A STUDY ON ASSOCIATION BETWEEN PROSTATE GLAND ENLARGEMENT AND INGUINAL HERNIA

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GOVERNMENT CHENGALPATTU MEDICAL COLLEGE

CHENGALPATTU – 603001.

MAY 2020

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I**, DR. GOKUL PRABHU. B** solemnly declare that the dissertation titled **"A STUDY ON ASSOCIATION BETWEEN PROSTATE GLAND ENLARGEMENT AND INGUINAL HERNIA"** has been prepared by me. This is submitted to The **Tamil Nadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of M.S. Degree (Branch I) General Surgery.

Place: Chengalpattu **Dr. Gokul Prabhu B**

Date: **Reg No: 221711255**

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This is to certify that this dissertation titled **"A STUDY ON ASSOCIATION BETWEEN PROSTATE GLAND ENLARGEMENT AND INGUINAL HERNIA"** submitted by **DR. GOKUL PRABHU. B** to the faculty of General Surgery, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. Degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from APRIL 2018 to APRIL 2019.

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INTRODUCTION

Prostate Gland Enlargement is an important cause of Bladder Outlet Obstruction in males leading to chronic straining on micturition. Inguinal Hernia can be precipitated by Chronic Straining for Micturition[1,2]. The occurrence of Inguinal Hernia and Prostate Gland Enlargement with accompanied urinary tract obstructive symptoms are related to age. Prostate Gland Enlargement also predisposes to hernia and aggravation of symptoms related to hernia.

In elderly males Inguinal hernia and Symptomatic prostate gland enlargement are found in high frequency. On the basis of this evidence significant correlation between Inguinal hernia and obstructing prostate gland enlargement may be expected[2].

Several standard General Surgical text books give chronic straining due to Prostate Gland Enlargement as an etiological factor for Inguinal hernia in elderly male population[1,5,13]. But some of the studies showed that their occurrence together is considered a chance co-existence rather than cause and effect[3,14,15,16]. This study is aimed to find out whether Prostate Gland Enlargement is a significant risk factor for developing Inguinal Hernia in males.

AIM OF THE STUDY

- To find out the incidence of Prostate Gland Enlargement (Benign Prostatic Hyperplasia) among male patients with inguinal hernia.
- To find out whether there is any causal association between Prostate Gland Enlargement (Benign Prostatic Hyperplasia) and Inguinal Hernia.

REVIEW OF LITERATURE

INGUINAL HERNIA

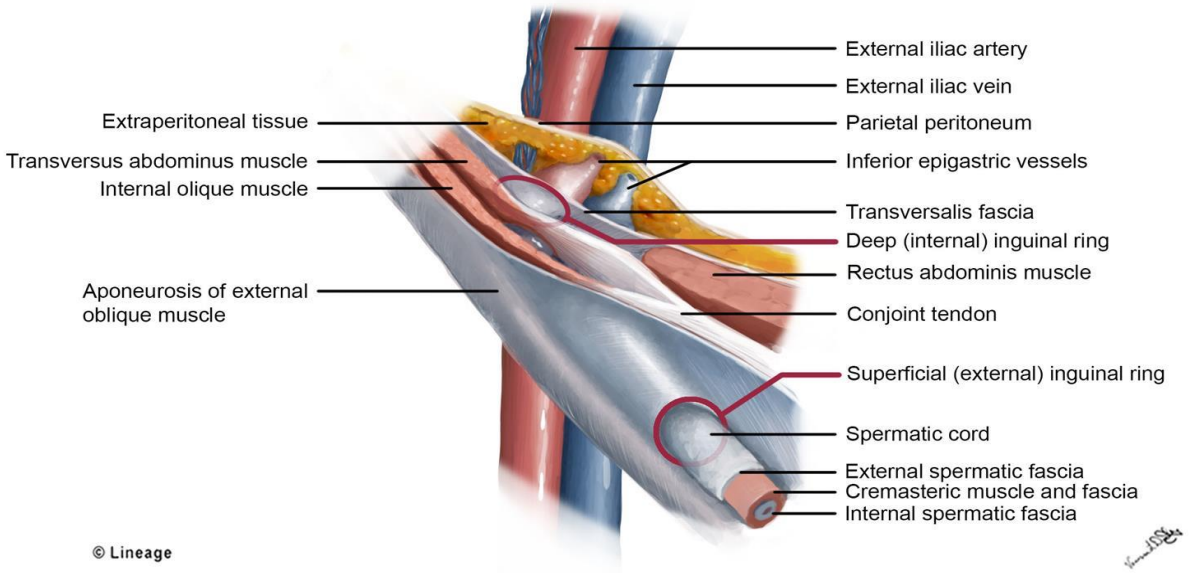
A hernia is a protrusion of a viscus or part of a viscus through an abnormal opening in the wall of its containing cavity [1].

The external abdominal hernia being the commonest variety, most frequent of it is the inguinal hernias. Important components of hernia are the hernia orifice and the hernia sac. Hernial orifice is the defect in the innermost aponeurotic layer of the abdominal wall and the hernia sac is the out pouching of the peritoneum. Neck of the sac corresponds to the hernia orifice.

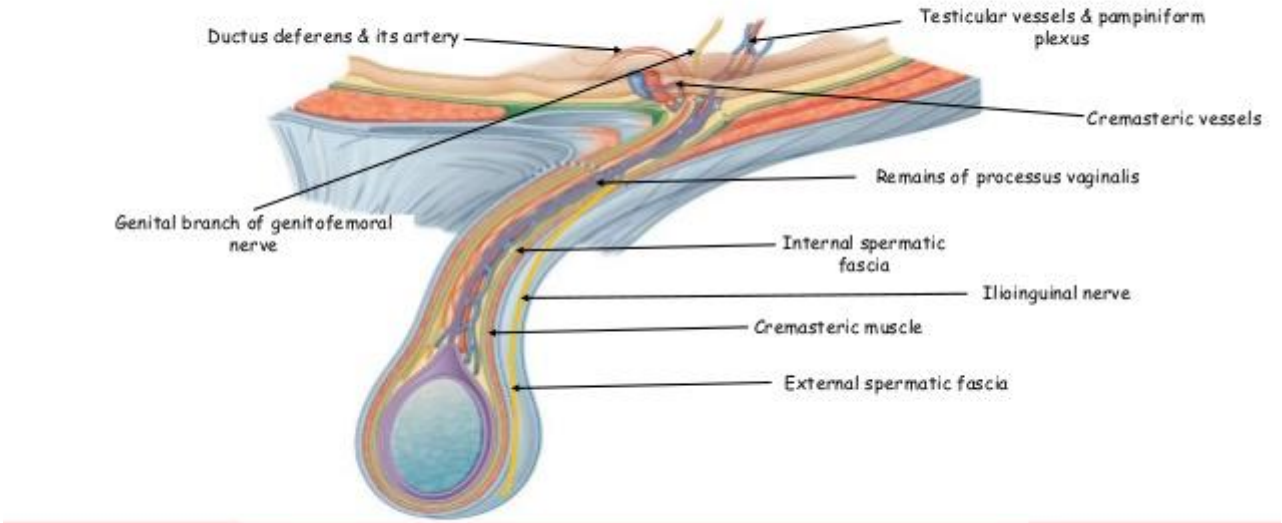
INGUINAL HERNIA – ANATOMY [4,5]

The groin region is a complex network of muscles, ligaments, and fascia that are interwoven in a complex fashion. Inguinal canal is 4 to 6 cms long and is situated in the anteroinferior portion of the pelvic basin. Shaped like a cone, its base is at the superolateral margin of the basin, with its apex pointed inferomedially towards the symphysis pubis. The canal begins intra abdominally on the deep aspect of the abdominal wall, where the spermatic cord passes through a hiatus in the transversalis fascia (in females, this is round ligament). This hiatus is termed the deep or internal inguinal ring. The canal ends at the superficial aspect of the abdominal musculature at the superficial ring, which is a defect in the external oblique aponeurosis.

Inguinal Canal



CONTENTS OF INGUINAL CANAL



Anteriorly the wall of the inguinal canal is formed by the external oblique aponeurosis and the internal oblique muscle laterally. Posteriorly, the wall is formed by the fusion of the transversalis fascia and the transverses abdominus muscle, although up to one fourth of persons are found to have only the transversalis fascia covering the posterior wall. Roof is formed by an arch formed by the fibres of the internal oblique muscle and the floor is formed by the inguinal ligament and the lacunar ligament.

The inguinal ligament is also known as the Paupart ligament and is comprised of the inferior fibres of the external oblique aponeurosis. The ligament stretches from the anterior superior iliac spine to the pubic tubercle. Cooper's ligament is otherwise known as the pectineal ligament. It is the lateral portion of the lacunar ligament that is fused to the periosteum of the pubic tubercle. The lacunar ligament, or the ligament of Gimbernat, is the triangular fanning out of the inguinal ligament as it joins the pubic tubercle.

Nerves of interest in the inguinal region are the ilioinguinal, iliohypogastric and genital branch of genitofemoral nerve. The ilioinguinal and iliohypogastric nerve arise together from first lumbar nerve (L1). The ilioinguinal nerve enters the inguinal canal by piercing the internal oblique muscle and exit through the superficial ring. The nerve supplies the skin of the upper and medial thigh and in males it also supplies the penis and upper scrotum, while supplying the monspubis

And labium majus in females.The iliohypogastric nerve arises from T12-L1 and follows the ilioinguinal nerve. After the iliohypogastric nerve pierce the deep abdominal wall, it courses between the internal oblique and transeversus abdominis, supplying both. Then it branches in to a lateral and an anterior cutaneous branch, which pierces the internal oblique aponeurosis and then external oblique aponeurosis above the superficial inguinal ring. The Genitofemoral nerve arises from L1-L2, courses along the retroperitoneum, and emerges on the anterior aspect of the psoas. It then divides into genital and femoral branches. The genital branch enters the inguinal canal just lateral to inferior epigastric vessels. In males it travels through the superficial inguinal ring and supplies the scrotum and the cremaster muscle. In females, it supplies the mons pubis and labium majora.

Myopectineal Orifice of Fruchaud[4]: Fruchaud believed that all hernias of the groin begin within the groin; in an area he named the myopectineal orifice. This area in the groin is bounded superiorly by Arch of internal oblique muscle and transversus abdominis muscle, laterally by the Iliopsoas muscle, medially by lateral border of rectus muscle and its anterior lamina and inferiorly Pubic pectin. Running diagonally through the myopectineal orifice is the inguinal ligament. The orifice is covered by the transeversalis fascia.

INGUINAL HERNIA – EPIDEMIOLOGY [1,5,6,7]

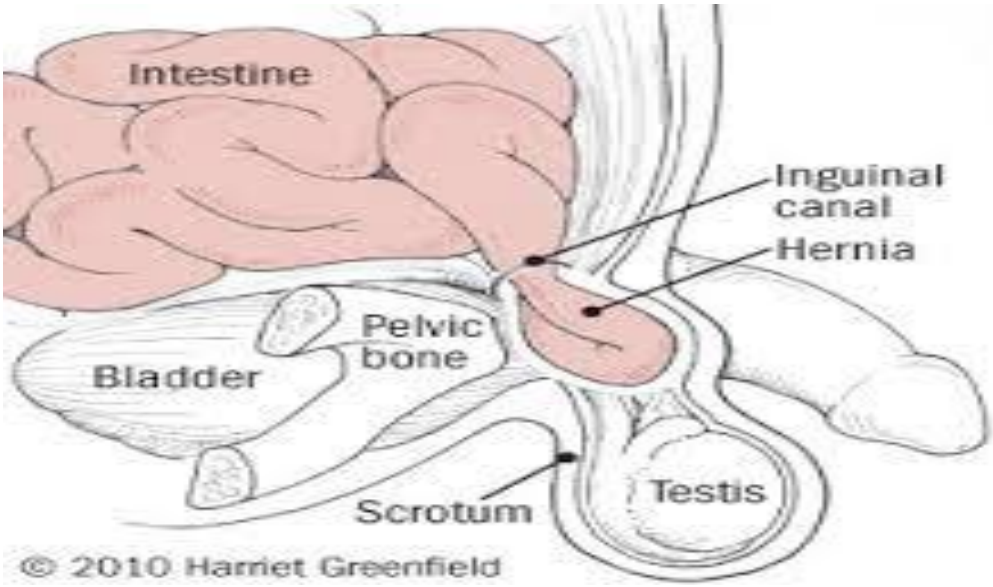
Inguinal hernia repair is one of the most commonly performed procedures by a General Surgeon. It is difficult to estimate the exact prevalence of inguinal hernia in the general population, but an overwhelming majority of the procedures are done in males 90% vs. 10% in females. Approximately 70% of femoral hernia repairs are done in females but inguinal hernia repairs are 5 times more common in females than femoral hernia repairs. Incidence of hernia have a bimodal distribution in males with peaks before 1 year of age and then again after 40 years of age. Indirect inguinal hernias are the most common hernias in both men and women; a right-sided predominance exists.

