

**EVALUATION OF NON SURGICAL VERSUS
SURGICAL TECHNIQUES
IN MANAGEMENT OF CHRONIC ANAL FISSURE**

A Thesis submitted for partial fulfillment of the degree of
M.Sc., in general surgery

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Abstract

This study compare using of non surgical methods included botulinum toxin injection and glyceryl tri nitrate 0.2% with surgical methods included fissurectomy with lateral internal sphinctrotomy in treatment of chronic anal fissure we found that using of surgical methods give results equal 100% after six month follow up while using of glycerial trinitrate 0.2% twice per day for 8 weeks the result was 70% healing rate while it was 75% in botulinum toxin injection we inject 20 unit of botulinum toxin in internal anal sphincter anterior after dilution of it in 50u saline .The complication was insignificant in three groups , incontinence was to flatus only and it disappear after three months follow up , we recommended using of lateral internal sphinctertomy as frist line in treatment of chronic anal fissure and using of chemical sphincterotomy in patient who unfit for surgery or refusing surgery.

Key wards :

Chronic-anal- fissure- surgical-botulinum

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LIST OF ABBREVIATIONS

BT	Botulinum Toxin
BTX	Botox
CAF	Chronic Anal Fissure
cGMP	cyclic Guanosine-3-5-Mono-Phosphate
EAS	External Anal Sphincter
FDA	Food and Drugs Administration
GTN	Glyceryl Tri-Nitrate
GTN	Glyceryl trinitrate
IAS	Internal anal sphincter
IMA	Inferior mesenteric artery
ISDN	Iso sorbide di nitrate
LIS	Lateral Internal Sphincterotomy
MRAP	Maximum Resting Anal Pressure
NO	Nitric oxide
RCTS	Randomised controlled trials
rIGLEs	Rectal intra ganglionic laminar endings
RN	Rectal neck
PR	Puborectalis
S3	Sacral 3
S4	Sacral 4

INTRODUCTION

INTRODUCTION

Anal fissure is defined as a longitudinal ulcer occurring in the region of the dry, sensitive anoderm, extending from below the dentate line to above the anocutaneous line. In 90% of cases, it occurs in the posterior midline. The second most common location of the fissure is the anterior commissure (**Jost, 1990**).

In females, about 80% of anal fissures are located posteriorly while 20% occur anteriorly (**Fry, 1997**).

Chronic anal fissure is a non-healing linear tear in the distal anal mucosa below the dentate line. An anal fissure is likely to be non-healing if the fissure persists beyond 4 weeks. A chronic fissure can be identified by the presence of indurated edges, visible internal sphincter fibers at the base of the fissure, a sentinel polyp at the distal end of the fissure or a fibroepithelial polyp at the apex. A chronic fissure classically occurs at the posterior midline position (6 o'clock position), with the anterior midline position occurring in 10% of females and 1% of males (**Van, 2006**).

There have been changes to the hypothesis of the development of anal fissures over years. The initial hypothesis was that of anal canal trauma, most commonly by the passage of hard stool or bouts of diarrhea. Subsequent studies have elucidated two additional factors that may account for the persistence of chronic anal fissures. The first factor is the presence of persistently high basal internal sphincter tone in the majority of individuals with chronic anal fissures. The second factor is the presence of ischemia causing non-healing of the anal fissure (**Utzig et al., 2003**).

The principle aim of the treatment of chronic anal fissures is to decrease internal sphincter tone and hence increase the blood flow with subsequent tissue healing. Treatment options include pharmacological and surgical means (**Aaron et al., 2010**).

There are different surgical modalities for the treatment of chronic anal fissure. Lateral internal sphincterotomy (LIS) is widely accepted as the procedure of choice in the management of anal fissures. It promises rapid relief of pain, simple to be performed, and most patients express satisfaction with the result. Lateral sphincterotomy may be performed using an open or closed technique, and under general or local anesthesia. It can be performed as an in office procedure (**Pernikoff et al., 2005**).

Nitric oxide (NO) has been shown to be an inhibitory neurotransmitter in the internal anal sphincter. Glyceryl trinitrate (GTN) ointment applied to the anus leads to a significant fall in the maximum anal resting pressure (MARP) by 20-27% and an increase in anoderm blood flow, thus resulting in healing of the fissure (**Lund and Scholefeld, 1997**).

Botulinum toxin (BTX) action on hyperactive smooth muscles such as the anal sphincter is mediated by its action on the autonomic nervous system (as in striated muscles). The treatment goal for BTX is the interruption of the internal sphincter spasm and thereby, the ischemic state. Indeed, sphincter manometry after BTX injection has demonstrated a lowering of resting intranal pressure (**Uwe, 2008**).

AIM OF THE WORK

This is a prospective randomized study comparing the classic internal anal sphincterotomy, with botulinum toxin injection into the internal anal sphincter and with medical sphincterotomy using topical GTN ointment application as a treatment for chronic anal fissure.

