CURLING'S ULCER
DUODENAL ULCER FOLLOWING SUPERFICIAL BURNS
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Case History.—A white boy of eleven years was admitted to my service at Touro Infirmary August 26, 1929, with superficial first-degree burns from gasoline, which involved both lower extremities from the ankle to the junction of the middle and upper third of the thigh. The previous history was irrelevant. On admission his temperature was 96.5° and his pulse 90. The following day they rose respectively to 100.8° and 160, and there was evidence of a severe toxæmia, with vomiting of all food, involuntary voiding and extreme delirium. Under local and constitutional treatment there was progressive improvement until September 2, when he complained of generalized abdominal pain, which was somewhat relieved by an enema. Shortly thereafter he vomited blood-stained material, and macroscopic blood was observed in the stools. Blood study at this time showed 3,240,000 red cells, 29,100 leucocytes, and 67 per cent. polymorphonuclears. Urinalysis was essentially negative, the trace of albumin evident on admission having disappeared. The blood-pressure was 100/64; September 3, copious haemorrhage from the bowel having continued, it fell to 88/0. On that day the abdomen, which had been quite soft, gradually became distended and tender, and tympanites was marked. Transfusion was done and repeated the following day, 250 cubic centimetres of citrated blood being given each time. The first injection resulted in some improvement in the general condition, but shortly after the second the patient became wildly delirious, the bowels moved involuntarily with profuse hemorrhage, although there was no further vomiting, and death ensued within a few hours.

Autopsy, which was done immediately, was essentially negative except as regards the gastrointestinal tract, the spleen and the kidneys. The latter organs showed acute splenitis and acute glomerulo-nephritis. The jejunum and ileum were both distended, and the ileum was full of blood. A small purplish-brown area, about 1/2 centimetre in diameter, was discovered at the junction of the first and second portions of the duodenum, and examination of the mucosal surface showed a small ulcerated area, about 3/4 centimetre in diameter, about to perforate, at the outer margin of the descending portion. Another larger but more superficial area, which involved only the mucosa, was evident at the same level in the posterior wall, and was considered to be a contact or "kissing" ulcer.

I am aware of the pitfall of reporting single cases, but I think circumstances such as these justify the act. This boy died from a disease which I knew could exist as a complication of burns, indeed which I had encountered at least once before, but which, because it was unusual, I failed to diagnose, in spite of the definite evidence that gastrointestinal pathology was present. Whether he could have been saved with the proper treatment I cannot say. Perhaps not. But that does not lessen my responsibility, and my failure to identify accurately an unusual but perfectly possible disease is my excuse for presenting this report.

Curling's ulcer is very largely an unknown lesion. If it is mentioned at all in the textbooks the description is of a very cursory character. This is partly due to the fact that it is seldom recognized during life, partly because
its incidence, as I shall point out shortly, is variable and rather low, and chiefly, I think, because it is most frequent in children, whose symptoms are notoriously vague and in whom it is easy to overlook the diagnosis of even more usual conditions.

Curling in 1842 first described duodenal ulcer associated with burns as a clinical entity, though he himself admits that Dupuytren, some ten years before, had called attention to ulceration of the intestines as a late finding in patients who survive the immediate effects of the injury. Curling, however, definitely localized the lesion in the duodenum, though for the sake of historical accuracy we might add that contributions to the subject had been made by Cumin in 1823, by Cooper in 1840 and by Long in the same year, and that Erichsen wrote a comprehensive paper upon it in 1843.

Curling, in the treatise which he read before the Royal Medico-Chirurgical Society, reported ten cases, four of which he had seen personally. The ulceration, as he described it, is of an acute character, the immediate cause of death being perforation with consequent peritonitis in three of his cases and haemorrhage in six. One patient survived five weeks, the others died in from seven to seventeen days. He also described three other cases, one seen personally, in which post-mortem examination showed a definite increase in vascularity as well as definite inflammation of the duodenum, though there was no destruction of substance. Finally, he described a cicatrizcd duodenal ulcer from the collection in the Hunterian Museum; this was exhibited in the body of a young woman who had died of exhaustion eight weeks after a burn, and is unquestionably of the same type.

It is nearly a century since Curling established the sequence of burns and duodenal ulcers, yet there is still no uniformity of opinion as to the incidence of the lesion. Erichsen, a year after Curling’s identification, found them in two of twenty-eight fatal cases. Ronchesi found them in one of 348. Fenwick states that they are present in 6.2 per cent. of all deaths from burns, while Holmes, in 125 cases, reports an incidence of sixteen, 12.8 per cent. Harris, with an experience of 567 cases and 138 deaths over a twelve-year period, found only one case. Levin, reporting for himself, his predecessor and two of his associates, with fifty composite years of service at Johannesburg, and with an experience of fully 12,000 autopsies, found only two cases, and those when he had begun to believe that the lesion was merely a medical myth. Bancroft and Rogers found none in 104 cases, and likewise seem doubtful of the existence of the disease. The indices of the New Orleans Charity Hospital show not a single instance, although this institution has probably the largest accident service in the South. The case I have reported is the only one recorded at Touro Infirmary. Are these ulcers decreasing in incidence, or are we simply failing to find them because we do not look for them? My own experience inclines me to the latter view.

