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Spontaneous Rupture of the Stomach

A most uncommon condition which usually produces a fatal, acute abdominal emergency is spontaneous rupture of the stomach. This term is applied when there is no history of external trauma and no pathological change, which could predispose to the rupture, is to be found in the stomach wall. Albo, de Lorimier and Silen in 1963 were able to find 43 recorded cases. Further cases were reported by Olsen and Foley (1961), Shaldon (1961), Foex (1962), Davis, Andersen, Akre and McCarthy (1963), Mirsky and Garlok (1965), and Fleming, Horna and Wagget (1966).

This paper presents a case which is unusual, not only because the patient survived, but also because he suffered simultaneously from mental disease.

Case report

Mr. J. R., a manual worker aged 41 years, was admitted to the Second Surgical Clinic of the Medical Academy, Lublin as an emergency on 20th Nov. 1964. Six days previously, he had entered the psychiatric ward because of mental deficiency. The past history was one of episodic pain for a few years in the right, upper quadrant of the abdomen with fullness and distress following meals. Occasionally, he developed nausea and vomiting. On the day of admission, a barium meal was administered. X-ray examination of the upper gastro-intestinal tract revealed no evidence of a stomach ulcer but showed delayed emptying of the stomach. After 2 hours, there was a gastric residue of approximately 80 per cent with distribution of the remaining barium throughout the small bowel. Later, half an hour after eating, while sitting in a chair, he developed a severe, upper abdominal pain which rapidly increased in severity and extended throughout the whole abdomen.

On admission. — The patient was obviously in great pain and had cold, cyanosed extremities. His respiration was rapid and deep, B. P. was 90/50 mms. of mercury and the pulse, which was weak had a rate of 120 per minute. His

temperature was 38.9°C, and the whole abdomen was extremely tender and distended.

Clinically, a suspected presence of a perforated peptic ulcer was diagnosed and plain x-rays of the abdomen were taken. These showed the stomach to be considerably dilated. There was considerable gaseous distension of the bowel and there was a suggestion of free intraperitoneal gas as shown on the lateral film. A nasogastric tube was passed, and it was decided that operation was necessary although a definite, pre-operative diagnosis had not been made. Macrodex was given intravenously and whole blood transfused rapidly during the induction of anaesthesia. Under a general anaesthetic of oxygen, ether and fluothane, the peritoneal cavity was entered through an upper midline incision. The peritoneal cavity contained a large quantity of gas and a small amount of fluid pus but no obvious food particles. A markedly distended, hypertrophied stomach was observed. There was no evidence of gastric ulceration but surrounding induration was causing pyloric obstruction. Numerous extravasations and a few small areas of necrosis in the cardiac region were seen. Having opened the lesser sac, barium was revealed in this cavity. Further exploration of the upper stomach revealed a high-lying perforation located in the posterior wall 1 cm. below the cardia. A longitudinal tear, approximately 4 cm. in length was observed midway between the lesser and greater curvatures, parallel to the lesser curvature and some 5 cm. from it. The edges of the tear were smooth, there was no evidence of induration and subsequent histological examination of an operative biopsy taken from this site revealed no peptic ulceration. Since the stomach contained necrotic foci, a total gastrectomy was carried out and a retrocolic oesophago-jejunostomy was performed, with preservation of normal peristalsis, before anastomosing the proximal and distal segments of the jejunal loop. Anticipating a possible post-operative leak, a drain was placed in the left, subdiaphragmatic space. The post-operative course was uneventful. The patient gradually improved and was discharged on the twenty-second post-operative day feeling well. The patient has remained free of symptoms to date i. e. nineteen months after the operation.

DISCUSSION

Examination of the literature shows that spontaneous rupture of the stomach is not common. Spontaneous rupture of the stomach in the newborn has been described in isolated case reports, but it is considered to be predominantly a disease of early or middle adult life. It was suggested that overdistension plays a primary role in these cases and Davis and his co-workers wrote: "when overdistension occurs, the stomach wall may be affected by local circulatory changes resulting in secondary ischemia and weakness of the musculature" (3). The distension may be observed in patients who have taken sodium bicarbonate or eaten a large amount of food or as a result of pyloric obstruction due to chronic duodenal ulceration.

