ACUTE PEPTIC ULCERATION AND CEREBRAL FAT EMBOLISM AFTER FRACTURE


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The relationship between acute intracerebral lesions and oesophago-gastro-duodenal ulceration has long been recognised, first by Rokitansky (1849), later by Pomorski (1891) and Lépine (1895), and in more recent years by Oppen and Zimmerman (1938), Strassmann (1947), Globus and Ralston (1951), Schlumberger (1951) and Dalgaard (1960).

Strassmann found fifty-six instances of peptic ulceration in 14,000 necropsies on mental hospital patients and for medico-legal purposes; all but two had intracranial lesions and twenty-six showed acute haemorrhagic ulceration of the stomach, oesophagus and duodenum. In addition thirty perforating lesions of the upper gastro-intestinal tract were noticed.

In 1960 Dalgaard reported his observations from 4,317 necropsies, one-third performed for medico-legal purposes. He found 208 cases of acute peptic ulceration including so-called oesophago-malacia and gastro-malacia and mucosal erosions. In 32 per cent cerebral vascular lesions were evident, compared with 12 per cent for the entire necropsy series.

Other causes of ulceration in Dalgaard’s cases were intracranial injuries (thirty-five cases), cerebral tumours (fifteen cases), craniotomy (twenty-three cases), infections (eleven cases), as well as burns (four cases), intoxication (twenty cases), cerebral hypoxia (nine cases) and acute stress (fifteen cases).

Microscopical examination confirmed the acute nature of the lesions, with necrosis, haemorrhagic and cellular response within the layers of the stomach wall.

Despite the variety of reported intracranial pathology that may give rise to acute peptic ulceration, we have been unable to find any instance or reference to fat embolism as a causative factor. This report is of three instances in which skeletal injury led to fat embolism, followed in two cases by perforation, and in the other by haemorrhage. All proved fatal and were recognised only at necropsy.

CASE REPORTS

Case 1—A woman of forty-five, a rear seat passenger in a motor car in head-on collision with another car, sustained a fracture of the pelvis and ribs and was admitted to hospital in January 1961. Apart from initial shock her condition was satisfactory and remained so for four days, after which she became drowsy and disorientated. Since an intracerebral injury or haemorrhage seemed a possible explanation she was transferred to a neurosurgical unit for investigation; a diagnosis of cerebral fat embolism was made. On account of increasing abdominal rigidity and a possible retroperitoneal haemorrhage complicating the fracture of the pelvis, laparotomy was carried out six days after the injury. No haemorrhage was found, but as the bladder wall showed bruising, it was thought advisable to insert a suprapubic drain. The patient died twelve days after the injury.

Necropsy—There was an oblique fracture of the right pubic ramus two inches from the mid-line, with fading bruising of the pelvic tissues. The right clavicle was fractured in its outer third, and the lowest five ribs on the right side were fractured in the anterior axillary line. Although there was no evidence of increased intracranial pressure, the capillary vessels were greatly dilated, and the white matter contained scattered multiple tiny haemorrhagic areas suggestive
of fat embolism. The peritoneal cavity showed an acute inflammatory reaction and contained four to five pints of bile-stained fluid, with some purulent exudate adherent to loops of small intestine. On the anterior wall of the duodenum, one inch distal to the pylorus, an irregular oval area of ulceration (one by two inches in diameter) had extended through the muscular coat to perforate the peritoneal coat; no induration was evident and the edge of the ulcer was soft and necrotic. Distal to the perforation two further ulcerations were seen, with serpiginous outlines and of recent origin, but only involving the mucosal surface. The oesophagus, stomach and remainder of the intestinal tract appeared healthy. The bronchial tree contained purulent exudate, and both lungs showed bronchopneumonic consolidation, many focal areas and some diffuse ones being apparent especially in the lower lobes. The cardiovascular system was healthy. The bladder wall showed an intense haemorrhagic cystitis and some residual haemorrhage in the serosal and muscular wall of the fundus, but no perforation had occurred.

**Histological examination** of frozen sections for fat revealed numerous droplets of sudan-positive material in the cerebral capillary vessels, and in the lung and kidney, all indicative of fat embolism. Microscopical examination of the duodenal ulceration showed necrosis of all layers at the site of the perforation, adjacent haemorrhage with thrombosis of small veins and scattered inflammatory infiltration of the submucosa. Because of the possibility of fat emboli within the gastric vessels, frozen sections of the ulcer were made, but no fat was seen.

The cause of death was generalised peritonitis from acute duodenal ulceration due to cerebral fat emboli complicating fracture of the pelvis and ribs.

**Case 2**—A man of fifty-two was admitted to hospital in May 1955 deeply unconscious after a road accident. An open comminuted fracture of the right femur was reduced and a plaster spica applied, but a mid-thigh amputation became necessary three days later on account of ischaemia of the leg. In addition the fifth metacarpal bone of the right hand was fractured and there were numerous lacerations on the face and scalp. After three days the patient was transferred to a neurosurgical centre, conscious but drowsy, and the following day ventriculography revealed some xanthochromic fluid in the left subdural space. Six days after the injury bloodstained fluid was aspirated from the right ventricle, but his general condition continued to deteriorate with episodes of Cheyne-Stokes respiration and hypotension. On the seventeenth day a melena stool was passed, and further intestinal haemorrhage necessitated the transfusion of six pints of blood in the course of two days. He was transferred to a general hospital with a view to gastrectomy, but he died twenty-four days after the injury after further episodes of hypotension and a terminal hyperpyrexia.