Such ulcers are commonest in children, the average age in Curling’s series being 10.8, and all but two of his patients being under 15. They are usually considered a late complication, but as a matter of fact, and as the literature
evidences from the beginning, they may occur at any time. Levin mentions Parfick's case, quoted in Choyce's System of Surgery, in which the ulcer was evident eighteen hours after injury, and in Harris's case death ensued on the third day, perforation, as the autopsy showed, having already occurred. In many instances, however, the lesion develops during the third or fourth week of illness, when the patient has apparently recovered from the critical stage of the injury. In Simpson's case the intestinal symptoms developed at the end of one hundred days, when the local condition was progressing to recovery. In spite of the epigastric pain, I am doubtful whether this patient really had a duodenal ulcer, though the intestinal ulceration is beyond question.

The symptoms, as Curling names them, include pain and tenderness to pressure on the right side, midway between the cartilage of the ribs and the umbilicus, uneasy digestion and vomiting. When ulceration ensues, the stools are dark and bloody. In very acute instances, either haemorrhage or perforation may be the initial sign, and there may be no symptoms at all, as in one of Levin's cases, in which death was definitely due to pneumonia, and in which, in spite of a complete lack of ante-mortem symptoms, the autopsy disclosed a typical ulcer which had evidently just perforated. In short, the symptomatology and clinical course are akin to those of the ordinary chronic or acute peptic ulcer. Curling is quite correct when he points out that the morbid action, however acute it may be, is deep-seated and limited in extent, as well as masked by the general derangement. The pathology of the burn itself—which is the pathology we look for and expect—dominates the clinical picture, and again I revert to my thesis, that only by bearing in mind the possibility of the unusual complication can we be certain of not overlooking it.

The etiology of Curling's ulcer, as is the case with other peptic ulcers, is still an unsolved problem. The burn, of course, introduces certain special considerations, for in addition to the local pathology there are inevitable systemic and constitutional complications. Severe burns are characterized primarily by pain and shock. Following these there is evident a profound toxæmia, decidedly more marked in children, which, by its effect on the central nervous system, may result in hyperpyrexia, vomiting, drowsiness and convulsions. Finally, there may be exhibited such complications as pneumonia, nephritis, or the protean manifestations of infection, the latter being especially evident in improperly treated cases.

Curling's theory of the etiology of these ulcers, considering the limited medical knowledge of the day, is rather an ingenious one. Congestion of the mucous membrane, he says, is an insufficient explanation, because the remainder of the alimentary tract, although participating equally in the vascular disturbance, rarely becomes affected. The glands of Brunner, however, he found enlarged and infiltrated in at least one fatal case, and his idea was that the sudden arrest of the important functions of the skin rouses in these glands an endeavor, by increased action, to compensate for the suppression of dermal exhalation, the irritation from hyperactivity leading eventually to inflamma-
tion and ulceration. The specific localization is thus explained, and the haemorrhagic feature is due to the fact that if perforation does occur, the arteria pancreatica-duodenalis, because of its position, necessarily becomes exposed. This theory he considered confirmed by the fact that the disease, if not fatal, goes on to recovery when the functions of the skin are reëstablished, though he apparently failed to see that it does not explain the fairly frequent cases in which the ulcer does not exhibit itself until the local condition is progressing toward recovery.

In more modern days Cooke and Falk have advanced as a cause a reflex inhibition of the intestinal circulation, Falk adding that the depressed action of the heart or the impaired nourishment of the mucous membrane is a predisposing factor. Billroth and Moynihan consider it an embolic process, Moynihan pointing out that the ulcer never occurs without septic changes in the burn. Simpson and others consider it due to a sort of ante-mortem digestion, that part of the mucous membrane being affected in which the circulation has been arrested by congestion or embolism.

The consensus of modern opinion regards the toxaemia of burns rather than the circulatory disturbance as responsible for the production of the associated duodenal ulcers. Certainly, since the toxaemia is most marked in children, this would explain their most frequent incidence in young subjects. According to Hunter, a toxic substance is elaborated in the burned tissues and excreted in the bile, and he was able to produce such ulcers experimentally in dogs by the injection of toluylenediamin. Fenwick, on the other hand, could not produce them by tying the common duct, and Busse produced general intestinal as well as duodenal ulceration and haemorrhage by the injection of extracts elaborated from the burned areas of the skin. DaCosta is opposed to Hunter's theory on the ground that the typical ulcer occurs well above the ampulla of Vater. Catiano and Harris advance rather similar theories, to the effect that the action of the toxin, by reducing the natural alkalinity of the intestinal wall, causes focal necrosis and haemorrhage, the areas being transformed into ulcers by the action of the pancreatic juice. Levin's theory is along the same lines. He believes that the shock incident to all burns causes either a true or a relative hyperacidity and hypoalkalinity, that the special toxin of burns is a substance which may be akin to histamine, and that the combination of hyperacidity and the devitalizing effect of the toxin on the mucous membrane favors the development of the ulcer. The specific localization he explains, as all duodenal ulcers may be at least partially explained, by the tortuosity of the vessels of the lesser curvature of the stomach and the first part of the duodenum, their relatively poor anastomosis rendering them more liable to thrombosis. This, of course, is in keeping with the well-known demonstration of Wilkie, amply corroborated by Finney, of the rôle played by the superior duodenal vein in most peptic ulcers involving the first portion of the duodenum.

