different viruses—for example, vaccinia (Anderson, 1949; Whittle *et al.*, 1950), herpes simplex (Jaquette *et al.*, 1946; Brain *et al.*, 1950), possibly influenza (Leverson and Whitlock, 1949)—or in which the virus is not definitely known (cf. Van Rooyen and Rhodes, 1948, p. 299). It would seem reasonable to apply the more specific term of “accidental generalized vaccinia” or “Kaposi’s varicelliform eruption (vaccinal type)” to cases in which the infection (a) is acquired by contact with a vaccinated person, (b) is definitely due to the vaccinia virus, and (c) causes a pox eruption on an eczematous soil (Fries and Borne, 1949).

The unusual lung lesions showed the same basic pathological features as those in the skin. Elementary bodies were present in large numbers. Viral lesions in bronchial walls have been described in mice inoculated intranasally (Nelson, 1938) and in generalized vaccinia in rabbits (Douglas *et al.*, 1929; McIntosh and Scarff, 1929), but not in man. In spontaneous rabbit pox, on the other hand, lung lesions exactly similar to those described here have been depicted by Nicolau and Kopciowska (1931). While the lesions in the stomach were only at the early “vesicle” stage, and no cellular infiltration had developed, it is assumed that they also were primarily due to the presence of the virus elementary bodies.

Large numbers of cytoplasmic and intercellular elementary bodies—Buist or Paschen type (Gordon, 1937)—occur in the skin lesions of vaccinia, but, so far as can be ascertained, lung and stomach lesions containing these bodies have not been previously reported in man. The large eosinophilic inclusion bodies of Guarnieri are specific; in this case they were seen only in the skin lesions, and not in those occurring in the lungs or stomach.

Summary

A fatal case of accidental generalized vaccinia is described; the clinical history lays fresh emphasis on the ease with which an eczematous infant may develop generalized vaccinia. In addition to the skin eruption, lesions of a similar nature were present in the lungs and stomach.

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People sometimes do too much to help the blind, and increase their feeling of helplessness. Unwittingly they leave too little of the initiative with the blind person, says the *Annual Report of the Guide Dogs for the Blind Association*. But the guide dogs treat their masters as normal in everything except that they cannot see. They guide their masters, but do not think for them. Dogs enable many people to do jobs that would otherwise be extremely difficult for them—those involving travelling, for instance. For hundreds of years people have used dogs to guide them, but not until 1915 was any organized training begun. This was in Germany, and the British association was founded in 1933. In addition to the centre at Leamington, a new one has been set up at Exeter.

Medical Memoranda

A Case of Mumps with Involvement of the Lacrimal Glands

Involvement of the lacrimal glands in mumps must be very rare, although reference to it can be found in some standard works: “The lacrimal gland is rarely attacked” (Price, 1950) and “Vaugiraud and Defaux have reported a case of bilateral dacryoadenitis manifested by swelling and tenderness of the lacrimal glands, which developed three days after the parotitis and subsided at the same time as the inflammation of the parotid” (Rolleston and Ronaldson, 1940). Nevertheless it was with great surprise that we encountered the following case in which an undoubted bilateral dacryoadenitis coexisted with, and clearly formed an integral part of, an otherwise typical mumps syndrome.

CASE REPORT

On March 18, 1951, a woman aged 29 complained of general aches, malaise, and pains in the left side of the neck. Next day the aches had disappeared and she was able to do her housework in the morning. The pain in her neck persisted, however; in addition her left upper eyelid began to swell and she became conscious of two swellings, which she could feel with the tip of her tongue, in the position of the sublingual glands. By the same evening she had developed a frontal headache, which she described as very severe and “more than just a headache.” At this stage her neck felt “stiff” and her left upper eyelid began to swell. On March 20 the frontal headache remained severe, both upper eyelids were very swollen (but without erythema), and the neck was painful on both sides and diffusely “stiff.” The patient remained conscious of the swellings under her tongue. At this time the oedematous upper eyelids were the outstanding sign and the possibility of nephritis was considered.

