the uterus appeared to be contracting, but this was difficult to decide because of the extreme tenderness of the abdomen. The foetus was palpable and the foetal heart heard in the R.O.A. position (rate 140). Per rectum the cervix was one finger dilated. There was a slight blood-stained loss per vaginam. The urine was clear except for a slight cloud of albumin: acetone and diacetic acid were present. The stools were normal, and the blood pressure was 100/60.

The differential diagnosis seemed to lie between acute cholecystitis and retroplacental haemorrhage. The height of the fundus was marked, and she was put on a half-hourly pulse and temperature chart. At 12 noon one-quarter grain of morphine was given, and was repeated in half an hour. Alkaline fluids with glucose by mouth were ordered, and continuous intravenous drip saline with 5 per cent. glucose was given into the antecubital vein. At 6 p.m. the fundus of the uterus had risen about two fingers above the level at which it had been marked at midday. The uterus was extremely tender to the touch and was of board-like hardness all over, as was also the whole hypochondrium now from side to side. The foetus was no longer palpable, but the foetal heart could still be heard. The morphine was repeated in the evening and at 11 p.m. The patient slept fairly well throughout the night, but in the morning was still very ill, with a running pulse of 160 to 170. The diagnosis of concealed accidental haemorrhage seemed the most likely one, and the question of Caesarean hysterectomy was considered, but owing to her extremely bad condition it was decided not to open the abdomen.

During the night and from the previous afternoon the patient had had about seven pints of fluid intravenously. About midday she showed signs of cardiac embarrassment and the drip was discontinued. Coramine 2 c.cm. was given, and the pulse steadied and regained volume. Later in the afternoon, at 4 o'clock, she was given 350 c.cm. of blood by the indirect citrate method with the addition of coramine 4 c.cm. Her condition improved. Morphine was repeated. At 11 p.m. her condition was still very grave, and the blood pressure 90/50. 300 c.cm. blood, with coramine, was repeated. The patient did not seem to respond, and she died at 3.30 a.m. on March 1, complaining to the end of acute pain over the stomach. Progressively, the abdomen appeared to become more distended and rigid.

Post-mortem Examination

The uterus and its contents were normal. There was a considerable peritoneal exudate, and in the upper abdomen the peritoneum was deeply congested and coated with flakes of lymph. The peritoneal coat of the stomach was deeply congested and the whole stomach wall swollen and firm. On section the wall of the stomach was increased in thickness to about half an inch, and this change was generalized, although most marked in the region of the pyloric antrum.

The stomach wall was infiltrated with thin pus, which oozed from the cut surface. The duodenum was not involved. The mucosa of the stomach was thick and intensely congested, so that it presented a generalized deep red coloration. The other abdominal organs were normal.

In smears made from the pus from the stomach wall a short-chained Gram-positive streptococcus was demonstrated in large numbers. As mentioned in the clinical history, there was present an otitis media, the discharge from which contained a streptococcus morphologically identical with that found in the stomach wall. Microscopically, section of the stomach showed the muscle coat widely infiltrated with polymorphs, together with fibrin and necrotic tissue; the liver and kidneys revealed post-mortem changes only.

Discussion

The first case of acute phlegmonous gastritis was reported in 1656 by P. Borel, and since then some 215 cases have been made available. Its association with pregnancy is not recorded. The condition is a suppurative cellulitis of the submucosa of the stomach, usually widely diffused but sometimes more or less localized. The causative organisms have been in most cases streptococci, but pneumococcus and colon bacillus have been found. It is thought that in most cases the mode of entry of the organism is through the mucosa of the stomach, either at the edge of a pre-existing ulcer or through some lesion not visible to the naked eye; but in those cases complicating other acute infectious diseases such as typhoid, pneumonia, and puerperal infections the infection may be blood-borne.

In the case here reported the infecting organism was a streptococcus, and from the standpoint of pathogenesis two explanations are forthcoming. The condition may have arisen as a spreading infection from a minute lesion of the gastric mucosa due to the effect of repeated vomiting, which was part of the hyperemesis syndrome. The streptococci must be assumed to have passed from the ear via the Eustachian tube to the mouth and into the stomach. On the other hand, the infection may have been blood-borne, the focus of origin being in the ear, and the localization in the stomach being due to a lowering of local resistance consequent upon excessive vomiting.

