INTESTINAL FISTULAE

Essay

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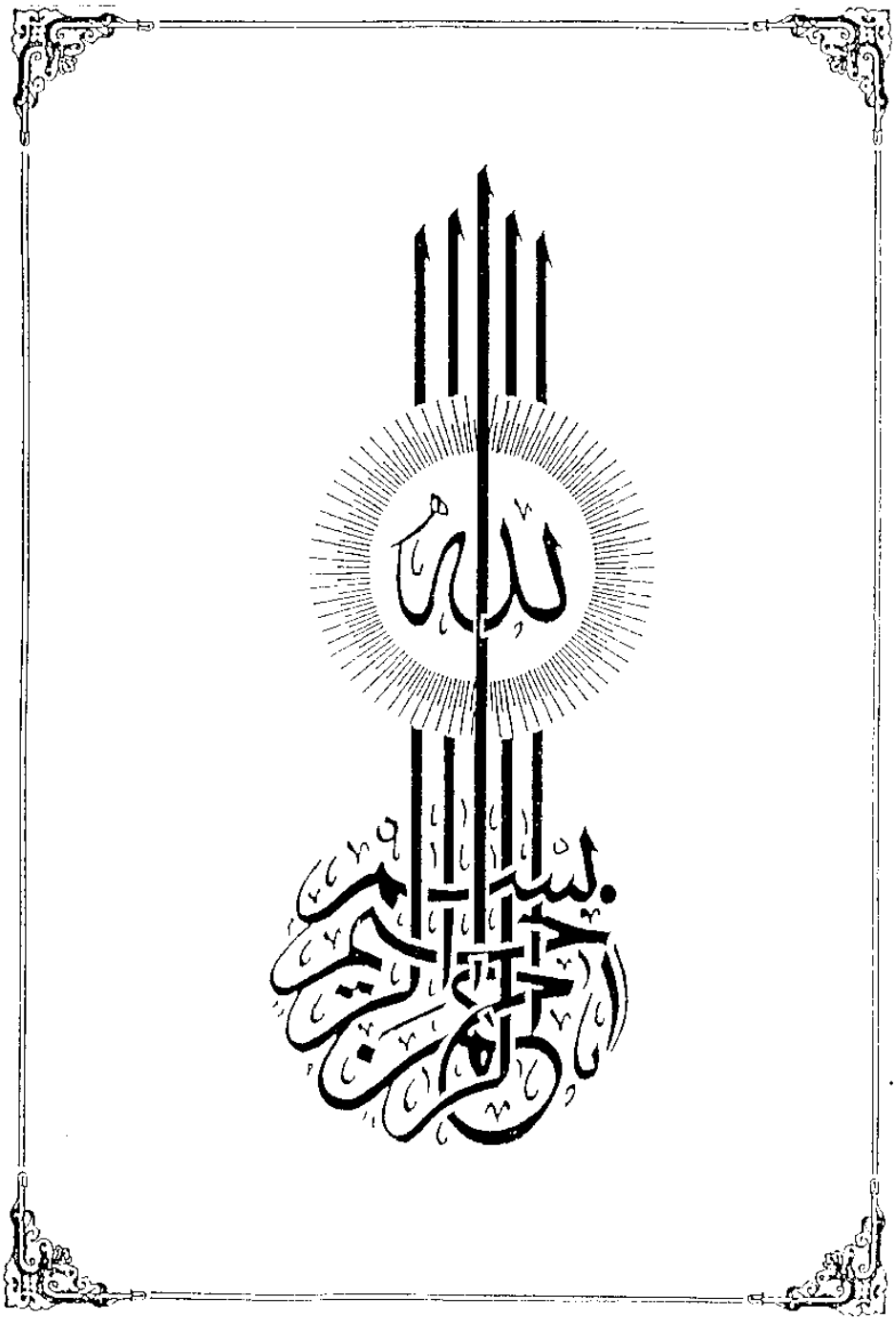

Dedication

To my Father,
Who gave me everything

CONTENTS

Intestinal fistulae often present a major complication facing the surgeon usually in the postoperative period. It involves metabolic and nutritional disorders together with antanomical abnormalities and extensive sepsis. A less serious but intolerable to the patient is the skin excoriation accompanying external fistulae.