**Necropsy**—The right upper thigh amputation stump was infected and the femoral artery and vein were thrombosed. There was no evidence of fracture of the skull. There was some xanthochromic discoloration of the meninges over the left side of the brain, where some softened brain tissue was adherent to the overlying dura. There was also a similar slight discoloration on the right temporal lobe and small haemorrhagic areas were scattered throughout the brain, greatest on the left parietal and right frontal regions. These haemorrhages were associated with small areas of infarction, yellowish in colour, the largest four millimetres in diameter. In all, approximately fifteen such areas were seen, none in the hypothalamic area. The heart showed no significant abnormality. The lungs were grossly congested, with some bronchopneumonic consolidation in the lower lobes. On the posterior wall of the first part of the duodenum, one centimetre from the pylorus, an acute ulcer 1.5 centimetres in diameter was seen, and a much larger and more superficial ulcer extended from a point 3 centimetres distal to the pylorus almost to the ampulla of Vater, its diameter varying from 1 centimetre to 3 centimetres. Both ulcers were superficial, with no evidence of induration, but some thick-walled small blood vessels were identified in the base of the larger ulcer. The liver showed some fatty change and cloudy swelling. The kidneys appeared normal.

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Histological examination of frozen sections from the brain confirmed the presence of fat in the cerebral capillary vessels.

The cause of death was intestinal haemorrhage due to acute duodenal ulceration from cerebral fat embolism after fracture of the right femur.

Case 3—A man of thirty-six was admitted to a general hospital after a road accident in January 1958 with a comminuted fracture of the shaft of the right femur and fractures of the right tibia, fibula and patella. Open operative reduction of the fracture of the femur was performed next day, and an intramedullary nail was inserted. He did not make a normal recovery from the anaesthetic, and the following day was unconscious; at no time were there any localising neurological signs. He died in coma on the seventh day after the accident.

Necropsy—There were small abrasions over the right eye and forehead. The limb fractures were confirmed. There was no fracture of the skull. The brain was large and soft, and the superficial vessels were congested. The cut surfaces of both cerebral hemispheres showed large numbers of petechial haemorrhages, almost entirely confined to the white matter. The whole of the cerebral white matter was soft, with early xanthochromic discoloration. The remainder of the brain was only slightly affected, and was macroscopically normal.

The abdomen was distended because of dilatation of the small intestine from a generalised fibrinous peritonitis. The oesophagus and stomach were normal, but in the first and second parts of the duodenum there were two large, superficial, acute ulcers up to 2.5 centimetres in diameter. One of these showed ulceration only, but the other presented a large ragged perforation into the peritoneal cavity. The bronchial tree contained turbid mucoid secretion, and there was early bronchopneumonia at the bases of both lungs. No significant abnormality was found in the heart or other viscera.

Histological examination of frozen sections showed fat in the cerebral capillaries and confirmed the perivascular haemorrhage and cerebral softening with early microglial proliferation.

The cause of death was peritonitis from perforated acute duodenal ulcer, due to severe cerebral fat embolism after multiple fractures of the right leg.

DISCUSSION

The position and types of ulcer seen here are exactly comparable with those found after severe burns (so-called Curling’s ulcer).

The exact pathogenesis of these ulcers is unknown: toxaemia, haemoconcentration and infection with so-called “neurohumoral” factors have been suggested. The most widely accepted theory of the relationship of cerebral damage and acute peptic ulceration rests on Cushing’s (1932) work and on that of Watts and Fulton (1935), who postulated hypothalamic control of gastro-intestinal activity whereby parasympathetic over-activity or sympathetic inhibition caused vasoconstriction of the gastric mucosa and subsequent ulceration. Feldman, Birnbaum and Behar (1961) investigated the effects of electrical stimulation of the hypothalamic area of cats, and found that, whereas stimulation of the anterior hypothalamus produced erosions or haemorrhages in over 50 per cent of cats, stimulation of the posterior hypothalamus only rarely produced such lesions. Furthermore there was no correlation between these lesions and the increased gastric acidity produced by hypothalamic stimulation. Their work lends further support to the significance of local mucosal vascular changes after minutely localised hypothalamic stimulation.

Watson and Netsky (1954) thought that a restraining cortical or subcortical centre should be prefixed to the concept of hypothalamic control, and suggested that it was only when this higher centre and pathway to the hypothalamus is blocked or destroyed that gastric vasoconstriction can lead to ulceration. They thought that there was insufficient evidence for the theory of “visceral circulatory stasis.” In their review of forty-four cases, including six of their own, they found no instance of hypothalamic localisation of disease. Dalgaard (1960),
too, found no predominance of diencephalic tumours in his cases. In many instances widespread diffuse cerebral damage including oedema has been associated with peptic ulceration, as in the cases reported here in which there was no evidence of hypothalamic localisation of fat emboli (particularly in Case 1), although the diffuse nature of the cerebral lesion may well have included the hypothalamic area.

The abdominal catastrophe that had occurred in these three cases was entirely unexpected, and it is therefore important to remember the possibility of development of perforating upper intestinal ulcerations in patients with cerebral fat embolism, although the degree of cerebral damage may completely mask an abdominal catastrophe.

**SUMMARY**

1. Three cases of acute peptic ulceration are reported after cerebral fat embolism complicating skeletal injuries. In two instances death occurred after perforation of an ulcer, and in the third intestinal haemorrhage led to death.
2. Although intracranial lesions and peptic ulceration have been reported on many occasions, so far as we know cerebral fat embolism as a cause has not been previously recognised.
3. The possible pathogenesis is discussed.

**REFERENCES**


