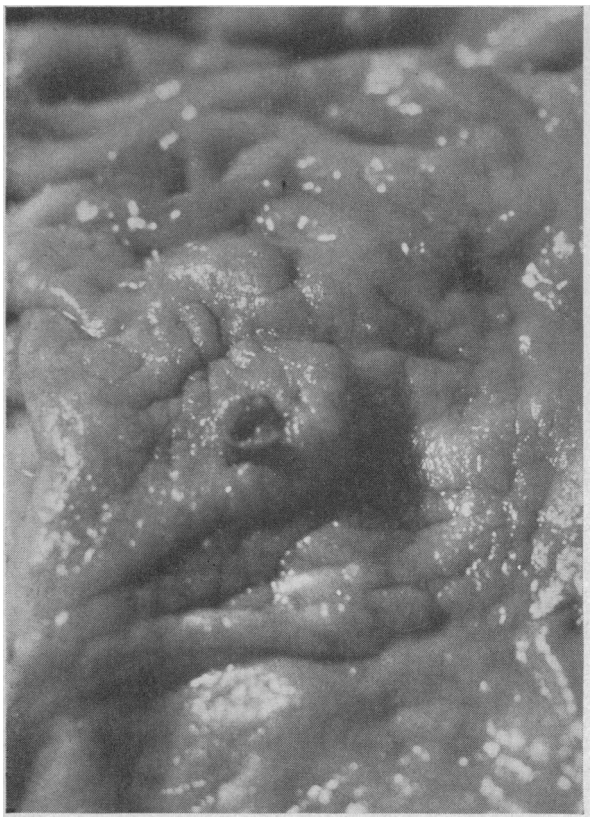

Figure 2.—Gross specimen showing the elevated 4 mm. defect in the gastric mucosa.

of the stomach wall was cut out and blocked for serial section. In cross-sections through the wall an underlying vessel about 4 mm. in diameter and lying very close to the margin of the ulcer was noted. No evidence of lesion was observed in macroscopic examination of the pylorus, duodenum and pancreas.

In microscopic examination of sections of the gastric lesion a small area of peptic erosion of the mucosa was observed. There was a dilated and tortuous artery in the submucosa of this region, and one loop of the vessel was prolapsed through the eroded zone, filling the mucosal defect. Weakened by peptic digestion, the wall of the artery was distended and ruptured. The cross-sectional contour of the vessel was that of a funnel with the wide mouth pointed toward the gastric lumen. There was practically no inflammation of the stomach wall beyond the small zone of acute erosion. No significant lesions were observed in sections of the other organs.

COMMENT

Other causes of massive hematemesis such as peptic ulcer, esophageal varices, gastritis, and carcinoma, have been adequately covered elsewhere.[5] During the final episode in this case, the results of studies previously carried out were not available. The presumptive diagnosis was bleeding esophageal varices.

Discussion of therapy is beyond the scope of this report. However, it might be pointed out that had blind subtotal resection, as advocated by some investigators,[2, 4] been carried out in this case, the lesion might still have been missed. Total gastrectomy done between bleeding episodes would have been necessary, unless reduction of acid secretion alone would have prevented rupture.

Jankelson and Milner[3] recently reported a follow-up study on 27 patients who had massive gastrointestinal bleeding of unknown origin. A correct clinical diagnosis was eventually made in nine cases, and in two others the source of bleeding was observed in exploratory operation. In 16 cases the cause of the hemorrhages was never discovered—in three cases despite a total of six exploratory laparotomies.

SUMMARY

A case of massive hematemesis in a 40-year-old man who was observed during the third (and lethal) hemorrhage in six years is reported. At autopsy, peptic erosion of a non-arteriosclerotic cirsoid aneurysm of the stomach was observed microscopically.

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Lipoid Nephrosis During and After Pregnancy

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LIPOID nephrosis is a rare complication of pregnancy. The following case history suggests such a diagnosis:

REPORT OF A CASE

A housewife, 26 years of age, was admitted to the medical service of Stanford University Hospital on May 28, 1947, with generalized edema of approximately three weeks' duration. She was six months pregnant. There was no history of preceding infection or illness and none of previous medication. The patient had had three earlier normal pregnancies, and her past health had been excellent.

Upon physical examination, pronounced generalized edema of the face, abdominal wall and extremities was noted. Both pleural cavities contained fluid, and ascites was present. The temperature and the pulse rate were normal. The systolic blood pressure was 110 mm. of mercury and the diastolic pressure 70 mm. Hemoglobin content of the blood was 11 gm. per 100 cc., plasma proteins 5.1 gm. per 100 cc. of blood, and the cholesterol level 1,000 mg. per 100 cc. of blood. The blood sedimentation rate was accelerated. The most significant observations in repeated examinations of the urine are summarized in Table 1. Urinary sediment was normal throughout the period of observation.

The patient was placed on a diet containing 2,500 calories and less than 3 gm. of sodium chloride. The protein content of the diet was 70 gm. From June 5 through June 16 infusions of 50 gm. of albumin, given in 200 cc. of water, with an added 200 cc. of 5 per cent glucose in water, were given daily.* This infusion was given over a period of three

*The albumin was furnished by the American Red Cross.

hours and there were no reactions. Diuresis was prompt; there was a decline in body weight from 69 to 57 kilograms and the edema cleared.

The patient was discharged and during the following month was given albumin at four- to five-day intervals, but there was a gradual increase in edema and in body weight and she was readmitted to the hospital on July 29, at term, with pronounced edema and body weight of 69 kilograms. A normal child was delivered July 30. There was no recession of the edema, and between Aug. 6 and Aug. 20, the date of discharge from the hospital, the patient received daily infusions of albumin, with excellent diuresis again resulting and a drop in body weight from 65 to 54 kilograms.