On March 21 the eyes were almost closed by oedema and a notable erythema involved the lids of both eyes, ceasing abruptly at the eyebrows and spreading downwards on to the cheeks, where it faded away without line of demarcation. The erythema did not straddle the nose or involve the forehead or neck, and it was not indurated or tender. The headache had by now disappeared. The pain in the neck remained, and there was also pain in both ears and underneath the right upper eyelid. On this day the patient was admitted to hospital as a presumptive case of facial erysipelas. Besides the facial appearances, tender smooth enlargements of moderate degree were found discretely affecting the parotid and submaxillary glands of both sides; there was also a moderate degree of trismus, and the tongue could not be protruded more than just to show the tip—owing, it appeared, partly to the mechanical effect of the sublingual swellings and partly to their tenderness. The dorsum of the tongue was dry and brownish and there was pronounced halitosis. The temperature on admission was 100.4° F. (38° C.) and the urine was not found to contain albumin or sugar. On March 22 the patient began to feel better, the temperature fell in the evening (subsequently remaining normal) and the oedema of the eyelids began to decrease.

On March 23 a blood count gave haemoglobin 90%; colour index, 1; red cells, 4,500,000; white cells, 9,500 (polymorphs 75%, lymphocytes 20%, large monocytes 4%, eosinophils 1%). On this day the upper eyelids could easily be lifted and a swelling could be discerned under the outer part of each; pressure on this swelling through the lid definitely caused pain—at least on the right side—and the examining finger left a pit in the subsiding oedema. Underneath each upper lid was a small amount of sticky yellowish exudate. The patient was seen by an ophthalmologist, Mrs. Dorothy Campbell, on March 23, who noted: “Acute

adenitis of lacrimal glands with local conjunctival injection. Sclerae white. No signs of iritis." Rapid recovery ensued, and by March 28 all symptoms and signs had disappeared.

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An Unusual Case of Acute Pancreatitis

The following case of a woman aged 39 who died of acute pancreatitis on the 37th day of her disease presented two unusual features: (1) the appearance, in the early stage of the disease, of a syndrome which was indistinguishable from diabetes mellitus; and (2) death from intra-abdominal haemorrhage.

CASE REPORT

The patient was admitted to hospital on January 9, 1950, with the following history. For two years she had been subject to repeated attacks of severe epigastric pain with some flatulence, the symptoms being unrelated to food and not relieved by taking more food or alkaline powders. On January 5 she had an extremely severe attack of this indigestion. There were no further definite symptoms, although she felt unwell, until three days later, when, after a normal bowel motion, she developed severe upper abdominal pain and vomited. The pain and vomiting continued until her admission. She admitted to some backache when questioned, but could not localize it to either the lumbar or the interscapular region. When the urine was found to be loaded with sugar and acetone she was questioned about a history of loss of weight, of polyuria, or of polydipsia. She denied ever having had these symptoms.

On examination she was pale, anxious, and moderately shocked: blood pressure 130/105, temperature 98° F. (36.7° C.), pulse 120, respirations 25. Her tongue was dry and furred. The normal respiratory movements of the abdomen were diminished; there was tenderness and guarding over the upper abdomen and slight tenderness over the lower abdomen; she felt a sharp pain on the release of pressure over the whole abdomen; there was no diminution of liver dullness; the abdomen was silent on auscultation. Rectal examination disclosed acute tenderness in the pouch of Douglas. Examination of the respiratory and cardiovascular systems revealed no abnormality. Urine: brick-red with Benedict's solution; acetone +++ (Rothera's test); chlorides, 3 g. per litre (Marriott's test); diastatic index (result not received until after laparotomy), 250 units. Blood chemistry: sugar, 258 mg. per 100 ml.; urea, 93 mg. per 100 ml.; chlorides, 468 mg. per 100 ml.; protein, 5 g. per 100 ml. Radiography showed no evidence of gas under the diaphragm.

