سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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سامية محمد مصطفى

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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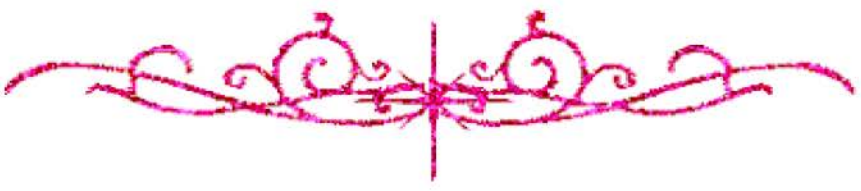

سامية محمد مصطفى

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بالرسالة صفحات لم ترد بالأصل

OMENTAL PLUG TECHNIQUE IN THE TREATMENT OF PERFORATED PEPTIC ULCER

THESIS

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In

General Surgery

By

Sobhy Noseir Helal

(M. B., B. Ch.)

SUPERVISORS

Prof. Dr.

Mostafa Abd- Elkader Eltablawy

Prof. of General and Gastrointestinal Surgery and Endoscopy Head of General Surgery Department

Faculty of Medicine

Tanta University

Prof. Dr.

Osama Abd- Elsamea Baza

Prof. of General and Gastrointestinal Surgery and Endoscopy

Faculty of Medicine

Tanta University

Dr.

Gamal Ibrahim Mousa

Lecturer of General and Gastrointestinal Surgery and Endoscopy

Faculty of Medicine

Tanta University

FACULTY OF MEDICINE

TANTA UNIVERSITY

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INTRODUCTION

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The commonset perforation of the upper gastrointestinal tract is a duodenal ulcer. In 1929 Cellan-Jones described the most widely used technique for dealing with perforated ulcers, in which a strand of omentum is drown under an arch of full thickness sutures placed on either side of perforation(Karanjia et al., 1993).

Although the omental patch technique is useful for small holes, it is associated with releakage when the perforation is large (Hugh, 1990). This prompted a consideration of an alternative technique for patching a large perforated peptic ulcer like omental plug, which avoids the development of omental ischaemia and subsequent releakage (Bodner et al., 1990).

Although perforated peptic ulcer remains a dramatic surgical emergency, nowadays it seldom result in death, the surgical mortality has declined steadily and become 5% (Boey et al., 1982). This improvement, as well as the high incidence of ulcer relapse after plication alone, persuaded many surgeons to adopt immediate definitive operations for acute perforation (Coutsoftides and Himal 1976). Emergency curative surgery is never entirely safe, and it can incur a higher mortality than does plication of perforation (Griffin et al., 1976). Obviously, patient characteristics are crucial in choosing optimal surgical treatment. Simple closure, or even non operative management is acknowledged to be most appropriate for patient who are markedly debilitated or in shock (Greco and Cahaw, 1974).

REVIEW OF LITERATURE

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Incidence of perforated peptic ulcer:

The incidence of perforated peptic ulcer is approximately 7 to 10 per 100.000 population per year (Watkins et al., 1984). The ratio of perforated duodenal to gastric ulcer is approximately 7 to 1, the ratio of men to women with perforated peptic ulcer is about 2 to 1 for duodenal ulcer and I to I for gastric ulcer (Pirnela et al., 1991).

Owing to a reduced incidence of perforation in men and an increased in women, the mean age of men with perforated duodenal ulcer increased from about 50 years to about 60 years (Walt et al., 1986). The majority of perforation (60%) are of chronic ulcers (Valerio et al., 1982).

The reduction in mortality from perforated peptic ulcer from 20% in the early 1960 to 13% in 1984, probably due to improvement in anaesthesia and postoperative care, despite an increase in the average age of the patients treated (Watkins et al., 1984).

Perforation is present in about 7% of patient hospitalized for peptic ulcer disease, and the first manifestation of the disease in about 2% of patients with duodenal ulcer. It is estimated that, after the diagnosis of duodenal ulcer, 0.3% of patients perforate annually in the first 10 years (Debas and Muldvhill 1997).

H_2-receptor antagonists induce healing of most duodenal ulcer (Winship, 1988). It is interesting therefore that some patients with chronic ulcers were receiving cimetidine at the time of perforation. However, compliance with this treatment may be poor especially if patients find that their dyspeptic symptons are still controlled despite intermittent usage of the drug (Watkins et al., 1984)[8].

ETIOLOGICAL FACTORS OF PERFORATED PEPTIC ULCER

1- Stress and psychological factors:

The role of stress in peptic ulcer disease remains unclear. Psychological factors have not been shown conclusively to be important in the etiology of peptic ulcer (McIntash et al., 1985).