Exact prevalence of inguinal hernia in Indian population is not available. An extrapolated statistical analysis from a study given by Asia Pacific Hernia Society states that around 1,95,7850 people are affected by inguinal hernia in India[7].

INGUINAL HERNIA – ETIOLOGY

Inguinal hernias may be considered congenital or acquired diseases. A number of studies have attempted to delineate the precise cause of inguinal hernia formation; however, the risk factors are likely multifactorial.

INGUINAL HERNIA



Presumed Causes of Groin Herniation[2,3,5,13]

COPD	Birth weight < 1500g
Coughing	Positive family history
Pregnancy	Cigarette smoking
Straining – prostatism	Ascitis
Straining – constipation	Valsalva maneuver
Obesity	Connective tissue diseases
Previous Right Lower Quadrant incision	Defective collagen Synthesis
Arterial aneurysms	Upright posture
Heavy lifting	Physical exertion

Congenital hernias are considered to be an impedance to the normal development, rather than acquired weakness. During the normal course of development, the testes descent from the intraabdominal space into the scrotum in the third trimester. Their descent is preceded by the gubernaculums and a diverticulum of peritoneum, which protrude through the inguinal canal and ultimately becomes the processus vaginalis. Processus vaginalis usually closes between 36 and 40 weeks, failure of which results in patent processus vaginalis and this explains the high incidence of indirect inguinal hernia in preterm babies.

The introduction of continuous ambulatory peritoneal dialysis for renal failure has demonstrated, as did ascites, that a patent processus vaginalis or canal of nuck, if subjected to increased intraabdominal pressure over a period of time, will dilate and produce a Hydrocele or hernia. Carcinomatosis, decompensated liver or heart disease can therefore present as recent onset herniation.

Microscopic examination of skin of inguinal hernia patients demonstrated significantly decreased ratios of collagen type I to collagen type III. Type III collagen have less tensile strength than type I collagen. Additional analysis of the skin revealed disaggregated collagen tracts with decreased collagen fiber density. These connective tissue defects are seen systemically – in skin, lung, pericardium etc. Most common cause for these

changes is found to be heavy smoking and the condition is termed as metastatic emphysema. Therefore, long term tobacco exposure is a risk for hernia formation. Patients with congenital connective tissue disorders like Ehler Danlos Syndrome, due to defective connective tissue, leading to less tensile strength of their tissues, can have hernia formation.

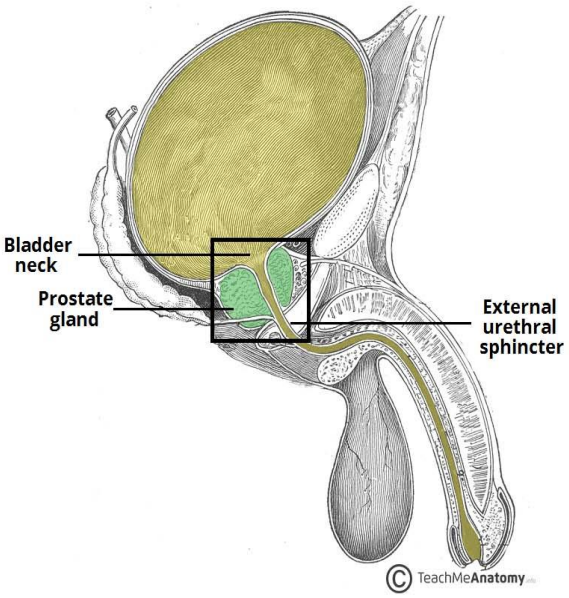
Historically, hernia causation was attributed to a mechanical disparity between visceral pressure and the resistance of the musculature. Cooper not only identified Transversalis fascia, but also pointed out it was the last barrier to groin hernia formation. Therefore, factors that increases intraabdominal pressure – cough, obesity, unusual exertion, pregnancy, prostatism in old age patients, over the long time can lead on to hernia formation. However, this occurs in a combination with a patent processus vaginalis or through age related weakness of the abdominal musculature is not clear. Both Inguinal Hernia and symptomatic Benign Prostatic Hyperplasia are often found together in increased frequency in elderly [2,3]. On the basis of this evidence significant correlation between inguinal hernia and obstructing benign prostate enlargement may be expected.

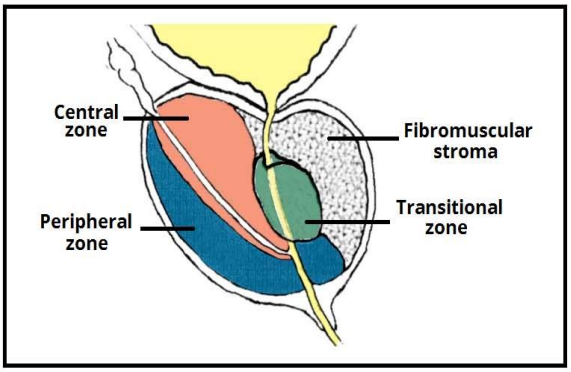
PROSTATE – ANATOMY[4,9]

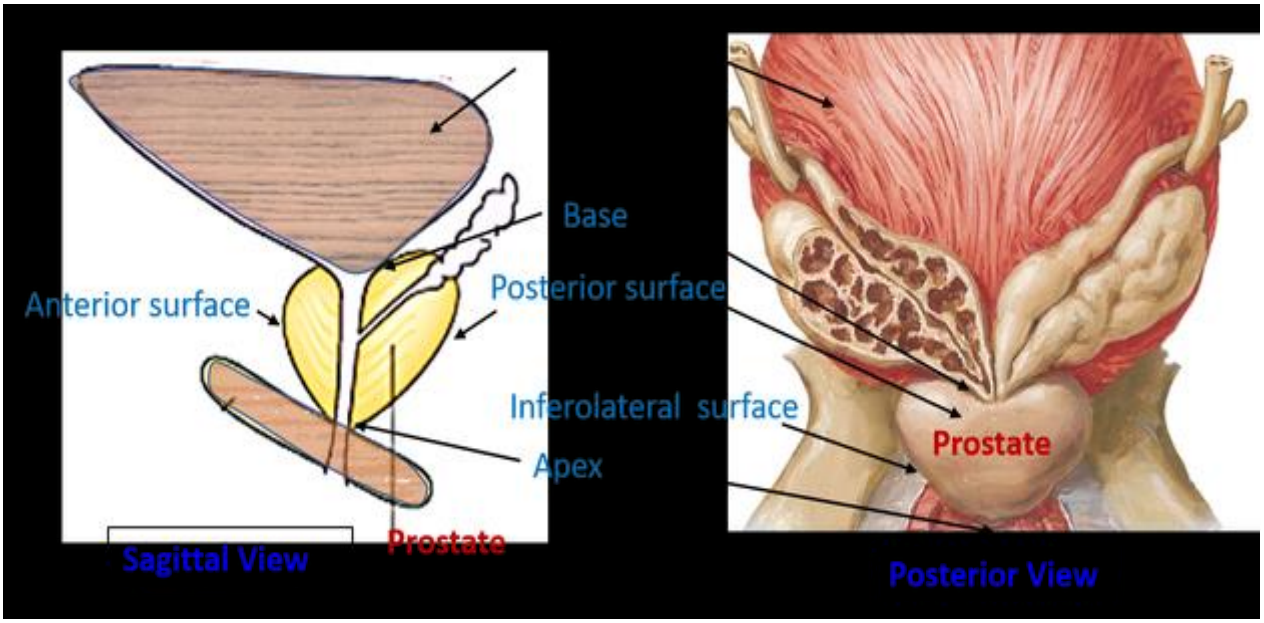
The prostate gland is formed around the end of the third month (first trimester) from the epithelium of the future prostatic urethra. The epithelium proliferates and penetrates the surrounding mesenchyme, which is the future fibro muscular prostatic tissue.

The classical description of the adult prostate is that it has the size, shape, and consistency of a large chestnut. The form of the prostate is that of a compressed inverted cone: pyramidal, having a base and an apex. It is located between the vesical neck of the bladder and the apex of the urogenital diaphragm. The normal weight of the prostate in a young adult is from 17 to 19g. The numbers 4, 3, 2 are useful as a mnemonic for remembering the transverse, vertical, and sagittal dimensions in centimeters, respectively, of the gland. The true capsule of the prostate is covered by Extraperitoneal tissues which also covers the proximal male urethra. The prostate is fixed to its location by Puboprostatic ligaments, Urogenital diaphragm, Bladder, Prostatic sheath and Fascia of Denonvilliers.

Anatomy of the Prostate







Prostatic Surfaces

There are four prostatic surfaces: one posterior, one anterior, and two inferolateral. The posterior surface is flat transversely and convex vertically. It is separated from the rectal ampulla by the bilaminar fascia of Denonvilliers. This surface is characterized by a midline groove that is wider toward the base of the gland, and serves to partially separate the gland posteriorly into left and right lobes. The posterior surface may be palpated by digital rectal examination. The vesicoprostatic junction is located at the upper border of the posterior surface.

The narrow and convex anterior surface is located between the apex and the base. Multiple large veins separate this surface from the symphysis pubis. The avascular puboprostatic ligaments are fibrous cords, wide or narrow. They connect the upper limits of the anterior surface of the prostate to the pubic bone, at the right and left sides of the cartilaginous area.

The right and left inferolateral surfaces are embraced by the anterior part of the levator ani muscles. They are fixed to the levator by the arcus tendineus of the fascia pelvis, sagittal connective tissue bands between the ischial spine, and the pubic bone. Here there is a very rich venous network and fibrous tissue which contributes part of the lateral prostatic sheath.

Prostatic Urethra

The prostatic urethra begins at the urethral meatus at the apex of the trigone of the bladder. This opening is crescent-shaped, invaginated posteriorly by a protuberance caused by the underlying glandular tissue (median lobe of the prostate), thus forming the uvula vesicae. This is continuous with a posterior midline urethral ridge, or crest, in the urethra. The urethral ridge has a distinctly expanded portion called the verumontanum, or seminal colliculus.

Prostate – Structure

McNeal described four regions or zones in the prostate: peripheral, central, transition, and anterior fibromuscular stroma. The urethra is the key anatomic entity defining these regions. Posterior to the urethra is the glandular area. Anterior to the urethra is the fibromuscular area; that is, the ventral portion of the glandular prostatic tissue is covered by the fibromuscular stroma.

Peripheral Zone:

It is likely that the glands of this zone develop from the urogenital sinus and drain into the prostatic urethra. Nearly 75% of the glandular prostate, the peripheral zone surrounds most of the central zone and much of the urethra; in other words, it surrounds the posterior and lateral areas of the prostate gland. Its glands drain into the prostatic urethra. This zone is formed

by multiple tubuloalveolar glands. The long, narrow ducts of this zone branch into small, round, regular acini with smooth, nonseptate walls. Epithelium is simple columnar; its pale cells have distinct borders and basally-placed small, dark nuclei. Most carcinomas develop in the peripheral zone.

Central Zone:

Ducts of this zone are probably of Wolffian origin. The central zone, which is nearly 25% of the glandular prostatic parenchyma, envelops the ejaculatory ducts and extends toward the base of the urinary bladder. The central zone is continuous with the peripheral zone and, like the peripheral zone, is formed by several tubuloalveolar glands (mucosal, submucosal, main prostatic) which are located around the urethra.

The acinar tissue consists of large, irregularly shaped spaces; the walls have intraluminal ridges or septa. The cells of the central zone differ significantly from those of the peripheral zone. They have more opaque, granular cytoplasm and less distinct cell membranes. Their cell length varies, they have an irregular luminal border, and they appear more crowded.

Their nuclei, which are slightly larger than those of the peripheral zone and stain paler, are displaced to variable levels from the basement membrane. Carcinoma seldom arises in the central zone.

Transition Zone:

Glands in the transition zone are formed from the junction of the proximal and distal urethral segments. This zone is less than 5% of the glandular prostate. The transition zone is composed of two minute glandular regions which are lateral to the preprostatic sphincter and directly related to the proximal urethral segment.

The periurethral region is related to this zone and to the junction of the proximal and distal urethral segments. Periurethral ducts, which are responsible for the genesis of benign prostatic hyperplasia, are present. In this zone one observes a minimal number of glands. Benign Prostatic Hyperplasia occurs mainly in Trasition zone and periurethral gland region. The area near or within the sphincter almost invariably produces the most numerous and largest nodules. Ten to twenty percent of carcinomas may develop in the transition zone.

Stroma:

The anterior fibromuscular stroma is nonglandular. It constitutes ⅓ of the prostatic tissue within the prostatic capsule but is in continuity with the detrusor muscle of the neck of the urinary bladder. It is heavily fixed with the anterior surfaces of the three glandular zones, and represents the periurethral gland region. The fibromuscular stroma is composed of striated and smooth muscles, as well as elastin and collagen.