Most intestinal fistulae follow surgical operations, however, other aetiological factors as Crohn's disease, Malignant disease and trauma are equally important.

Recent management of intestinal fistulae involves enteral or parenteral nutritional support, elimination of sepsis, stoma therapy and finally proper surgical intervention when the patient condition permits.

Aim of work

The aim of this work is to spotlight aetiological factors, presentation, and ideal management for a patient with intestinal fistula. To construct a strategy that leads to spontaneous closure of the fistula or, at least renders the patient in a better condition for further surgical procedures is the goal.

Definitions and Aetiology

DEFINITIONS AND AETIOLOGY

A fistula is an abnormal communication between two epithelialised surfaces. Usually it is formed of granulation tissue, however, it may be lined by epithelium as in congenital fistulae or when the epithelium grows from either side of the fistula.

Anatomically, a fistula may be external or internal. The former, also called entercutaneous fistula, connects the gut directly or indirectly with the body surface discharging the enteric contents. Internal fistula connects two viscera of the same or different systems. A mixed fistula is a combination of both types contributing both elements.

A simple fistula, means that there is a single track between both viscera or between a viscus and the body surface, while complicated fistulae, are those associated with multiple tracks or draining to the surface through an associated abscess cavity.

Lateral fistulae, are those originating from a partial defect in the side wall of the gut, whereas end fistulae, are those arising from the whole circumference of the involved bowel and there is no further continuity of the gastrointestinal tract.

Classifying intestinal fistulae on physiological basis is of major importance in reflecting their metabolic effects. So, according to their level they are classified into proximal or distal fistulae. Proximal fistulae usually cause more serious metabolic disturbances than distal fistulae. A daily discharge of 500 ml of enteric content distinguishes between high and low output fistula, except for pancreatic fistulae in which 200 ml/day is the level of differentiation between a high and low output pancreatic fistula *(Alexander Williams and Irving, 1982)*.

Apart from pancreatic fistula, serious metabolic sequelae are rarely seen as a result of loss of less then 500 ml/day of enteric contents *(Irving, 1983)*.

Aetiology of Intestinal fistulae

In the past, most intestinal fistulae were due to advanced untreated abdominal diseases and penetrating injuries. At present, surgical operations and

[illegible] *(Irving, 1983).* Most intestinal fistulae follow perforation of the bowel wall either by the underlying disease or breakdown of an anastomosis. Perforation of the gut is usually followed by abscess formation which can result in either internal or external fistulation depending on the site of its drainage.

Congenital Fistulae

Developmental errors can result in both internal and external fistulae. Tracheo-oesophageal fistula due to incomplete division of the foregut into a respiratory and a digestive portion, as well as rectovaginal fistula in females or rectovesical fistula in males due to failure of closure of the cloaca, are the best known internal congenital fistula. Vitello-intestinal duct will produce an umbilical fistula discharging enteric contents usually in the neonatal period, but can present in later life.

Traumatic fistulae

Trauma, both blunt and penetrating,is a major factor in producing intestinal fistulae. Penetrating trauma may be due to extraluminal penetrating object as gun-shots and stab-wounds, or intraluminal as with an ingested foreign body leading to immediate fistula formation if the defects are not recognized or are inadequately repaired at laparotomy. Unlike this, blunt or closed trauma may lead to delayed fistula formation due to retroperitoneal rupture of duodenum, pancreas or colon. In these circumstances, the leaked enteric content gives rise to a retroperitoneal abscess with subsequent fistula formation *(Alexander Williams and Irving, 1982).*

Inflammatory Fistulae

Intestinal fistulae may be the outcome of various inflammatory conditions as tuberculosis, actinomycosis, diverticular disease of the colon and Crohn's disease. Today in Western countries the commonest inflammatory cause of intestinal fistula is Crohn's disease, which is associated with fistula formation in about 30 percent of patients with small bowel affection *(Irving, 1985).*

The mechanism of fistula formation in this disease is not clear, but probably related to the fissures that are well recognized histologic manifestation of this disease. *Irving (1983)* recognized two types of fistula associated with

Crohn's disease; *Type I fistulae*, are those arising from an area of active Crohn's disease; whereas, *Type II Fistulae*, are those arising from the suture line of anastomosis undertaken following resection of the affected segment of bowel. *Type I* will always require operation toresect the diseased bowel together with the fistula, whereas, *Type II* Crohn's fistula like any other anastomatic leak will usually close with conservative treatment. Spontaneous external fistula occurring with Crohn's disease is typically seen in relation to a previous postoperative scar, as following appendectomy in someone found to have a segment of terminal ileal Crohn's disease. In such a patient it would be usual and correct to remove the appendix unless the caecum is heavily involved by Crohn's disease *(Irving, 1983)*, and it is noteworthy that it is not the appendix stump to which the fistula travels. However, rarely spontaneous external Crohn's fistula present through the umbilicus without a previous scar *(Hiley et al., 1971; Jensen and McClenathan, 1987)*.