All theories fail, I might remark, to explain the case reported by Leonard and Dayton. Post-mortem examination of a patient with cervical carcinoma
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who had been treated by the Percy method revealed a typical Curling ulcer. Since the skin factor is entirely lacking here, no explanation yet advanced fits the case.

It has long been recognized that while these ulcers might exist anywhere in the intestinal tract, they were most common in the first portion of the duodenum, but it remained for Pack, in 1926, to describe the histopathology definitely. They may vary in size, he says, from a pinhead to a quarter, and the amount of tissue loss depends on whether the lesion is a mere erosion or a rapidly sloughing perforative process. The ulcer is frequently funnel-shaped, due to loss of more mucous membrane than muscle tissue, and the shape may be irregular and dentate, long and narrow, or, less often, circular. The edges are sharply and cleanly cut, and the base is clean and grayish, though, in the rare instances in which the lesions tend to chronicity, the edges may be indurated and there may be some inflammatory reaction about the margin. Lymph and fibrinous exudation may be seen on the peritoneal surface, as a pre-formed and protective barrier to the lethal progress of the disease if perforation threatens. The factor of time may exert some influence on the depth of penetration, but the age cannot be accurately estimated from this, since the lesion is so insidious and so asymptomatic in its incipiency as to elude diagnosis. The outcome is perforation, haemorrhage or spontaneous healing.

As a remote result of a healed duodenal ulcer of the Curling type, I quote the following case: Early in my practice I treated a white male, then aged forty-eight years, for extensive burns of the chest, epigastric region and both legs, the recovery being complicated by lobar pneumonia, and by definite symptoms of a duodenal ulcer. At the age of sixty-five he consulted me again, complaining of various digestive disturbances which rather suggested gall-bladder pathology, though in view of his age malignancy had to be considered. He refused operation. Three years later, at sixty-eight years, he returned again, this time with a history of recent acute digestive symptoms, constant epigastric pain, very marked loss of weight, in short, the whole symptom complex of gastric malignancy. Exploratory incision revealed inoperable carcinoma of the upper abdomen, with extensive retroperitoneal metastases. There was definite pyloric infiltration. Nothing could be done for his relief, and he died of inanition two weeks later. There is little doubt in my mind that in this instance the carcinoma was superimposed upon the old cicatrix, and the chain of events raises the question of whether the Curling variety of ulcer, unlike the usual duodenal ulcer, predisposes to malignancy.

In no case of Curling's ulcer reported in the literature, so far as I can ascertain, was the specific lesion treated. It was either revealed at post-mortem, there being no suspicion of its previous existence, or the patient recovered spontaneously. But such therapeutic nihilism is obviously without justification. Curling stated in his paper that in any case which in the future he should recognize during life, he would apply leeches to the skin.
of the corresponding part of the abdomen, and would give mercury and chalk, with opium for pain, and bland fluid nourishment. We have no record of whether the treatment he outlined was ever applied, and in any event it is not a regimen suitable today for any sort of ulcer. For my own part, in another case of bleeding ulcer such as I have reported, I shall be tempted to resort to surgery, as I should resort to it for other bleeding peptic ulcers. Transfusion availed nothing, and surgery, even though the boy was a frankly poor risk, might have saved his life. In the non-acute type of ulcer, prophylactic excision might be justified, even in seriously burned patients, where the risk would be very high, to avoid the possibilities—which are not remote—of perforation or hemorrhage. The exact procedure, however, is beside the point. My premise is that something should be done. There is no justification, in this epoch of medical achievement, for permitting patients to die from a disease in which, if it were promptly diagnosticated and properly treated, they might have at least a chance of life.

SUMMARY

1. Ulcer of the duodenum, described in 1842 by Curling as a complication of burns, has an estimated incidence in fatal cases of 6 per cent. or more, and occurs most frequently in young children.

2. Its symptomatology and clinical course do not differ from those of the ordinary peptic ulcer, but its existence is prone to be overlooked in the severe constitutional and systemic manifestations which follow burns.

3. The etiology is still unsettled, but the theory of a toxæmic origin seems more reasonable than the theory of a circulatory origin.

4. The histopathology has recently been comprehensively described by Pack.

5. The literature contains no treatment for this type of ulcer, and it is suggested that at least in some cases surgery, as applied to the ordinary peptic ulcer, would seem to warrant a trial.

6. Two additional cases are reported, one in a boy, with fatal result, the other in a patient who recovered and who, twenty years later, died of carcinoma, probably superimposed upon the site of his original ulcer.

BIBLIOGRAPHY