Capsules of the Prostate

There are three capsules of the prostate; two (the true and false) are anatomic the third is pathologic. The true capsule is a very thin covering surrounding the gland in toto. The false capsule (periprostatic fascia or prostatic sheath) is an extraperitoneal fascia (visceral layer of endopelvic fascia). This capsule is continuous with 4 fasciae; anteriorly, fascia of the bladder, puboprostatic ligament, laterally, arcus tendineus of the fascia pelvis, Posterior: fascia of Denonvilliers and urogenital diaphragm inferiorly. Between the true and false capsules is a venous plexus, the prostatic or pudental venous plexus.

Part of the normal aging process is progressive prostatic growth due to benign prostatic hyperplasia (BPH), the peripheral part of the prostate becomes compressed against the surrounding endopelvic connective tissue, forming a surgical capsule (pathologic capsule). When enucleation of the prostate is performed, the plane between the compressed peripheral tissue and the adenomatous tissue permits removal of the adenoma, leaving behind the peripheral condensed prostatic tissue and the anatomic capsule.

Prostate Vascular Supply

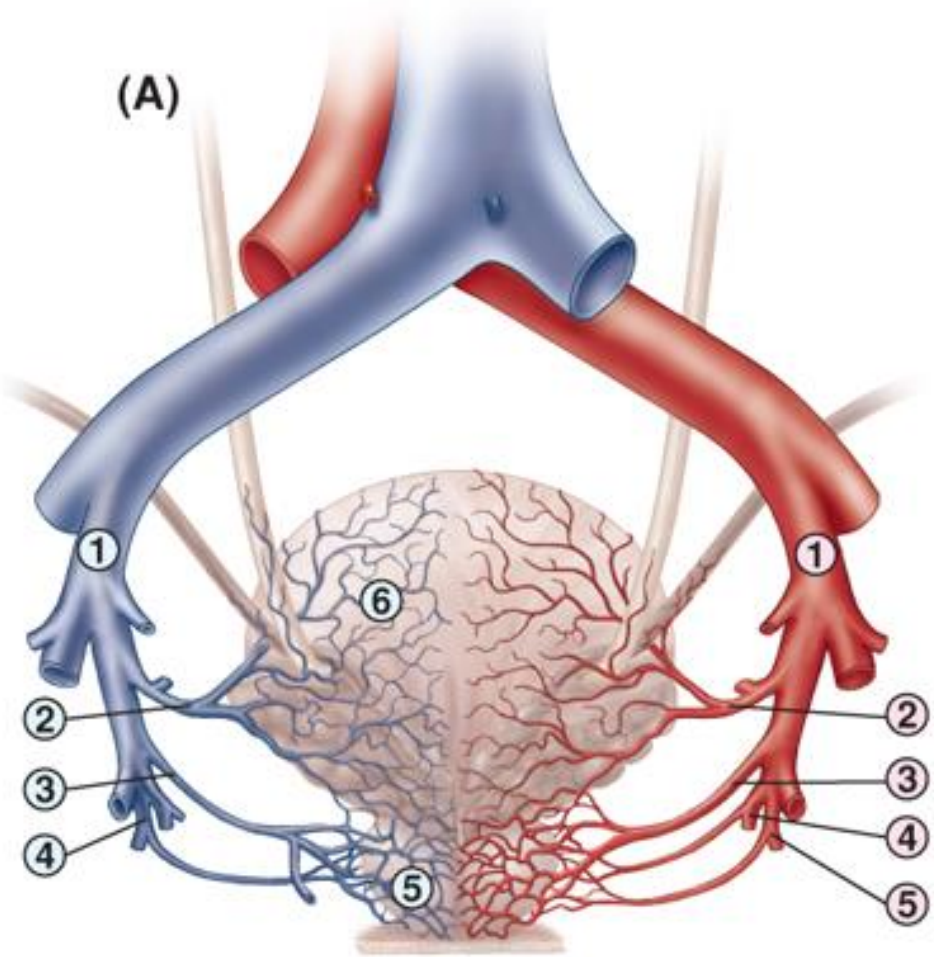
Arterial Supply:

The blood supply of the prostate is derived primarily from inferior vesical artery. A branch of this artery enters the prostate laterally at the prostatovesical junction. This artery divides into two branches, the peripheral and the central. The peripheral branch serves the majority of the prostatic parenchyma; the central branch supplies the urethra and the periurethral tissues. Other arteries contributing rami to the prostate are the internal pudental and middle rectal arteries. The middle rectal artery is considered to be poorly named, since most of its blood goes to the prostate gland.

Venous Drainage:

There is a rich venous plexus (prostatic plexus) between the prostate gland and the prostatic sheath. It communicates with the internal iliac venous system and the presacral veins. The prostatic venous plexus receives the deep dorsal penile vein and the veins of the base of the bladder. The vesical and internal iliac veins receive most of the venous blood.

Blood Supply of Prostate

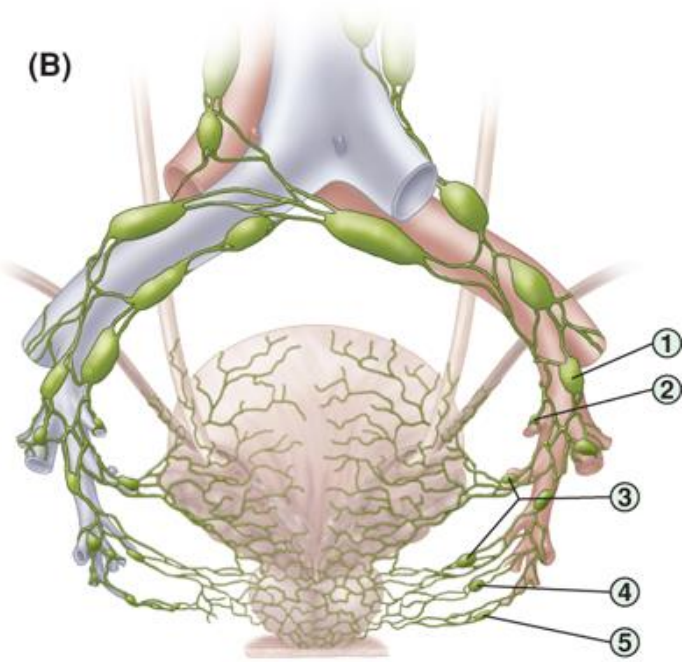


Veins	Arteries
1. Internal iliac	1. Internal iliac
2. Superior vescial	2. Superior vesical
3. Inferior vesical	3. Inferior vesical
4. Internal pudendal	4. Middle rectal
5. Prostatic venous plexus	5. Internal pudendal
6. Vescial venous plexus	

Source: Mark. H. Hankin, Dennis.E. Morse, Carol. A. Bennett-Clarke: Clinical Anatomy: A Case Study Approach

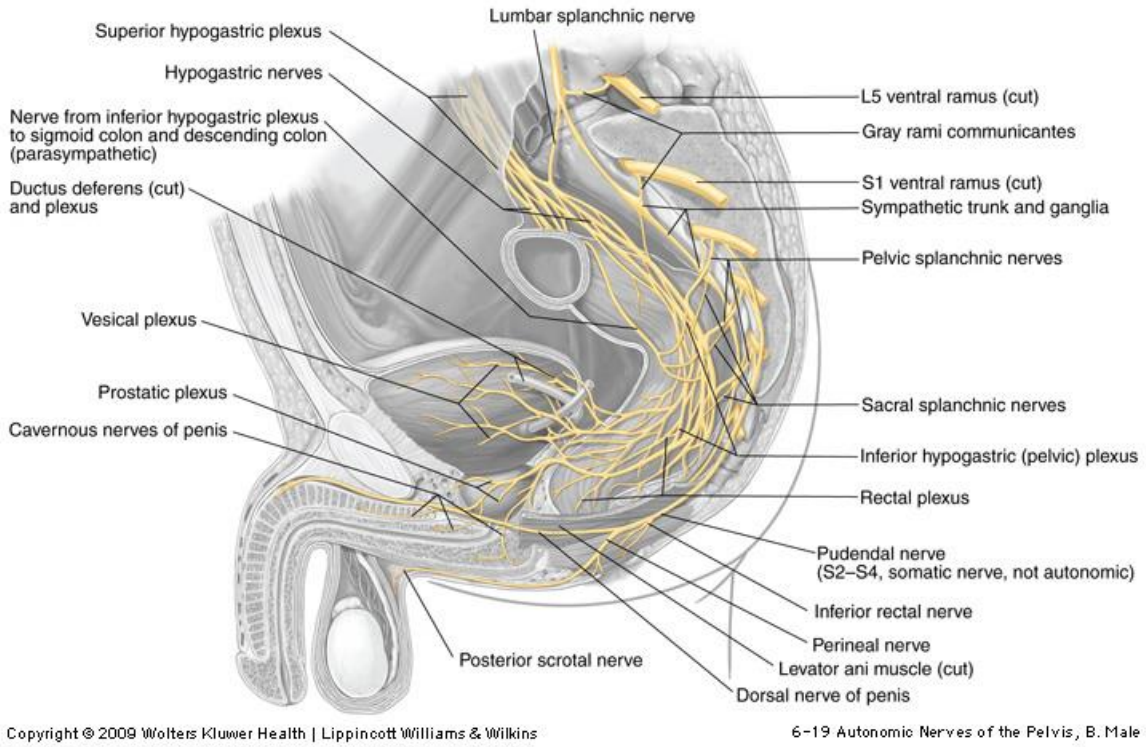
Lymphatic Drainage:

From the prostatic acinus, large intraprostatic trunks are formed. These penetrate the prostatic capsule and form the periprostatic lymphatic plexus. This plexus yields lymphatic vessels which follow the vascular network of the prostatovesical arteries. The lymph vessels that follow the prostatovesical arteries travel to the internal iliac lymph nodes. The vessels also travel to the presacral lymph nodes and, occasionally, to the external iliac lymph nodes.



Prostate – Innervation

The preganglionic sympathetic nerve supply to the smooth muscle of the seminal vesicles, ejaculatory ducts, and prostate gland arises in the intermediate gray area of spinal cord levels L1 and L2 (or L3). Postganglionic fibres arise in the preaortic or pelvic plexuses. Smooth muscle contraction and seminal fluid expulsion are components of sympathetic activity . Parasympathetic fibres from sacral cord levels S2, S3,and S4 synapse in pelvic ganglia and periprostatic ganglia. Stimulation of secretion from prostate gland and blood vessel dilatation are components of parasympathetic fibers.



6-19 Autonomic Nerves of the Pelvis, B. Male

Histology of Prostate

The glandular epithelium covers approximately seventy percent of the prostate mass and the rest is fibromuscular. The glandular part contains ducts and acini which are lined with columnar epithelium and drain in the posterior and lateral walls of the prostatic urethra. In all regions, ducts and acini are lined with secretory epithelium, with a layer of basal cells and interspersed endocrine- paracrine cells beneath. The peripheral zone has small, rounded, uniform glands. The central and transitional zones have very large and irregular acini.

Physiology of Prostate

The prostate gland secretes alkaline fluid. The sperm inside the vaginal tissue and ductus deferens produce acidity which inhibits fertilization and prostatic gland fluid plays a mojor role in neutralizing it .it also enhances the fertility and motility of the sperm. The prostatic fluid also contains, phosphorus, calcium, citric acid and other substances.

BENIGN PROSTATIC HYPERPLASIA[9]

Benign prostatic hyperplasia (BPH) may contributes to, but is never the very cause of, lower urinary tract symptoms (LUTS) in elderly males. The cause and effect relationships were not established to confirm the underlying etiology of prostatic growth in elderly males.

For example, androgens are not proved to be clearly having a causative effect on BPH. The nomenclature of voiding dysfunction in aging men is confusing and often inaccurate. The term BPH should be used with reference to the histologic process of hyperplasia, which can be demonstrated microscopically.

Men with benign prostatic enlargement (BPE) presumably have an increase in total prostate volume because of BPH. BPE may or may not produce clinically significant LUTS and may or may not produce urodynamically proven bladder outlet obstruction.

ETIOLOGY OF BENIGN PROSTATIC HYPERPLASIA

BPH is but one cause of the LUTS in aging men commonly, and probably incorrectly, referred to as prostatism. Histopathologically, increased number of stromal and epithelial cells in prostate are characteristic of BPH.

The observed increase in cell number may be due to epithelial and stromal proliferation or to impaired programmed cell death leading to cellular accumulation. Androgens, estrogens, stromal-epithelial interactions, growth

factors and neurotransmitters may play a role, either singly or in combination, in the etiology of the hyperplasic process.

Hyperplasia

In a given organ, the number of cells, and thus the volume of the organ, is dependent upon the equilibrium between cell proliferation and cell death. An organ can enlarge not only by an increase in cell proliferation but also by a decrease in cell death. The relative role of cell proliferation in human BPH is questioned because there is no clear evidence of an active proliferative process.