The mechanisms linking psychological factors to ulcer disease are unclear. However, it has been shown that increased gastric acid secretion may be associated with stressful life event. Also hard physical exercise with sleep deprivation results in a three fold increase in basal acid secretion (Oktedalen et al., 1984).

II- Smoking and ulcer perforation:

The association between ulcer perforation and smoking seems biologically plausible. Smoking is known to have several adverse effects on the upper gastrointestinal tract (Eastwood, 1988), of particular interest for ulcer perforation is the finding that smoking causes immediate vasoconstriction in the mucosal blood vessels (Iwao et al., 1988).

Ischaemia reduces mucosal resistance against the action of acid and may thus contribute to ulcer perforation, this mechanism could explain why we observe an increased risk of perforation in current smokers but not in former smoker (Sørbye and Svanes 1994).

In elderly persons smoking seemed to be of less importance than in the younger age group. Excess mortality after ulcer perforation, may suggest a shift in the aetiology of ulcer perforation over time, with smoking having a more predominant role in younger generation (Svanes et al., 1996).

Tobacco smoking is a well known risk factor for uncomplicated peptic ulcer. Smokers have an increased risk for dying from peptic ulcer disease (Kuata et al., 1992). However, the incidence of ulcer perforation can only be markedly reduced, on a population basis, by smoking prevention(Svanes et al., 1997).

III- Non-steroidal anti-inflammatory drugs (NSAIDs) and peptic ulcer perforation:

Two groups of drugs, Corticosteroids and NSAIDs, have been suggested as possible trigger agents for peptic ulcer perforation. Since these drugs have come into widespread use, a high proportion of patients who have perforated ulcer, give a history of taking NSAIDs, corticosteroids or both(Horowitz et al., 1989). NSAIDs are associated with approximately a five fold relative risk of developing a gastric ulcer (Duggan et al., 1986). The incidence of gastric ulcers in patients taking NSAIDs and aspirin is about 10-15% during the first 3 months of use (Piper et al., 1981). Duodenal ulcers also occur as a result of NSAIDs use, but generally less frequently than gastric (Duggon et al., 1986).

In normal individuals ingestion of aspirin has damaging effects on the gastric and duodenal mucosa, lesions which resemble duodenal ulcer may results from ingestion of aspirin for one day in normal individuals (Hoftiezer et al., 1982).

In patient taking eight aspirin tablets daily for more than 3 months endoscopic examination has observed duodenal ulcer in 4%, duodenal erosions in 13%, and gastric ulcer in 31% (Lockand et al., 1980). The percentage of patients with perforations taking NSIDs only increased in those used multiple NSAIDs, high doses of these agent and in those aged over 65, especially women.

Therefore the elderly, particulorly women, appear to be unduly susceptible to the effect of NSAIDs. This may be because of pathophysiological changes associated with aging (Collier and Pain 1985).

Elderly women seen to be particularly susceptible to perforation and this raises the possibility that postmenopausal hormonal changes may be involved in the pathogenesis. Oestrogens have been used successfully in the treatment of peptic ulceration (Truelove, 1980). As oestrogens and progestogens increase the production of mucus within the stomach. Postmenopausal women may have a lowered mucous production, and this could be further decreased by the action of NSAIDs thereby losing cytoprotection (Parbtoo and Johnston 1986). Conversely hormonal protection may account for the low

incidence of perforation in women aged under 65 years (O'Malley et al., 1991).

The mechanisms of aspirin and NSAIDs induced damage to the gastro-duodenal mucosa are:-

1- Muscus synthesis and release inhibited by aspirin and NSAIDs (Hoftiezer et al., 1982).
2- Block the synthesis of prostaglandin by inhibiting cyclo-oxygenase which predspose to mucosal injury and peptic ulceration (Hawkey, 1990).
3- It causes an increase in the exfoliation of surface epithelial cells from the mucosa, as demonstrated by cytological studies and by measurements on the DNA content of gastric aspirate (Croft, 1983).

Corticosteroids have similarly been implicated. The proportion of patients taking these is, of course, less than that for NSAIDs but the association with perforation appears just as strong. The proportion of patient taking corticosteroids prior to perforation varies from 5% to 10.4% (Watkins et al., 1984).

IV- The role of H. pylori in perforated peptic ulcer:

The role of H. pylori in perforated peptic ulcer disease is not clear. It showed that 48% of patients with acute perforated duodenal ulcer were H. pylori postive (Jensen et al., 1992). In contrast, it has been reported that 24-29 only patients with perforated peptic ulcer were infected with H. pylori (Sebastion et al., 1995).