The stomach is not a fixed organ but it possesses a fixed inlet and outlet i. e. cardia and pylorus. Theoretically, one could visualise that closure of both exits by spasm or kinking would favour rupture. Usually, organic or non-organic obstruction of the oesophagogastric junction sug-

gests that the gas bubble, rapidly produced in the fundus by fermentation of the gastric contents is compressing the lower oesophagus. Sometimes this could result from the swallowing of air or, possibly, from gaseous transfer across the walls of distended capillaries. The powerful contractions of the abdominal wall during vomiting may also increase intragastric pressure sufficiently to produce a rupture. A few ruptures which occurred after labour were produced by severe retching rather than by the ingestion of food.

Perforations may be single or multiple. They may involve one or all the layers of the stomach wall, and most frequently occur in the cardiac area. Clinical diagnosis has always been difficult. These cases were frequently diagnosed as cases of perforated peptic ulceration. From a review of the literature it appears that the most significant symptoms consist of: 1) severe abdominal pain, 2) tympanic distension of the abdomen, 3) abdominal rigidity, 4) subcutaneous emphysema, and 5) evidence of shock. According to Davis and his co-workers, abdominal distension develops due to a pneumoperitoneum but according to Shaldon, it is due partly to the presence of a pneumoperitoneum and partly to gaseous distension of the stomach. The detection of emphysema is, above all, the factor which leads to the correct diagnosis on clinical grounds. Shaldon considered that when the air in the peritoneal cavity was under tension, subcutaneous emphysema may be expected to develop but, when the pneumoperitoneum was small, compared to the degree of gastric distension, emphysema would probably not develop. Mirsky and Garlock suggested that the absence of subcutaneous emphysema or peritonitis may be explained by immediate fixation of the tear to the parietal peritoneum, and this could be observed in the present case.

X-ray examination of the abdomen is an important diagnostic aid (3, 10). A plain x-ray of the abdomen shows an enormously distended stomach and a small pneumoperitoneum. Shaldon considered that a close inspection of the outline of the lesser curvature revealed a break in the continuity of the lesser curvature which anatomically would correspond to the site of the rupture, as seen at the operation.

There is, however, a lack of general agreement regarding therapy. Some authors favour simple closure of the perforation, in an effort to spare the patient a bigger operation (2, 3, 9, 10). Some perform a closure of the perforation and then vagotomy, with stomach drainage by a short loop with posterior gastroenterostomy (5), while some prefer a gastrectomy (6). Simple closure of the perforation is probably safest, but it must be remembered that people with pyloric obstruction subsequently require further operative procedures.

Definitive operation was essential in the present case. Because of pyloric obstruction and necrotic foci in the stomach wall, total gastric resection was necessary. This case also seems to be of value since the patient did not demonstrate subcutaneous emphysema which is often present and suffered from mental deficiency.

REFERENCES

1. Albo R., de Lorimier A., Silen W.: *Surgery* 53, 796—799, 1963.
2. Bromilski J.: *Pol. Przegl. Chir.* 28, 77—81, 1956.
3. Davis C., Anderson R., Akre O., Mac Carthy W.: *Arch. Surg.* 86, 170—176, 1963.
4. Fiedorczuk Z.: *Pol. Tyg. Lek.* 8, 889—890, 1953.
5. Fleming L. B., Horton J. A. G., Wagget J.: *Brit. J. Surg.* 53, 384—387, 1966.
6. Ganszer W.: *Pol. Tyg. Lek.* 7, 252—254, 1952.
7. Gross K.: *Pol. Przegl. Chir.* 26, 199—204, 1954.
8. Miller J. M.: *Pol. Tyg. Lek.* 4, 1219—1222, 1949.
9. Mirsky S., Garlock J. H.: *Ann. Surg.* 161, 466—468, 1965.
10. Shaldon C.: *Brit. J. Surg.* 48, 640—641, 1961.

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Samoistne pęknięcie żołądka

Streszczenie

Autorzy omawiają etiopatogenezę i klinikę samoistnego pęknięcia żołądka. Spośród czynników sprzyjających pęknięciu wymieniają przede wszystkim zmiany organiczne toczące się w żołądku oraz zaburzenia czynnościowe.

Autorzy przytaczają własny przypadek, w którym przyczyną pęknięcia żołądka było nadmierne jego wypełnienie barytem i pokarmem przy istniejącym zwężeniu odźwiernika.

Самопроизвольный гастрорексис

Резюме

В работе описывается этиопатогенез и клиника самопроизвольного гастрорексиса. Среди факторов, способствующих возникновению гастрорексиса, по мнению авторов, главную роль играют органические изменения желудка и расстройство его функций.

Авторы приводят наблюдаемый ими случай, когда гастрорексис был вызван избыточным наполнением желудка баритом и пищей при одновременном сужении его привратника.