Although it is possible that he early phases of BPH are associated with a rapid proliferation of cells, the established disease appears to be maintained in the presence of an equal or reduced rate of cell replication and increased expression of antiapoptotic pathway genes. inhibition of cell death and cellular proliferation and differentiation are caused by androgens. The hyperplasia results in a remodeling of the normal prostatic architecture. Epithelial budding from pre-existing ducts and the appearance of mesenchymal nodules characterize the early stages of the process. When the proliferating cell matures through a process of terminal differentiation, they have a finite life span before undergoing programmed cell death. Blockage of this maturation process is caused by ageing resulting in reduced progression of cell to terminally differentiated cells which in turn reduces the overall rate of cell death.

The Role of Androgens

BPH requires the presence of androgens during prostate development and puberty but androgens itself are not responsible for BPH. In converse, ageing patients, patients who are castrated before puberty and who are affected by genetic disease that impair androgen action or production do not tend to develop BPH. Inspite of decreasing peripheral levels of testosterone in ageing, the prostatic levels of androgrn receptor and dihydrotestosterone remains very high. Moreover, androgen withdrawal leads to partial involution of established BPH. Assuming normal ranges, there is no clear relationship between the concentration of circulating androgens and prostate size in aging men.

In skeletal muscle, brain and seminiferous epithelium, testosterone directly stimulates androgen-dependent processes. In the prostate, however, the nuclear membrane bounded enzyme steroid 5 α –reductase converts the hormone testosterone into DHT, the principal androgen in the tissue. DHT derived from testicular androgens covers ninety percentage of total prostatic androgen. Both testosterone and DHT bind to the same androgen receptor protein inside the cell. The hormone receptor then binds to specific DNA binding sites in the nucleus, which results in increased transcription of androgen-dependent genes and stimulation of protein synthesis.

Androgen withdrawal from androgen-sensitive tissue results in a decrease in tissue involution and protein synthesis. Besides inactivation of key androgen-dependent genes (e.g., prostate-specific antigen), androgen withdrawal leads to the activation of specific genes involved in programmed cell death. Inspite of the importance of androgens in normal prostatic development and secretory physiology, there is no evidence the either DHT or testosterone serves as the direct mitogen for prostatic growth in elderly males. However, many growth factors and their receptors are regulated by androgens. The autocrine and paracrine pathways indirectly mediate the action of both testosterone and DHT.

Androgen Receptors

The prostate, unlike other androgen dependent organs, maintains its ability to respond to androgens throughout life. AR levels in the prostate remain high throughout aging. In fact, there is evidence to suggest that nuclear Androgen Receptor levels may be higher in hyperplastic tissue than in normal controls. Estrogen which increases over age and other factorsin an ageing prostate may increase the AR expression which leads to further growth of the gland.

5 α reductase enzyme

Two steroid 5 α reductase have been discovered, each encoded by a separate gene. Type 1: 5 α-reductase, the predominant enzyme in extraprostatic tissues, such as liver and skin, Type 2: 5α-rducataseis the predominant prostatic 5α-reductase. It is exquisitely sensitive to inhibition by finasteride and dutasteride. The type2 enzyme is critical to normal development of the prostate and hyperplastic growth later in life.

Immunohistochemical studies with type 2 5α-reducatase specific antibodies show primarily stromal cell localization of the enzyme. Epithelial cells uniformly lack type 2 protein. The stromal cell plays a central role in androgen-dependent prostatic growth and that the type 2 5α-reductase enzyme within the stromal cell is the key androgenic amplification step. Thus, a paracrine model for androgen action in the gland is evident. In addition, it is possible that circulating DHT produced in the skin and liver may act on prostate epithelial cells in a true endocrine fashion.

Polymorphism in the type 2 5α-reductase enzyme (SRD5A2) has been reported, but their linkage to BPH is uncertain. Androgen withdrawal may partially exert its effect on the prostate through vascular effects. There is indirect evidence to suggest that abnormalities in the prostatic vascular system produced by other disease states may be a risk factor of BPH.

The Role of Estrogens

There is animal model evidence to suggest that estrogens play a role in the pathogenesis of BPH. The role of estrogens in the development of human BPH, however, is less clear.

Serum estrogen levels increase in men with age, absolutely or relative to testosterone levels. There is also suggestive evidence that intraprostatic levels of estrogen are increased in men with BPH. Patients with larger volumes of BPH tend to have higher levels of estradiol in the peripheral circulation. At present, however, the role of estrogens in human BPH is not as firmly established as the role of androgens.

Stromal -Epithelial Interaction

There is abundant experimental evidence to demonstrate that prostatic stromal and epithelial cells maintain a sophisticated paracrine type of communication. This is strong evidence that one class of stromal cell excretory protein partially regulates epithelial cell differentiation. Thus, BPH may be due to a defect in a stromal component that normally inhibits cell proliferation, resulting in loss of a normal “braking” mechanism for proliferation.

The process of new gland formation in the hyperplastic prostate suggests a “reawakening” of embryonic processes in which the underlying prostatic stroma induces epithelial cell development. Many of the prostatic

stromal-epithelial interactions observed during normal development and in BPH may be mediated by soluble growth factors or by the extracellular matrix (ECM). CRY61 (An Early Immediate Reponses Gene) is an ECM-associated protein that promotes adhesion, migration, and proliferation of epithelial and stromal cells. CRY61 expression is significantly increased in human BPH tissues and is induced by lysophosphatidic acid (and endogenous lipid growth factor).

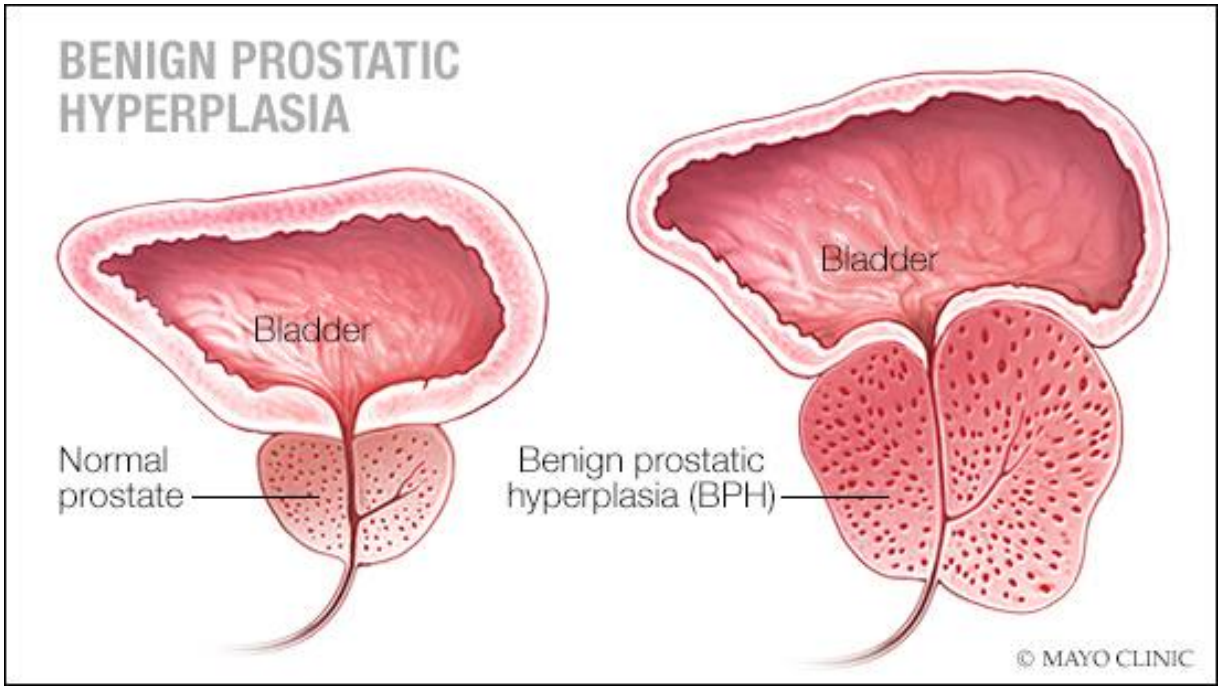
Growth Factors:

Growth factors are small peptide molecules that stimulate, or in some cases inhibit, cell division and differentiation processes; Cells that respond to growth factors have on their surface receptors specific for that growth factor that in turn are linked to a variety of transmembrane and intracellular signaling mechanisms. Interaction between growth factors and steroid hormones may alter the balance of cell proliferation versus cell death to produce BPH.

In addition to FGF-2, acidic FGF (FGF-1), Int-2 (FGF-3), keratinocyte growth factor (KGF, FGE-7), transforming growth factors (EGF) have been implicated in prostate growth. Similar mechanisms may be operational in BPH, leading to the accumulation of epithelial cells. If cellular proliferation isa component of the BPH process, it appears that growth stimulator factors such as FGR-1, -2, -7, and -17 families; vascular endothelial growth factor

(VEGF); and insulin-like growth factor (IGF) may play a role, with DHT augmenting or modulating the growth factor effects. In contrast, TGF-β, which is known to inhibit epithelial cell proliferation, may normally exert a restraining influence over epithelial proliferation that is lost or down regulated in BPH.

Although data on the absolute level of growth factor and growth factor receptors in hyperplastic as opposed to normal tissue are conflicting, it is likely that growth factors play some role in the pathogenesis of BPH.



Other Signaling Pathways

Sympathetic signaling pathways are important in the pathophysiology of LUTS. In addition, there is increasing evidence that sympathetic pathways may be important in the pathogenesis of the hyperplastic growth process. Alpha blockade, in some model systems, can induce apoptosis. Adrenergic pathways can also modulate the smooth muscle cell phenotype in the prostate all the components of the renin-angiotensin system (RAS) are present in prostatic tissue and may be activated in BPH. Either with or without sympathetic modulation, local RAS pathways may contribute to cell proliferation and smooth muscle contraction.

The Potential Role of Inflammatory Pathways and Cytokines in Benign Prostatic Hyperplasia

An additional source of growth factors in human BPH tissue may be the inflammatory cell infiltrates seen in many men with BPH. Thayer and associates (1992) reported extensive infiltration of human BPH tissues by activated T cells. Peripheral blood and tumor infiltrating T cells are known to express VEGF, a potent epithelial mitogen T cells are known to produce and secrete a variety of other growth factors including HB-EGF and bFGF/FGF-2. Thus T cell present in the local prostate environment were thought to be capable of secreting potent epithelial and stomal mitogens that promote stromal and glandular hyperplasia

A large number of cytokines and their receptors are seen in BPH tissue.

Specifically, significant levels of IL-2, IL-4, IL-7, IL-17, interferon γ (IFEN- γ) and their relevant receptors are found in BPH tissue. Macrophage inhibitory cytokine I is expressed in normal prostate tissue but significantly down regulated in BPH. To date, however, no firm cause-and-effect relationships have been established between prostatic inflammation and related cytokine pathways and stromal-epithelial hyperplasia.

Genetic and Familial Factors

There is substantial evidence the BPH has an inheritable genetic component. Sanda and colleagues (1994) conducted a retrospective case-control analysis of surgically treated BPH patients and control subjects at Johns Hopkins. The BPH patients were men whose respected prostate weights were in the highest quartile (greater than 37 g) and whose age at prostatectomy was in the lowest quartile. The hazard-function ratio for surgically treated BPH among first-degree male relatives of the controls was 4.2 (95% confidence interval [CI] demonstrating a very strong relationship [9,10].

A segregation analysis showed that the results were most consistent with an Autosomal Dominant inheritance pattern. Approximately 50% of men undergoing prostatectomy of BPH at less the 60 years of age could be attributable to inheritable form of disease. In contrast, only about 9% of men

undergoing prostatectomy of BPH at more than 60 years of age would be predicated to have a familial risk [9,10]. However, the specific gene or genes involved in familial BPH or that contribute to the risk of significant prostatic enlargement in sporadic disease remain to be elucidated.

Other Etiologic Factors

Androgens and soluble growth factors are clearly not the only important factors for the development of BPH. All mammalian prostates studied have testosterone, DHT, and AR as well as most of the known growth factor signaling pathways; however, only dog and man develop BPH. Interestingly, another glandular organ that remains androgen responsive throughout life, the seminal vesicle, does not develop hyperplasia.

Obviously, other mechanisms or cofactors must be present in these two unique species making them susceptible to the disease. Non androgenic substances from the testis perhaps transmitted through the vas deferens or deferential blood vessels, for example, may play some role. Prolactin has long been speculated to play a role in BPH because of the known effects of this hormone on prostate cells in vitro.

However, despite the documented presence of prolactin receptors in the human prostate and low circulating levels of the hormone, the role of prolactin in human prostate disease is unclear.

PATHOPHYSIOLOGY OF BENIGN PROSTATIC HYPERPLASIA[9]

The pathophysiology of BPH is complex. Prostatic hyperplasia increases urethral resistance, resulting in compensatory changes in bladder function. However, the elevated detrusor pressure required to maintain urinary flow in the presence of increased outflow resistance occurs at the expense of normal bladder storage function. Obstruction induced changes in detrusor function, compounded by age- related changes in the bladder and nervous system function, lead to urinary frequency, urgency, and nocturia, the most bothersome BPH-related complaints.