The clinical features in the non-pregnant include the rapid onset of severe upper abdominal pain, vomiting, abdominal rigidity, and rapidly progressive prostration due to severe toxæmia. The temperature is raised, the pulse rapid, and signs of dehydration appear. Recovery from such a condition is rare, and in the generalized variety it is unknown. If the condition be localized and an abscess form in the stomach wall, recovery has followed rupture of the abscess into the stomach, and also surgical incision. Diagnosis in the non-pregnant is difficult and has rarely, if ever, been made before exposure of the stomach. The condition closely resembles perforative lesions of the stomach or duodenum, acute pancreatitis, and acute cholecystitis. In the pregnant woman diagnosis is further confused by the presence of the enlarged, hard, tender uterus with its suggestion of concealed accidental haemorrhage.

In the pregnant woman acute abdominal pain, with uterine tenderness and rigidity, vomiting, and increasing prostration, may be associated with a varied underlying pathology, and in our own experience such diverse lesions as acute phlegmonous gastritis, spontaneous rupture of the spleen at term, general peritonitis following suppuration of an ovarian dermoid cyst, spreading peritonitis following perforation of a gangrenous appendicitis, and red degeneration of large fibroid have been met with. In all these conditions there is a peritoneal reaction, the

effect of which is to cause abdominal tenderness, which may be impossible to distinguish from uterine tenderness. In addition the uterus in the presence of peritonitis may take on a board-like rigidity indistinguishable from that due to a concealed accidental haemorrhage. This is a most important diagnostic point, since in conditions of peritonitis or severe peritoneal irritation the continued board-like rigidity of the uterus may well lead to a diagnosis of concealed intra-uterine bleeding. This uterine rigidity coincident with peritonitis is not always present, and in some cases the examining hand will detect periodic relaxation of the uterus. In our experience, however, a continued rigidity without any relaxation will often be found in conditions of generalized peritonitis.

In such cases, however, it will be noted that the foetus is still alive, and, although it may not be palpable owing to rigidity, the foetal heart can be well heard—a finding which must throw serious doubt on the diagnosis of concealed accidental bleeding. In peritonitic conditions, except in their terminal stages, the temperature is raised and gross signs of pregnancy toxæmia are absent. In late peritonitis, however, the temperature may be low, the pulse running, and the urine scanty and albuminous.

There is a further feature of interest, and so far as we know not recognized in the literature. In the presence of peritonitis or severe peritoneal irritation not only does the uterus become rigid and tender but the level of the fundus definitely rises in the abdomen. Whether this is due to a straightening out or erection of the uterus coincident with the continued contraction, or whether it is due to a hyperacute hydramnios consequent upon uterine hyperæmia, we have no means of deciding at present. In the case here reported, and in which the initial diagnosis was concealed accidental haemorrhage, the level of the fundus was carefully marked at 12 midday. By 5 p.m. the level had risen one and a half inches. At the time of making the original measurement of the fundus there was not continued uterine rigidity, but five hours later, when the fundus had risen, the uterus was uniformly rigid and tender. We have seen the fundus rise four inches in twelve hours in a case of peritonitis due to a suppurating ovarian dermoid in a pregnancy at thirty-six weeks, and the same sign has been observed in a case of appendicitis with spreading peritonitis.

Cases of pregnancy with associated acute suppurative intra-abdominal lesions are uncommon, and must lead to diagnostic uncertainty. The occurrence of this sign in three instances in our experience suggests that it may be of some use in diagnosing the nature of the causative lesion in these cases, and serves to emphasize the fact that a rigid, tender uterus with a rising fundus does not necessarily point to a diagnosis of concealed accidental bleeding.

The life of the patient may well depend upon avoidance of this mistake, since such a diagnosis leads to conservative treatment and morphine, while suppurative intra-abdominal lesions generally require active treatment and laparotomy.

Summary

A case of acute phlegmonous gastritis associated with pregnancy is placed on record, possibly for the first time.

The clinical features of acute phlegmonous gastritis are described and the difficulty of diagnosis is stressed.

The further difficulty arising when the condition occurs in pregnancy is discussed.

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