EMBRYOLOGY OF THE ANAL CANAL

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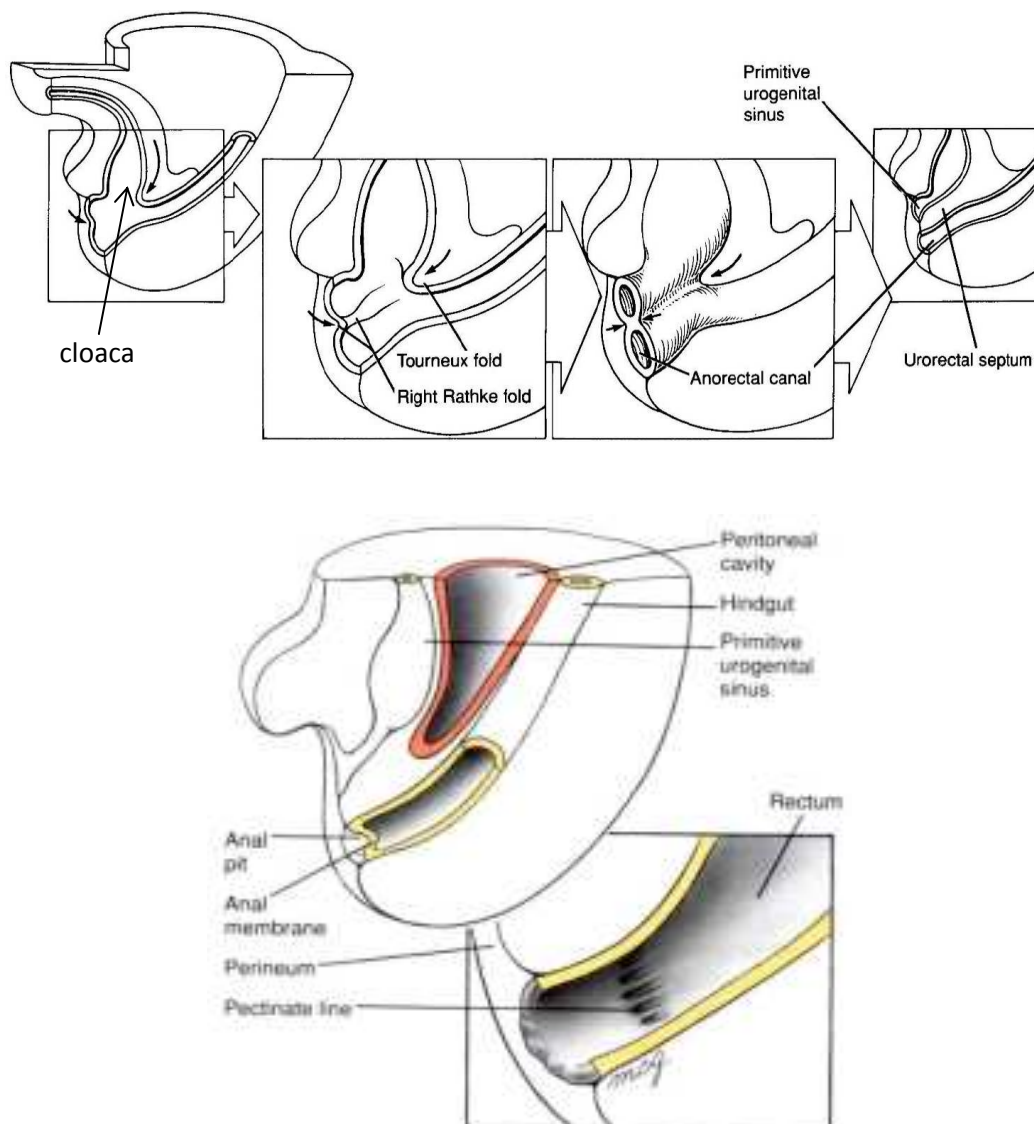
The anal canal is derived from cloaca, the enlarged caudal part of the digestive tract, which very early gives rise ventrally to the allantoic stalk and ventrolaterally receives the mesonephric ducts. Caudal to the allantois, the endodermal ventral wall of the cloaca is in contact with the ectoderm of the ventral body wall, and this apposition forms the cloacal membrane. Caudal to the cloaca and cloacal membrane, there is at first an extension of the gut into the tail of the embryo, but this tail normally become atretic, leaving the cloaca as the terminal part of the gut **(Hollinshead, 1990)**.

The cloaca is subdivided by growth of the urorectal or cloacal septum, a mass of mesoderm in the angle between the allantois and the hindgut at the cranial end of the cloaca, into the ventrally situated bladder and urogenital sinus and the dorsally situated rectum and upper 2/3 rds of anal canal. This septum does not grow in the direction of the cloacal membrane, and fusion of these structures is likewise never observed. The cloaca remains as such until the cloacal membrane ruptures by apoptotic cell death **(Nievelstein et al., 1998)**.

External to the cloacal membrane, anal tubercles develop and fuse with each others, then extend around the side of the bowel to meet and fuse with the posterior surface of the urorectal septum, thus providing a part of the anal canal with an ectodermal rather than an endodermal lining **(Hollinshead, 1990)**.

At the junction of these two zones, the columnar epithelium of the upper two thirds of the anal canal shades off into stratified squamous epithelium of the lower third and the area has been termed as “pectin” or “pectinate line” because its upper limit is the line of the anal valves is found at the disital ends of the grooves lying between the anal columns of Morgagni **(Ketth and Arthur, 1985)**.

So, the pectinate line (dentate line) is a line which divides the anal canal into upper 2/3 rds and lower 1/3 rd. Developmentally, this line represents the hindgut-proctoderm junction. It is an important anatomical landmark and several distinctions can be made based upon the location of a structure relative to this line (**Hollinshead, 1990**).



Figure(1): Embryology of anorectal region

Quoted from (**larsen, 1997**)

The external anal sphincter is developed from the mesoderm of the anal tubercles and its presence is not dependent upon the formation of a perforate anal canal; so an external sphincter is present in imperforate anus (**Hollinshead, 1990**).

ANATOMY OF THE ANAL CANAL