Pathology[9]

Anatomic Features:

BPH first develops in the periurethral transition zone of the prostate. The transition zone consists of two separate glands immediately external to the preprostatic sphincter and the main urethral wall at the point of urethral angulation near the verumontanum.

All BPH nodules develop either in the transition zone or in the periurethral region. Although early transition zone nodules appear to occur either as the disease progresses and the number of small nodules increases, they can be found in almost any portion of the transition or periurethral zone. However, the transition zone also enlarges with age unrelated to the development of nodules.

One of the unique features of the human prostate is the presence of the prostatic capsule, which plays an important role in the development of LUTS, In the dog, the only other species known to develop naturally occurring BPH, symptoms of bladder outlet obstruction and urinary symptoms rarely develop because the canine prostate lacks a capsule.

Presumably the capsule transmits the “Pressure” of tissue expansion to the urethra and leads to an increase in urethral resistance. Thus, the clinical symptoms of BPH in man may be due not only to age-related increases intraprostatic size but also to the unique anatomic structure of the human gland. Clinical evidence of the importance of the capsule can be found in series that clearly document that incision of the prostatic capsule (transurethral incision of the prostate) results in a significant improvement in outlaw obstruction, despite the fact that the volume of the prostate remains the same.

Histologic Features:

BPH is a true hyperplastic process. Histologic studies document and increase in the cell number the majority of early periurethral nodules are purely stomal in character. It is unclear whether these early stromal nodules contain mainly fibroblast-like cells or whether differentiation toward a smooth muscle cell type is occurring. In contrast, the earliest transition zone nodules represent proliferation of glandular tissue that may be associated with an actual reduction in the relative amount of stroma.

These glandular nodules are apparently derived from newly formed small duct branches that bud off from existing ducts, leading to a totally new ductal system within the nodule. This type of new gland formation is quite rare outside embryonic development. This proliferative process leads to a tight packing of glands within a given area as well as an increase in the height of the lining epithelium.

During the first 20 years of BPH development, the disease may be predominantly characterized by an increased number of nodules, and the subsequent growth of each new nodule is generally slow. Then a second phase of evolution occurs in which there is a significant increase in the size of nodules. In the first phase, the glandular nodules tend to be larger than the stromal nodules. In the second phase, when the size of individual nodules is increasing, the size of glandular nodules clearly predominates.

The Bladder's Response to obstruction

Current evidence suggests that the bladder's response to obstruction is largely an adaptive one. However, it is also clear that many lower tract symptoms in men with BPH or prostate enlargement are related to obstruction – induced changes in bladder function rather than to outflow obstruction directly.

Obstruction-induced changes in the bladder are of two basic types. First, the changes that lead to detrusor instability or decreased compliance and second, the changes associated with decreased detrusor contractility, which are associated with further deterioration in the force of the unitary stream, hesitancy, intermittency, increased residual urine, and (in a minority of cases) detrusor failure. Acute urinary retention should not be viewed as inevitable result of this process. Many patients presenting with acute urinary retention have more than adequate detrusor function, with evidence of a precipitating event leading to the obstruction.

There is considerable evidence that the response of the detrusor smooth muscle cell to stress (increased load related to outlet obstruction) is not as adaptive as the response of skeletal muscle to stress. In the latter case, a relatively normal repertoire of contractile protein genes is upregulated and an increased number of normally organized contractile units assemble in the muscle cell. In the detrusor smooth muscle cell load induced hypertrophy

leads to change in myosin heavy chain isoform expression and to a signification alteration in the expression of a variety of thin filament-associated proteins.

In addition to obstruction – induced changes in the smooth muscle cell and ECM of the bladder, there is increasing evidence that obstruction may modulate neural – detrusor responses as well

EPIDEMIOLOGY AND NATURAL HISTORY

Definitions

The study of epidemiology is concerned with the distribution and determinants of diseases in humans. Form this evolve the components for descriptive epidemiology, description of disease incidence, mortality, and prevalence by person, place, and time, and analytic epidemiology, the search for determinants of disease risk that may sever to increase prospects for prevention.

There is no globally accepted epidemiologic definition of BPH, and thus prevalence and incidence rates must be viewed in the context of the definitions chosen by the investigator reporting the data.

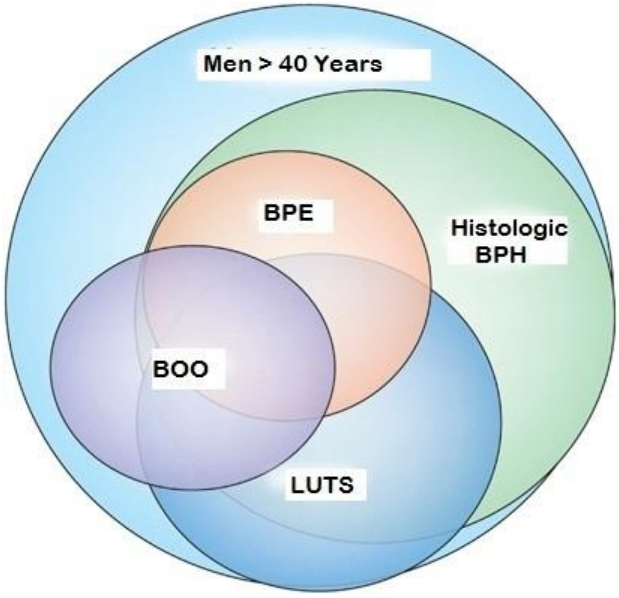
Cross-Sectional Studies of Clinical Prevalence:

Descriptive epidemiology relies on the presence of a single universally accepted definition of “disease.” The definitions of BPH, however, have undergone several changes in the past decade, and, at present, no single

criterion can beapplied. In the past, the term “prostatism” was used, incorrectly referring to the prostate as the sole source of the typical LUTS founds in aging men. It has been pointed out that there are at least three interrelated phenomena that can be assessed independently, namely the symptoms (formerly called prostatism), enlargement of the prostate gland, and presence of obstruction. In a given patient, all three, two of the three, or only one of the three entities might be present. Paul Abrams coined the term lower urinary tract symptoms to replace the old and inappropriate term prostatism. The same patients then can be further classified based on the degree of prostatic enlargement as measured by digital rectal examination (DRE), transrectal ultrasonography (TRUS), or magnetic resonance imaging (MRI) and lastly by the presence and degree of bladder outlet obstruction as measured by flow rate recordings or invasive pressure flow studies.

The diagram [9,11] shown below attempts to illustrate the difficulties in using different disease definitions. Of all men older than 40, a certain proportion develop histology hyperplasia of the prostate, that is BPH. Of those, some but not all develop LUTS, and other may have LUTS for reasons other than BPH**.** Prostate enlargement occurs in some but again not all men with histologic BPH and LUTS, and some men with enlarged glands may not have any symptoms at all. Lastly, urodynamically proven obstruction may be present in men who have either one several or all of histology BPH, LUTS,

and enlarged glands, yet other may have obstruction without having any evidence of BPH.



In addition to the mere enumeration of symptoms by frequency of occurrence, the bother associated with the symptoms, interference with activities of daily living, and the impact the symptoms have on quality of life are important distinguishing characteristics.

Accordingly, when studying the prevalence of clinical BPH admittedly an imprecise tem describing the constellation of LUTS, bother, interference, quality of life impact, with or without enlargement, obstruction, and so forth disease definitions may be applied that take either one or several of these items into consideration. For the subsequent discussion it is important to recognize that very few if any clear cut-off points have been established that allow differentiation between disease absent or present states (e.g., one might

argue that a prostate volume over 30ml constitutes clinical BPH, but others might argue for a higher or lower cut-off point; similar observations apply for symptoms and degrees of obstruction).

Symptom Severity and Frequency[9,17]

Form a pragmatic point of view, studies of symptom severity and frequency are of greatest importance in a disease that is rarely fatal and is characterized by its effects on the quality of life. The development, validation, and translation with cultural and Linguistic validation of the standardized, self-administered seven item American Urological Association (AUA) symptom index (also known as the International Prostate Symptom Score [IPSS]) has been a pivotal event in the clinical research on LUTS and BPH.

With the total score running from 0 to 35 points, patients scoring 0 to 7 points are classified as mildly symptomatic, those scoring from 8 to 19 points as moderately symptomatic, and those scoring 20 to 35 points as severely symptomatic. The instrument is an integral part of virtually every epidemiologic study as well as treatment studies in the field, and the availability of validated translations in many common languages allows cross-cultural comparisons of unprecedented scope.

Socioeconomic factors do not seem to influence responses to the questionnaire and fundamentally similar responses are obtained when the

questionnaire is self-administered, read to the patient mailed in, or administered in some other way.

In the past month:	Not at All	Less than 1 in5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
Incomplete Emptying: How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
Frequency: How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
Intermittency: How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency: How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
Weak Stream: How often have you had a weak urinary stream?	0	1	2	3	4	5	
Straining: How often have you had to strain to start urination?	0	1	2	3	4	5	
	Nil	1 **Time**	2 **Time**	3 **Time**	4 **Time**	5 **Time**	
Nocturia: How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total IPSS Score							

Score: 1-7:Mild 8-19:Moderate 20-35:Severe

Apart from these 7 questions regarding symptom severity International Prostate Symptom Score also contains an 8th question which is a Quality Of Life (QOL) question. This question is added as the International Scientific Committee (SCI), under the patronage of the World Health Organization (WHO) and the International Union Against Cancer (UICC), recommends the use of only a single question to assess the quality of life. The answers to this question range from

"delighted" to "terrible" or 0 to 6.

Quality of Life Due to Urinary Symptoms	Delighted	Pleased	MostlySatisfied	Mixed	Mostly Dissatisfied	Unhappy	Terrible
If you were to spend the rest of your life with your urinary condition just the way It is now, how would you feel about that?	1	2	3	4	5	6	

A very large international investigation of LUTS in Asian men was undertaken by Homma and colleagues (1997) in which 7588 men from Japan, China, Taiwan, Korea, the Philippines, Thailand, Singapore, Pakistan, India, and Australia were, queried. The finding of 18%, 29% , 40% and 56% of men in their 40s, 50s, 60s, and 70s having moderate to severe symptoms is line with the other studies reported both form Asia and from Europe and North America.

Prostate Size

Prostate size can be estimated by DRE, although the reliability across observers is in general considered poor. In addition, DRE tends to underestimate true prostate size as determined by TRUS or other imaging modalities. The magnitude of the underestimation increases with increasing prostate size for 25% up to 50% or more For the purpose of epidemiologic studies, TRUS and MRI measurements are preferred, although MRI measurement are somewhat expensive when attempting cross-sectional examinations of populations. TRUS volume measurements using the prostate ellipsoid volume formula are the most widely accepted measure of prostate volume with reasonable statistical performance characteristics, particularly when performed by a single or several well trained examiners.

Measures of obstruction

Subvesical obstruction can be measured only by invasive pressure-flow studies; non intubated free flow rates provide at best an indirect measure for the probability of obstruction being present. Unfortunately, no large-scale cross- sectional studies have been done employing pressure-flow tests because of the invasive and costly nature of the test, and it is unlikely that significant data sets will ever become available.

It is commonly accepted that a maximum flow rate of less than 10ml/sec indicates a high probability of obstruction and a flow rate of greater than 15ml/sec indicate a low probability, with 10 to 15 ml/sec presenting an intermediate range.

Post Void Residual Urine Volume[19,20]

One of the important subjects of tests for urinary incontinence is the post void residual urine volume (PVR), the amount of urine left after urination. Normally, about 50 mL or less of urine is left; more than 200 mL is a definite sign of abnormalities. Measurements inbetween require further tests. The most common method for measuring PVR is with a catheter, a soft tube, which is inserted into the urethra within a few minutes of urination. PVR can also be measured using transabdominal ultrasonography.

A study on the distribution of Post – void residual urine volume in randomly selected men published in the Journal Of Urology suggests little variation in the distribution of post-void residual urine volume across age groups or levels of urinary symptoms and peak urinary flow rate. However, a somewhat stronger relationship was found between residual urine and prostate volume.

Prostate-specific antigen (PSA) [8,12,18]

Prostate-specific antigen (PSA) is a protein produced by normal prostate cells. This enzyme participates in the dissolution of the seminal fluid coagulum and plays an important role in fertility. The highest amounts of PSA are found in the seminal fluid; some PSA escapes the prostate and can be found in the serum.

The level of PSA in the blood can be determined by a simple blood test. PSA blood test results are reported as nanograms per millilitre or ng/ml. Normal level usually range from 0 ng/ml to 4ng/ml.

Rising levels of PSA in serum are associated with prostate cancer. The PSA level also tends to rise in men with benign prostatic hyperplasia (BPH) and is a good marker for prostate volume. PSA levels are usually elevated in men with acute bacterial prostatitis. As BPH is a true hyperplasia, more cells produce a greater amount of PSA. It has been suggested that many PSA elevations detected and investigated in clinical practice may in fact be due to BPH leading to the argument that PSA is a better marker of BPH than of prostate cancer. One approach to distinguish the two conditions when PSA is elevated is to perform a free-to-total PSA ratio: more free PSA than complexed PSA suggests BPH rather than prostate cancer. A ratio of around 20% or greater for free PSA is considered more likely to represent BPH than cancer. PSA is discussed further in the investigations section below.