TABLE 1.—*Twenty-four Hour Urine Excretion Study*†

Date	24 Hr. Urine Volume (cc.)	Specific Gravity	Protein, gm. per 24 hr.	Chlorides (NaCl), gm. per 24 hr.
May 30	1,565	1.020	8.8	9.08
June 5	684	1.010	6.0	0.96
June 9	3,480	1.010	27.3	14.62
June 13	2,800	1.010	35.2	5.88
June 17	2,350	1.011	30.4	5.88
June 20	1,780	1.015	18.4	1.34
June 24	3,490	1.012	42.8	1.57
Aug. 7	4,000	1.011	43.8	15.5
Aug. 10	3,560	1.010	51.1	16.4
Aug. 17	3,700	1.012	40.0	0.54

†Dr. David Rytand of the department of medicine of the Stanford University School of Medicine supplied the data.

On Oct. 1, 1947, the patient was admitted to Providence Hospital, Oakland. Anasarca was again present and the body weight was 65 kilograms. There were no symptoms other than mild dyspnea, attributed to mild bilateral pleural effusion, and moderate ascites. The temperature was 37°C., the pulse rate 72, the systolic blood pressure 114 mm. of mercury and the diastolic pressure 76 mm.

The hemoglobin content of the blood was 10.6 gm. per 100 cc. Leukocytes numbered 9,500 and the cell differential was within normal limits. The specific gravity of the urine was 1.020, the reaction alkaline. The albumin content was 0.62 per cent by weight. Sugar was absent. In examination of the urinary sediment a few granular casts, erythrocytes and leukocytes were observed. The blood sedimentation rate was 18 mm. in nine minutes (Linzenmeier). The plasma protein content was 4.6 gm. per 100 cc. and the urea nitrogen content of the blood was 35 mg. per 100 cc.

The patient was placed on a diet low in sodium, containing 2,000 calories and 100 grams of protein, and was given ammonium chloride, 2 gm. by mouth three times daily. No attempt was made to restrict fluid intake. On October 3, 4, 6, and 7 the patient received 2 cc. of Mercuhydrin® by intramuscular injection. Diuresis was prompt, with a body weight decreasing 13 kilograms in five days. The patient was discharged from the hospital October 8, and from then until June 1950, when she moved to another city, she was observed at frequent intervals. During this period the low sodium diet was fairly well followed. Ammonium chloride and Mercuhydrin at weekly or bi-weekly intervals were required to control edema which tended to recur periodically. Iron, vitamin B complex, and desiccated thyroid were given by mouth.

In June 1948 the urine was found to be negative for albumin, and the blood sedimentation rate and the hemoglobin content had returned to normal limits. The patient was returned to a diet containing a liberal amount of salt and got along well for about five months. Then, following an infection of the upper respiratory tract, symptoms typical of nephrosis again developed. A prescribed diet, acidification, and mercurial diuretics were again started, with a less rapid but gradual improvement. One year later a similar episode occurred following a gastrointestinal disturbance. Protein hydrolysates were added to the diet and this time Mercuhydrin injections were increased to 4 cc. bi-weekly before satisfactory diuresis occurred. This larger dosage was continued for several weeks without evidence of toxicity. Improvement again followed. No treatment was given after December 1949, and when last observed on Feb. 2, 1951, the patient felt and looked well. The hemoglobin content of the blood was 12 gm. per 100 cc. and the serum protein level was 6.85 gm. per 100 cc. of blood. There was no albumin in the urine and the sediment was normal.

COMMENT

The patient was initially considered to have toxemia of pregnancy, and it was thought that the symptoms of nephrosis would clear following termination of pregnancy. This did not occur. Infusion of albumin was followed in each instance by satisfactory diuresis, but little if any influence on the level of serum proteins was noted. Albumin recovered in the urine in any given period corresponded roughly to that administered. Salt depletion, as measured by sodium chloride excretion in the urine, corresponded in general to water loss, and tended to be reduced as edema disappeared. Diuresis and reduction of edema were as well accomplished by the administration of acidifying substances and mercurial diuretics as by the administration of albumin. No toxic reactions were noted.

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Anserina Bursitis

Report of a Case

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ANSERINA bursitis is an uncommon entity in medical literature. Le Fort and Albert reported five cases, Le Bourge three, and Zodek one. Moschcowitz mentioned reports of 20 to 25 cases. Meyerding and Chapman have reported 14 cases from the Mayo Clinic. Because of the rarity of the disease, it is not often that an accurate preoperative diagnosis is made. The usual diagnosis is soft tissue tumor or cystic mass on the medial aspect of the knee. Oftentimes the mass is considered to be a malignant tumor because it may rapidly increase in size.

The anserina bursa is located on the inner aspect of the knee between the lower portion of the tibial collateral ligament and the tendons and insertions of the sartorius, gracilis and semitendinous muscles (Figure 1). These tendons insert by means of a fan-shaped aponeurosis (pes anserina) on the proximal portion of the medial surface of the tibia, and beneath this aponeurosis lies the anserina bursa, which is 3 to 6 cm. in length and 4 cm. in width.

The typical patient with anserina bursitis is a middle-aged male laborer with complaint of a swelling or tumor which has been increasing in size and is located on the medial aspect of the knee. Associated with this swelling is aching or burning pain which is made worse by motion.

On examination, a mass firmly attached to the lower medial side of the knee will be observed. It may feel quite solid when the knee is extended; but when the knee is flexed, relieving the tension on the ligaments, the mass will often become fluctuant. The swelling itself in most cases is not tender, and there is no stiffness of the knee joint although the knee may not be fully extended because of