Tuberculosis and actinomycosis are rarely seen in well developed countries, but they are still common in underdevloped countries.

Pancreatitis may result in the formation of a pancreatic pseudocyst or an abscess with subsequent fistula formation. The fluid contained in these cysts is highly digestive and can dissect through the diaphragm leading to a pancreatico-pleural or bronchial fistula *(Igelhart et al., 1986)*.

Extra intestinal inflammation may be a cause for fistulation, as in the case of biliary-bronchial or biliary-pleural fistula which may complicate a subphrenic abscess. Also, pyeloduodenal fistula, from rupture of a perinephric abscess into the second part of the duodenum *(Ross and Tanna, 1974)*.

Neoplastic Fistulae

Malignant tumours remain a well-recognized cause of internal fistula formation, produced simply by direct invasion of the tumour into adjacent viscera. Common examples of this type are the gastro-colic fistula produced by extension of a carcinoma of the stomach, and colovesical fistula from extension of a sigmoid carcinoma. Rarely, such tumours produce external fistulae, either by direct invasion of the abdominal wall, or by obstruction with proximal perforation and abscess formation.

Postoperative fistulae

Ninty percent of external fistulae result from some sort of surgical operations, and the original procedure is frequently an emergency one *(Monod Broca, 1977; Soeter's et al., 1979).* In a collective review of 384 small bowel fistulae *Edelmann and his associates* found that 60 percent followed an operation for intestinal obstruction or acute peritonitis. The cause is usually unrecognised injury of the bowel or breakdown of an anastomosis. The later, usually results from faulty technique as tension on an anastomosis, ischaemia of the bowel ends, or the presence of distal obstruction. Anastomosis are also more likely to breakdown in the presence of sepsis and hypoalbuminaemia. Another cause for anastomotic leakage with subsequent fistulation, is the presence of a tube or corrugated drain on the anastomotic line. Unrecognised injury of the bowel occurs with certain operations as duodenal injury during nephrectomy and injury of the greater curvature of the stomach during splenectomy. Deep tension sutures used in closure of difficult abdominal wounds may be a potent cause of fistula formation *(Alexander Williams and Irving, 1982)*, either because the suture penetrates the bowel wall or because a loop of bowel gets trapped between the abdominal wall and the suture loop.

Post-Irradiation fistulae

Radiation enteritis is an increasing problem that follows abdomino-pelvic radiotherapy for malignancies. It can lead to disability and ultimately death long after the original malignancy has been controlled *(Galland and Spencer, 1987).* The incidence of radiation enteritis appears to be increasing, *Swan et al. (1976)* stated that it ranges from 0.5 - 5 percent of all abdomino-pelvic irradiation. In 70 patients presented to *Galland and Spencer (1958 - 1984)* with radiation enteritis, 14 cases of which were presented by intestinal fistula. The combination of internal and external treatment which has been shown to increase the likelihood of radiation damage to the intestine, has become increasingly popular recently, and the newer technique of *"after loading"* may be among the factors responsible for this increase.

The effect of ionizing radiation on the gut wall is two fold, there are direct or acute effects on the proliferating cells, followed by, indirect or late effects due to progressive obliterative vasculitis resulting in ischaemic changes in the small vessels of the intestine which may result in localized perforation of the

[illegible] depletion of the actively proliferating cells in an otherwise stable cell-renewal system. Histologic abnormalities can be seen within hours of administration of therapeutic doses of radiation, small bowel mitosis are reduced in number, and the villi become shortened with a reduction in the total absorptive area. During this acute phase there is malabsorption and impaired metabolism of bile salts, fat, carbohydrates, proteins and vitamin B_{12}. This will be manifested clinically by diarrhea, nausea, vomiting, abdominal pain or tenesmus. There is some evidence that patients who experience the worst acute reactions are at greater risk of developing late gastrointestinal complications.