Correlations between Parameters

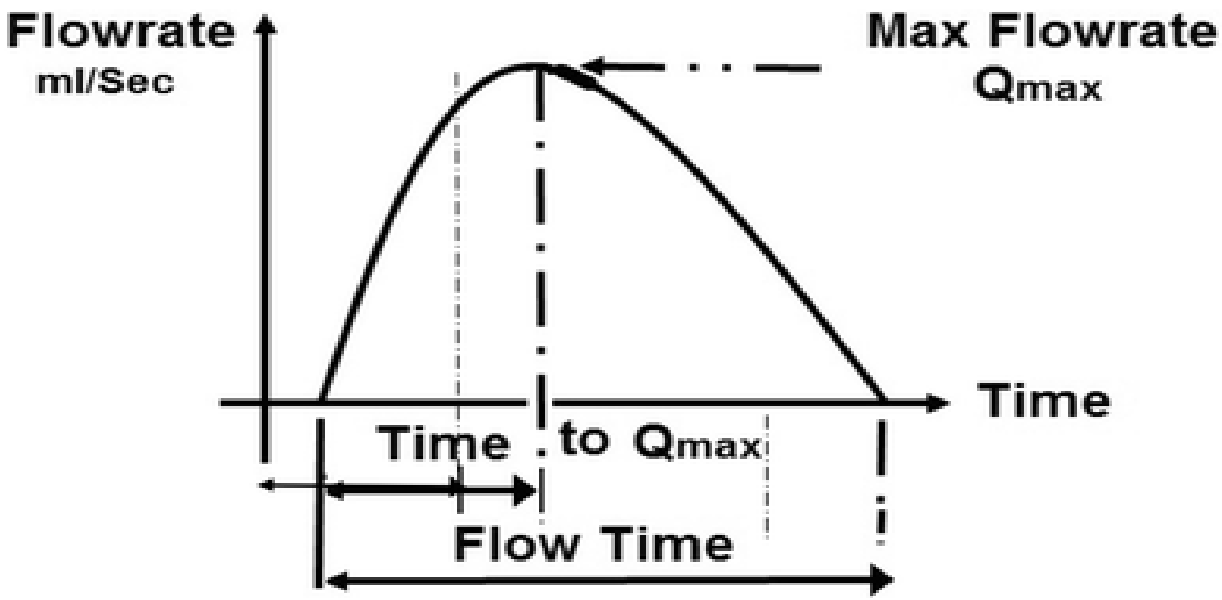
As noted, all relevant parameters such as symptom severity and frequency, bother, interference, disease-specific health-related quality of life (HROQOL),

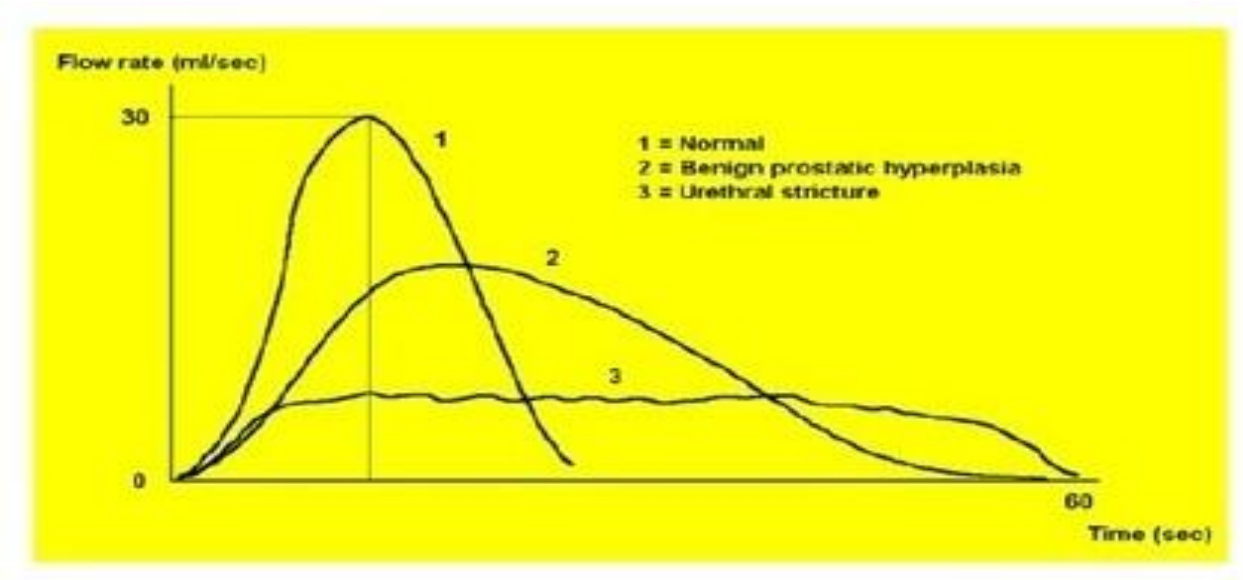
maximum flow rate, and prostate volume tend to worsen with advancing age. However, reported correlations between these parameters as well as urodynamic pressure-flow studies are in general weak with some exceptions. Strong correlations exist between measures of symptom severity and frequency (I-PSS score), bother, disease-specific HRQOL, and interference scores.

With the exception of age, correlations between various measures of LUTS and BPH are modest in community based population studies and weak in BPH clinic and trial populations, not precluding, however, a clinical meaningful relationship. The relationship between serum PSA and prostate volume is moderate and dependent on age and racial and ethnic origin. Neither symptoms nor flow rate nor prostate volume measures can predict presence and degree of obstruction reliably.

Uroflowmetry

Uroflowmetry involves the electronic recording of the urinary flow rate throughout the course of micturition. It is a common, noninvasive urodynamic test used in the diagnostic evaluation of the patients with symptoms of BOO. The results of uroflowmetry are nonspecific for causes of the symptoms. For example, an abnormally low flow rate may be caused by an obstruction (e.g., hyperplastic prostate, urethral stricture, meatal stenosis) or by detrusor hypocontractility.





Uroflowmetry is measurement of the rate of urine flow over time. It is also an assessment of bladder emptying. Multiple data points can be reported from non-invasive uroflowmetry. These include the following:

- Voided volume (VV in milliliters)
- Flow rate (Q in milliliters per second)
- Maximum flow rate (Qmax in milliliters per second)
- Average flow rate (Qave in milliliters per second)
- Voiding time (total time during micturition in seconds)
- Flow time (the time during which flow occurred in seconds)
- Time to maximum flow (onset of flow to Qmax in seconds)

The Agency for Healthcare Research and Quality guideline panel reached the following conclusions regarding uroflowmetry:

- Flow rate measurements are inaccurate if the voided volume is less than 125 to 150 mL.
- Flow rate recording is the single best noninvasive urodynamic test to detect lower urinary tract obstruction. Current evidence, however, is insufficient to recommend a given cutoff value to document the appropriateness of therapy.
- The peak flow rate (PFR; Qmax) more specifically identifies patients with BOO than does the average flow rate (Qave).
- Although Qmax decreases with advancing age and decreasing voided volume, no age or volume correction is currently recommended for clinical practice.
- Although considerable uncertainty exists, patients with a Qmax greater than 15 mL/sec appear to have poorer treatment outcomes after prostatectomy than patients with a Qmax of less than 15 mL/sec.
- A Qmax of less than 15 mL/sec does not differentiate between obstruction and bladder decompensation (or detrusor underactivity, which is present in 9% to 48% of men undergoing urodynamic evaluation for non-neurogenic LUTS)

In addition to these objective measurements, it is also important to observe the pattern or shape of the uroflow curve. A normal uroflow curve is bell-shaped.

Uroflow curve interpretation is somewhat subjective because of difficultly in qualitatively judging a pattern. When the flow rate is reduced or the pattern is altered, this could indicate bladder (underactivity) or bladder outlet (anatomic or functional obstruction) dysfunction.

Although certain patterns are suggestive of certain voiding dynamics (e.g., an interrupted or straining pattern with detrusor underactivity [DU] and a flattened pattern with a fixed obstruction), specific underlying abnormalities cannot be definitively identified without detrusor pressure data.

MATERIALS AND METHODS

50 male patients aged more than 50 years admitted with inguinal hernia to ALL the Surgical Units of Government Chengalpattu Medical College Hospital, Chengalpattu between April 2018 and April 2019 are selected as cases.

Inclusion criteria were,

1. Those with Inguinal Hernia,
2. Male sex,
3. Age more than 50years.

Exclusion criteria were,

1. Known case of BPH, who are already on drugs or have had any form of surgery for BPH in the past,
2. Presence of complications of hernia, such as irreducibility, strangulation or obstruction,
3. Female sex,
4. Age less than or equal to 50 years,
5. Known case of connective tissue disorders.

The method of selection was to select the first 50 male patients in the order date of their admission to the Government Chengalpattu Medical College Hospital without any other methods of randomization. The case

selection was independent of the side of the Hernia or whether the hernia is unilateral, bilateral or recurrent.

Every week, after selecting cases, the corresponding number of **CONTROL** subjects was selected randomly from the patients admitted to ALL the Surgical Units of Government Chengalpattu Medical College Hospital, Chengalpattu for conditions other than inguinal hernias so as to make a control group of 50 subjects.

The Inclusion criteria for the controls include,

1. Not seriously ill,
2. Male sex,
3. Age more than 50years.

The Exclusion criteria for the controls include,

1. Presence of inguinal hernia unilateral, bilateral or recurrent
2. Known case of BPH, who are already on drugs or have had any form of surgery for BPH in the past,
3. History of surgery done for inguinal hernia in the past
4. Female sex,
5. Age less than or equal to 50years,
6. Known case of connective tissue disorders,
7. Seriously ill or bedridden patient.

Informed written consent was obtained from each of the cases and controls. All subjects were interviewed and examined by the single observer.

Very few if any clear cut off points have been established that allow differentiation between disease absent and present states [9]. Hence for this study three independent variable - International Prostate Symptom Score, Prostate- specific antigen (PSA), prostate volume, post voidal residual urine, Uroflowmetry studies were taken and prevalence of BPH in cases and controls were found out for each of the three variables separately.

International Prostate Symptom Score:

International Prostate Symptom Score was obtained by reading out the questionnaire and answer options in the prescribed format as many of the study subjects were not able to read the questionnaire and mark the answers in the prescribed format. Each question was read out with its answer options and score for each question was marked separately and the sum is calculated to find out the International Prostate Symptom Score of each subject. The International Prostate Symptom Score (IPSS) is an 8 question (7 symptom questions + 1 quality of life question) written screening tool used to screen for, rapidly diagnose, track the symptoms of, and suggest management of the symptoms of the disease benign prostatic hyperplasia (BPH). The score of 7 symptom questions were added to get the International Prostate Symptom Score (IPSS).

IPSS result of 7 symptoms questions

Score	Correlation
0 – 7	Mildly symptomatic
8 – 19	Moderately symptomatic
20 – 35	Severely symptomatic

Subjects who are moderately or severely symptomatic i.e., those with International Prostate Symptom Score more than or equal to 8 were taken as having significant benign prostatic hyperplasia. Those who are mildly symptomatic i.e. those with International Prostate Symptom Score <8 were taken as having no significant benign prostatic hyperplasia.

PROSTATE SPECIFIC ANTIGEN[8].

Prostate-specific antigen (PSA) is an enzyme produced by the prostate gland. Normally, PSA is secreted in small amounts into the bloodstream. However, larger amounts of PSA are released when the prostate gland is enlarged, infected, or diseased. The level of PSA in the blood can be determined by a simple blood test. PSA blood test results are reported as nanograms per millilitre or ng/ml. Normal level usually range from 0 ng/ml to 4ng/ml.

The blood samples from the subjects were taken before doing Digital Rectal Examination. Subjects with Prostate-specific antigen values > 4ng/ml were taken as having significant benign prostatic hyperplasia in this study. Those subjects whose Prostate-specific antigen values less than or equal to 4ng/ml were taken as having no significant benign prostatic hyperplasia.

PROSTATE VOLUME:

The normal weight of the prostate in a young adult is from 17 to 19g[4]. Since 1cm³ of prostate tissue equals approximately 1g of prostate tissue, >20cc of prostate volume was taken as significant benign prostatic hyperplasia in this study[9]. Since Trans Rectal Ultra sonogram is not available in our hospital trans abdominal ultrasonogram was used to measure the Prostate size in this study. So subjects with prostate volume > 20cc are taken as having significant benign prostatic hyperplasia and those with prostate size less than or equal to 20cc are taken as having no significant benign prostatic hyperplasia.

UROFLOWMERTY STUDY:

Uroflowmetry is measurement of the rate of urine flow over time. It is also an assessment of bladder emptying. Multiple data points can be reported from noninvasive uroflowmetry.