Because the clinical features were so much more like a perforated peptic ulcer than an acute pancreatitis and the diastatic index was not available, it was decided to do a laparotomy. An intravenous infusion of normal saline was started, and 10 units of soluble insulin was given together with 30 g. of glucose intravenously. On opening the peritoneal cavity sero-sanguineous fluid escaped. Numerous areas of fat necrosis were seen on the peritoneum and the great omentum. The gall-bladder contained at least one large calculus. A diagnosis of acute pancreatitis having been made, a drain was placed in the lesser sac and the incision closed.

The patient made satisfactory progress for the first 10 days. On the morning after operation the blood-sugar level had fallen to 153 mg. per 100 ml. A further dose of 20 units of soluble insulin covered by 50 g. of glucose intravenously was given. The urine was tested with Benedict's solution every four hours, and, apart from one orange result on the third post-operative day (this was associated with a blood-sugar level of 267 mg. per 100 ml.), the test

persistently gave a green or blue result. A glucose-tolerance test was done on the 13th day after operation. The result was as follows:

Time	Blood-sugar Level	Urine
Fasting	90 mg. per 100 ml.	No sugar
½ hour	175 " " "	"
1 " "	205 " " "	"
1½ hours	174 " " "	Sugar "+++
2 " "	202 " " "	" "+++

The whole-blood chlorides remained within normal limits.

During the first week there was a purulent bronchitis, but up to the 10th post-operative day the patient's general condition gradually improved. On this day a thick yellow discharge from the drain began, and she started to go downhill. She lost her appetite and began to waste rapidly. The nature of the discharge soon altered, portions of necrotic pancreas being seen. The discharge began to digest the abdominal wall. Continuous suction was applied to the drain, and this proved very effective in protecting the abdominal wall. A severe anaemia was associated with the rapid wasting. On the 14th post-operative day the haemoglobin was 10.56 g. per 100 ml. (72%). There was no response to iron, liver, or transfusion, and her condition deteriorated rapidly. Thirty-seven days after the onset of the acute illness dark blood began to pour from the drainage tube. A blood transfusion was begun, and she received 3 litres of stored blood. She died six hours after the haemorrhage began.

Post-mortem examination showed thick blood-stained fluid and clots in the peritoneal cavity, a plastic peritonitis involving particularly the small intestine, fat necrosis of the greater omentum, and disintegration of the left half of the pancreas. Histological examination of the surviving portion of the pancreas showed the normal architecture to be quite well preserved; groups of acini showed disorganization and autolysis, but there was no haemorrhagic infiltration. The pancreatitis in this situation appeared to be resolving. The gall-bladder contained a large stone and several smaller calculi; its wall was moderately thickened.

COMMENT

The interest in this case arises not from the presenting symptoms and signs but from the complications.

Although it is not uncommon for there to be a slight glycosuria, so far as can be ascertained it is uncommon for a severe glycosuria and hyperglycaemia to be found. There can be no doubt that before the onset of the acute pancreatitis this patient had no symptoms to suggest that she suffered from severe diabetes mellitus and it seems unlikely that she had even mild diabetes. Yet 36 hours after the onset of the acute illness her urine was loaded with sugar and acetone and her blood-sugar level was considerably raised. This diabetic state slowly subsided and her blood-sugar level and urine were normal by the 12th post-operative day. Next day the glucose-tolerance test was abnormal. The pathogenesis of this phenomenon was probably a complete temporary inhibition of the islet tissue due to the action of the escaping enzymes. The islet tissues then gradually recovered, though incompletely. It is well known that mild diabetes mellitus may follow recovery from acute pancreatitis. It is interesting to note that although this was a severe case of the disease the histological examination of the pancreas showed many areas of apparently normal pancreatic tissue.

I do not know of any other case of acute pancreatitis in which haemorrhage from a splenic vessel was the cause of death, but it cannot be considered a surprising complication. It must be presumed, in spite of the histological findings, that the disease was still active, that the enzyme was still being liberated, and that this digested the wall of the vessel.

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