The late sequelae of radiation enteritis are apparent macroscopically as the gut becomes thickened with areas of telangiectasia. Mucosal ulceration is common and may involve the whole gut wall. Extension of this process leads to perforation with fistula formation, and/or the formation of local abscesses, while resolution results in stricture. In a study of the microradiology of transected specimens, *Carr et al. (1984)* demonstrated reduced and abnormal vascularity in irradiated compared with non-irradiated colon.

The latent period between completion of the course of radiotherapy and the subsequent development of radiation bowel damage is usually 6 -24 months. However, it is clear that radiation enteritis can become apparent many years after the original treatment and should always be considered when a patient who has had radiotherapy presents with abdominal problems. Recurrence of the original malignancy should not be assumed without excluding radiation damage *(Galland and Spencer, 1987).*

Pathophysiology

PATHOPHYSIOLOGY

The main complications responsible for morbidity and mortality in intestinal fistulae are malnutrition, fluid and electrolyte disorders, and sepsis. Malnutrition is simply caused by the premature loss of intestinal contents through an intestinal fistula hence by-passing the part of the gut distal to the site of origin of the fistula, preventing it from performing its normal absorptive function. Moreover, being rich in water and electrolyte content, loss of the gastrointestinal juice will also lead to fluid and electrolyte disorder with consequent acid-base imbalance and hypovolaemia. However, with the advent of efficient methods of fluid and electrolyte replacement together with nutritional support, only uncontrolled sepsis remains the principle determinant of mortality *(Soeter's et al., 1979).*

Sepsis results from the escape of the enteric contents through a poorly formed fistula track into normally sterile areas as the peritoneal or the pleural cavities with subsequent abscesses formation (Irving, 1981). Abscess formation is a step in the pathogensis of fistula formation in certain diseases as Crohn's disease and diverticular disease of the colon. In these diseases, abscess formation follows local perforation of the gut and is followed by perforation into another viscus with internal fistula formation or, is drained to the body surface with external fistula formation.

Sepsis is considered as a major factor causing nutritional disorder, fluid and electrolyte disorder with subsequent acid-base imbalance *(Beisel,1986).* Meanwhile, malnutrition will lower the patient's resistance to infection with increased liability for septic complications *(Kahan, 1981).* So, it is clear how the triad constituting the major complications of intestinal fistulae form a vicious circle together pointing to a deteriorated metabolic condition of the patient.

Other less common complications of intestinal fistulae are intestinal obstruction and bleeding secondary to the primary disease itself such as inflammatory bowel disease and malignancy *(Soeter's et al., 1979).* A less serious but intolerable by the patient is the skin excoriation which will occur due to the discharge of the enteric contents onto the skin. Facing all the previous complications of enteric fistulae, the effects of the underlying disease which may need management in its own must not be forgotten.

Table (1): Complications of gastrointestinal fistula in 119 patients 1960 - 1970 and 128 patients 1970 - 1975 *(Soeter's et al., 1979).*

	1960-1970	1970-1975	Hyper alimented	Non-Hyper alimented
Total	119 (100 %)	128 (100 %)	73 (100 %)	55 (100 %)
1. **Malnutrition**	104 (97 %)	65 (51 %)	44 (60 %)	21 (38 %)
a. Moderate	68 (57 %)			
b. Severe	36 (30 %)			
2. **Sepsis**	66 (55.5 %)	95 (74 %)	69 (94.5 %)	26 (47 %)
a. Controlled	30 (25 %)	45 (35 %)	30 (41 %)	15 (27 %)
b. Uncontrolled	36 (30 %)	50 (39 %)	39 (53.4 %)	11 (20 %)
3. **Electrolyte disorder**	54 (45 %)	35(27 %)	29 (40 %)	6 (11%)
4. **Obstruction**	30 (25 %)	13 (10 %)	3 (4 %)	10 (18 %)
5. **Bleeding**	4 (34 %)	10 (8 %)	10 (13.7 %)	—
6. **Wound dehiscence**	—	4 (3 %)	3 (4 %)	1 (1.8 %)
7. **Renal / Hepatic failure**	—	2(1.6 %)	2 (2.7 %)	—