These include Voided volume (VV in milliliters), Maximum flow rate (Qmax in milliliters per second), Average flow rate (Qave in milliliters per second), Voiding time (total time during micturition in seconds), Flow time (the time during which flow occurred in seconds), Time to maximum flow (onset of flow to Qmax in seconds).

Out of these parameters Maximum flow rate and average flow rate was taken into consideration for my study. Maximum flow rate < 15ml/ sec and Average flowrate < 8ml/ sec was considered to be associated with BPH. So patients were studied based on this data.

International Prostate Symptom Score (IPSS), Serum Prostate-specific antigen (PSA), and Prostate volume and Uroflowmetry study were found out for all cases and controls. The prevalence of benign prostatic hyperplasia in both cases and controls were done for each of the three variables studied here.

Univariate analysis of the association between inguinal hernia and benign prostatic hyperplasia was done for each of the five variables separately.

Chi Square test is used to find out the association and P values were calculated for each variable. A P value <0.05 is taken as statistically significant.

ANALYSIS AND DISCUSSION

The study was conducted over a period of one year from 1st April, 2018 to 30th April, 2019. A total no of 50 cases and 50 controls were selected. The cases were all males, aged between 52 years and 78 years with a mean age of 63.24 years.

The age distribution of cases was as follows.

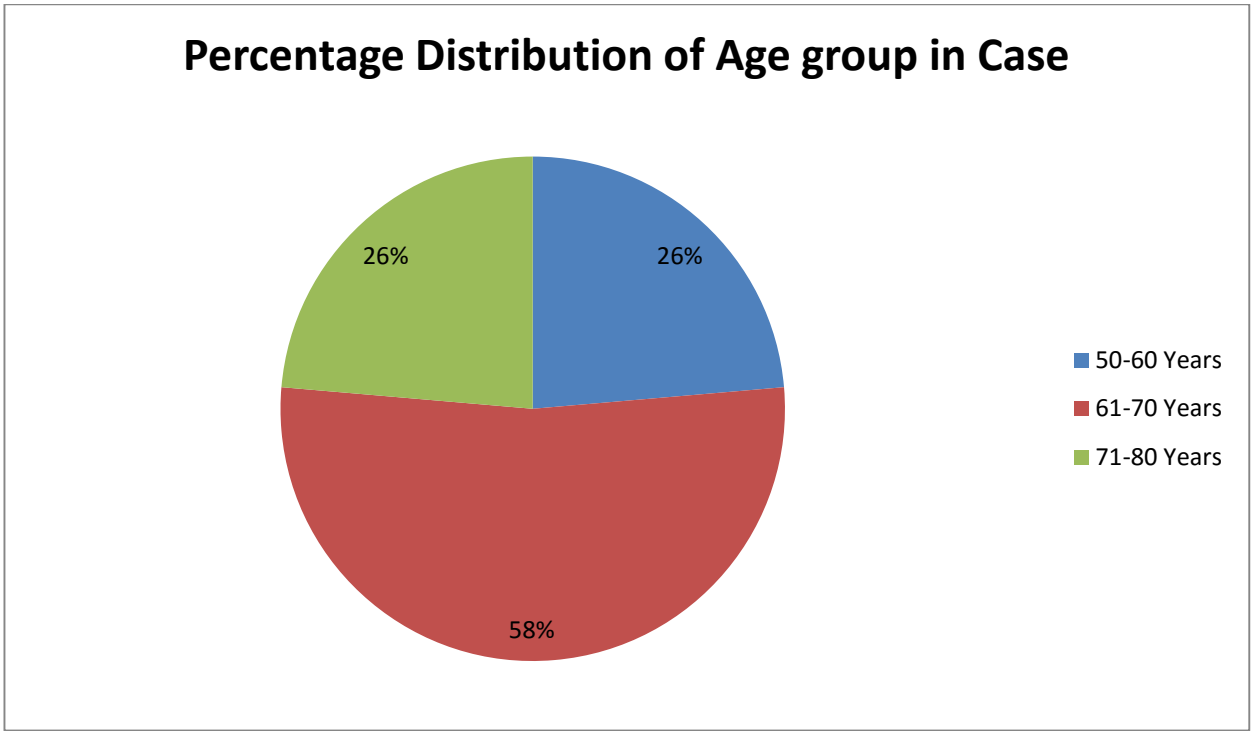
Age group	**51 – 60 years**	**61- 70 yrs**	**71 - 80 yrs**
Number of cases	13	29	8

The controls were all male subjects, aged between 52years and 80 years with a mean age of 63.66.

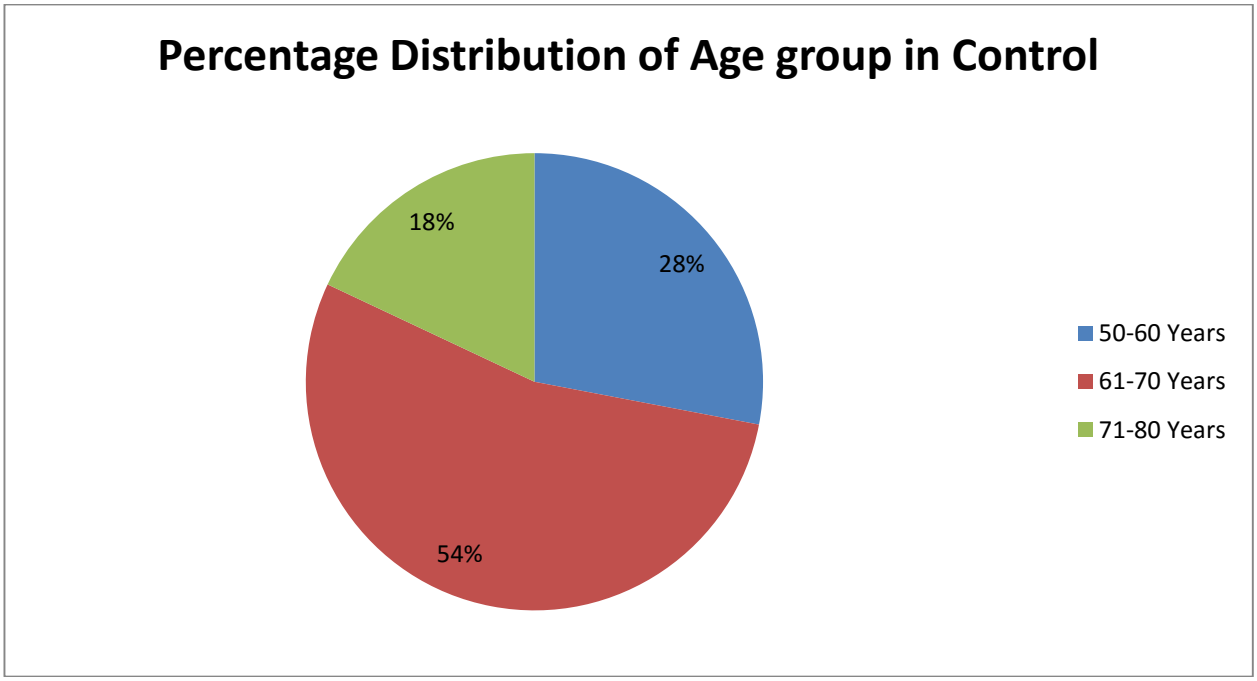
The age distribution of controls was as follows.

Age group	**51 – 60 years**	**61- 70 yrs**	**71 - 80 yrs**
Number of controls	14	27	9

THE AGE DISTRIBUTION OF CASES

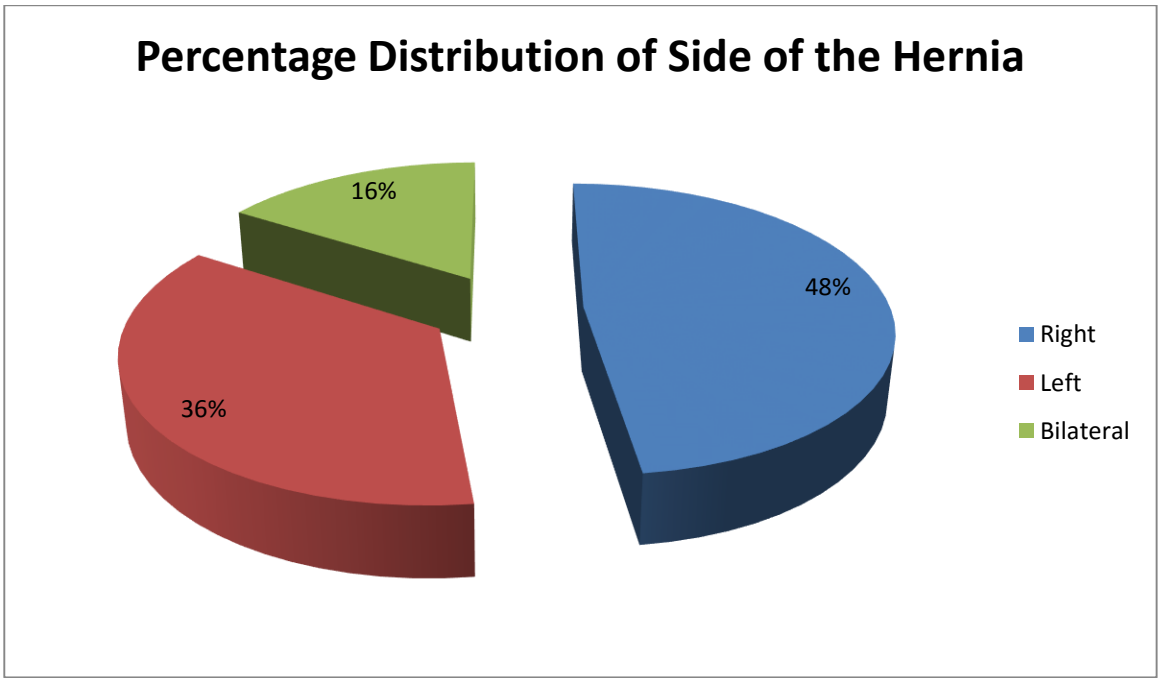


THE AGE DISTRIBUTION OF CONTROLS



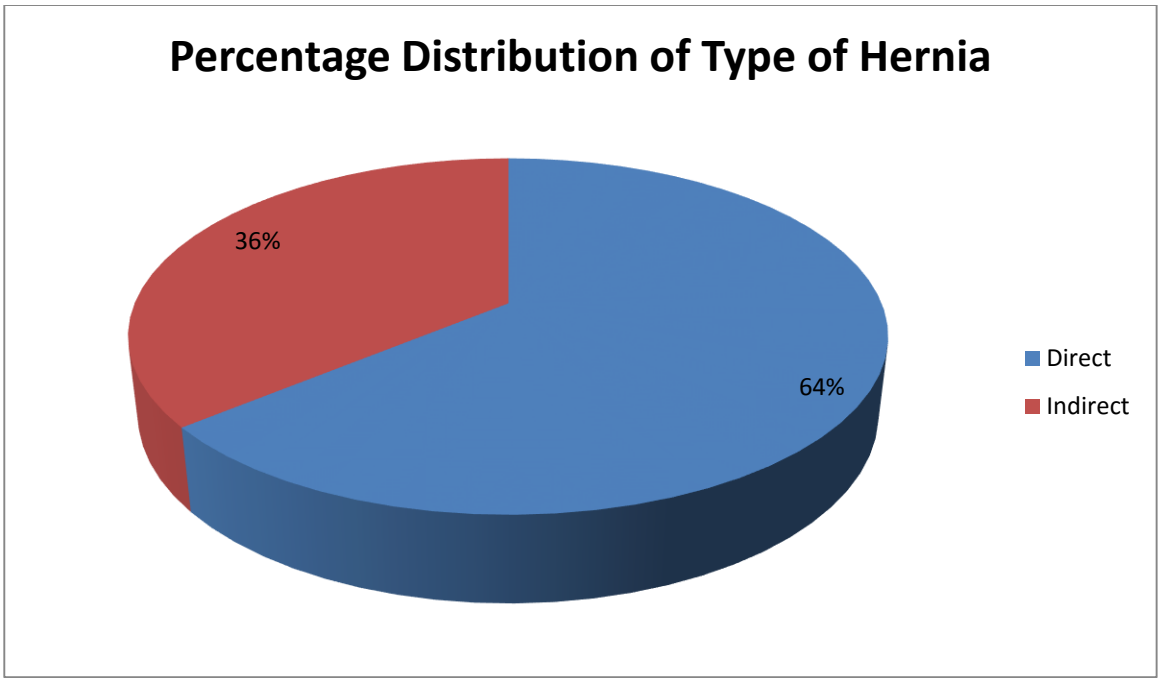
The side of the hernia among cases were as follows

Side	Right	Left	Bilateral
Number	24	18	8
Percentage	48%	36%	16%



Type of hernia – Direct or Indirect

Type of Hernia	Direct	Indirect
Number	32	18
Percentage	64.0%	36.0%

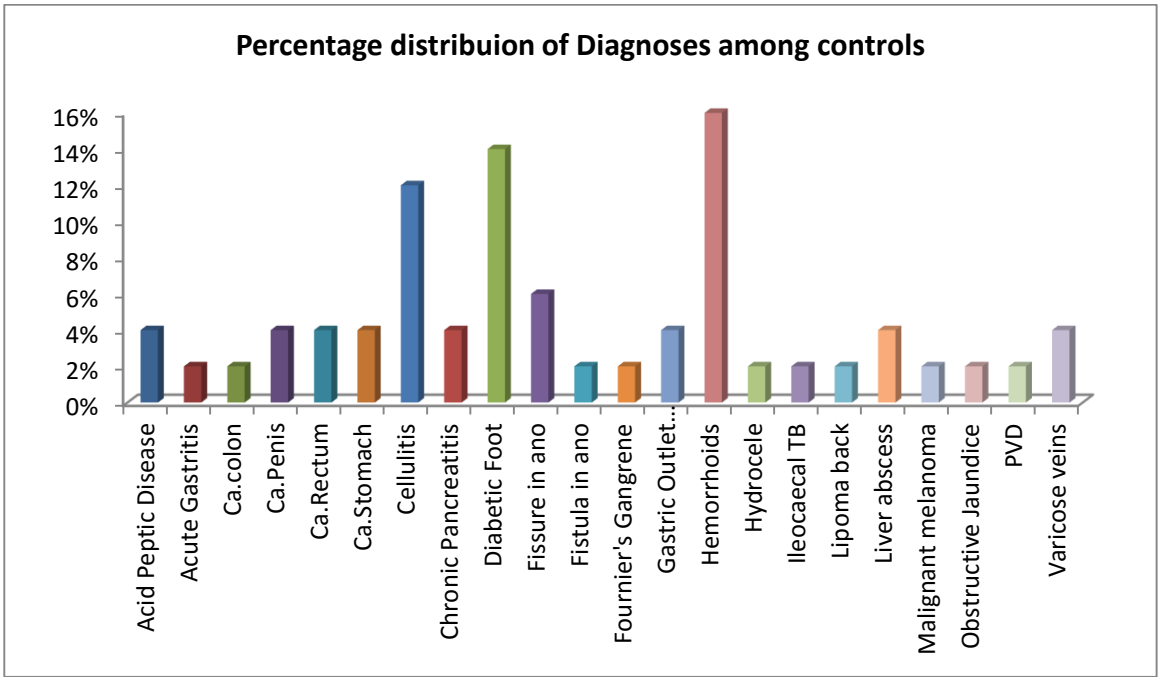


All the subjects were having reducible hernia only.

The control subjects were also selected from the ward from those patients admitted for diseases other than Hernia. The control subjects were having different illness such as Diabetic Foot, Haemorrhoids, Hydrocele, Acid Peptic Disease, Acute Gastritis, Carcinoma Rectum, Chronic Pancreatitis, Fissure in ano, Ileocaecal Tuberculosis, Gastric Outlet Obstruction, Cellulites, Varicose Veins, Liver Abscess, Carcinoma Stomach, Peripheral Vascular Disease, Fournier's Gangrene, Lipoma, Malignant Melanoma, Basal Cell Carcinoma, Carcinoma Colon, Fistula in Ano, Obstructive Jaundice and Carcinoma Penis.

Diagnoses among controls

Diagnosis	Frequency	Percent
Acid Peptic Disease	2	4.0
Acute Gastritis	1	2.0
Ca.colon	1	2.0
Ca.Penis	2	4.0
Ca.Rectum	2	4.0
Ca.Stomach	2	4.0
Cellulitis	6	12.0
Chronic Pancreatitis	2	4.0
Diabetic Foot	7	14.0
Fissure in ano	3	6.0
Fistula in ano	1	2.0
Fournier's Gangrene	1	2.0
Gastric Outlet Obstruction	2	4.0
Hemorrhoids	8	16.0
Hydrocele	1	2.0
Ileocaecal TB	1	2.0
Lipoma back	1	2.0
Liver abscess	2	4.0
Malignant melanoma	1	2.0
Obstructive Jaundice	1	2.0
PVD	1	2.0
Varicose veins	2	4.0
Total	50	100.0



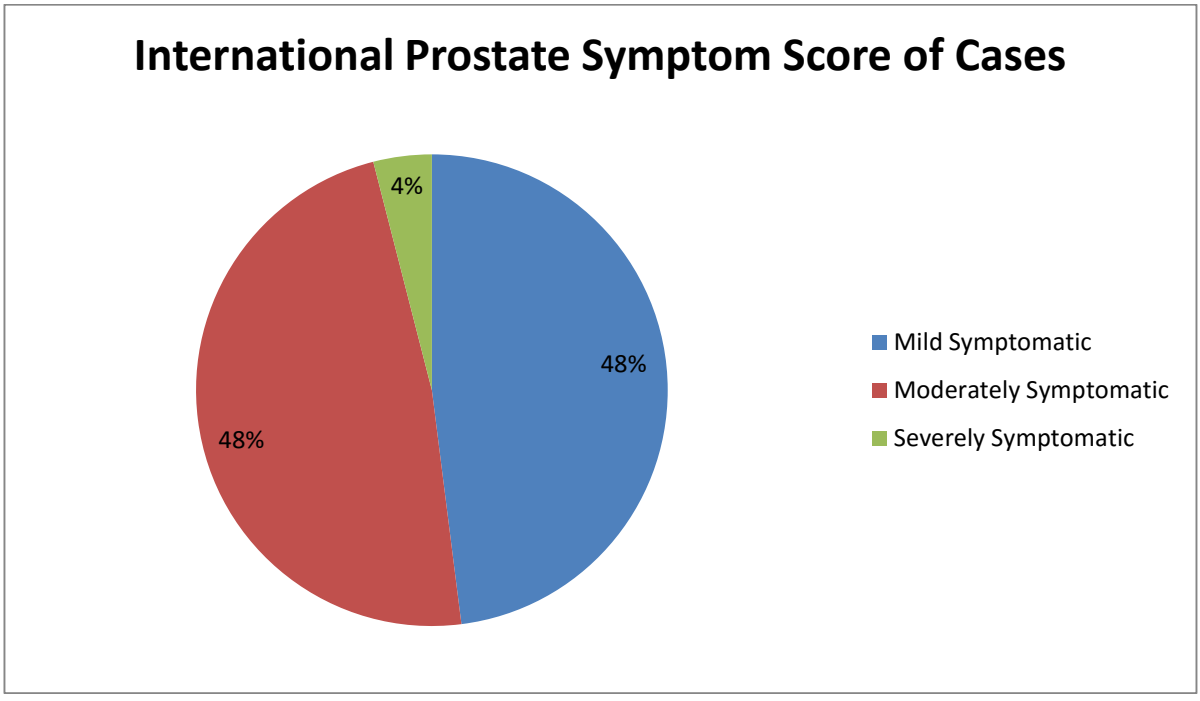
None of the control subjects had inguinal hernia or have had any kind of surgery for inguinal hernia in the past.

INTERNATIONAL PROSTATE SYMPTOM SCORE:

Among the cases none of the subjects were asymptomatic, 24 were mildly symptomatic, 24 were moderately symptomatic and 2 were severely symptomatic at the time of admission. Among controls, 33 were mildly symptomatic or having no symptoms, 15 were moderately symptomatic and 2 were severely symptomatic.

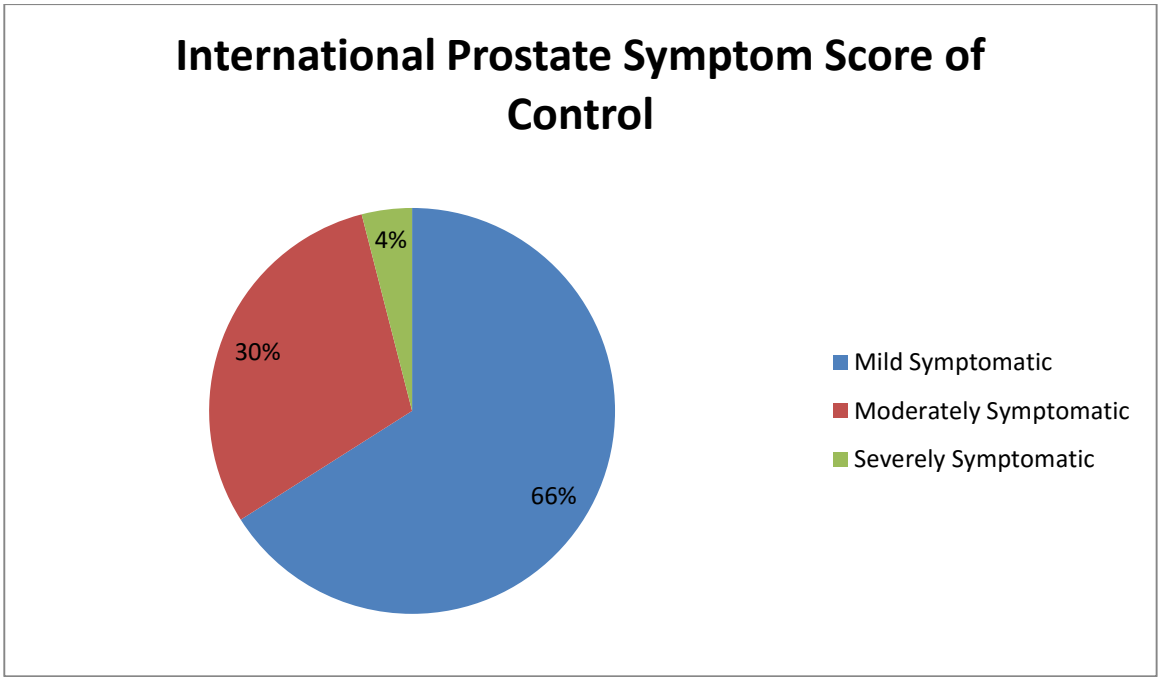
International Prostate Symptom Score of Cases:

IPSS_Score	Frequency	Percent
Mild	24	48.0
Moderately Symptomatic	24	48.0
Severely Symptomatic	2	4.0
Total	50	100.0



International Prostate Symptom Score of controls

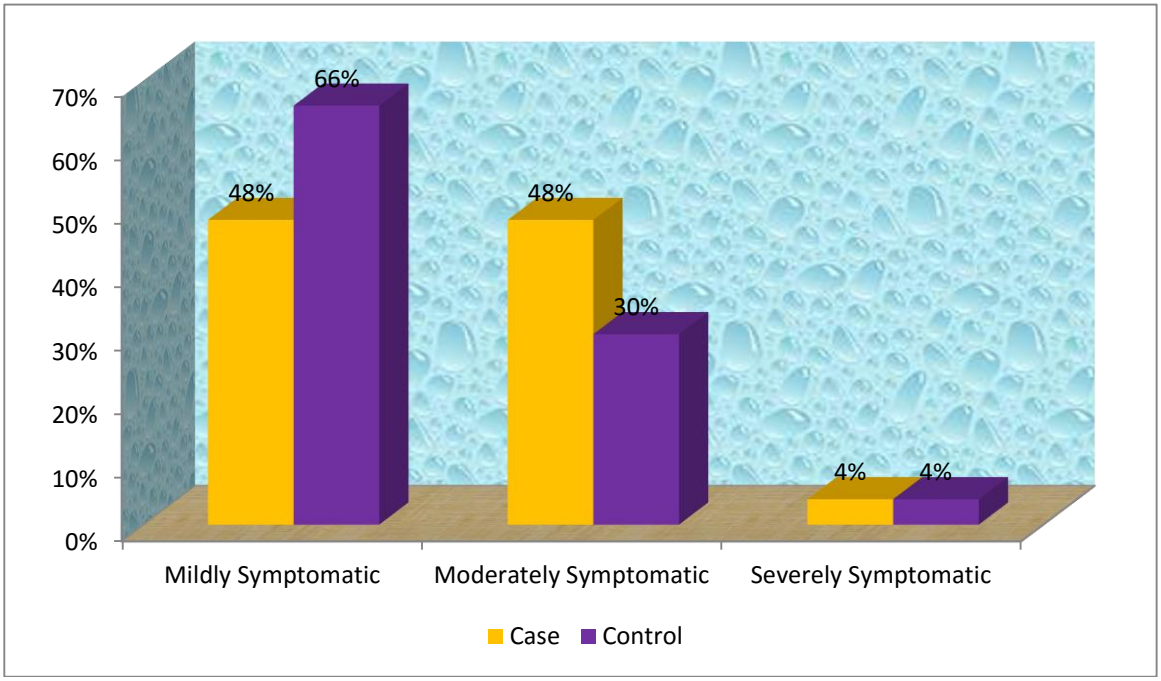
IPSS_Score	Frequency	Percent
Mild	33	66.0
Moderately Symptomatic	15	30.0
Severely Symptomatic	2	4.0
Total	50	100.0



All subjects who were moderately or severely symptomatic were considered as having significant BPH. Hence there were 26 subjects among cases and 17 subjects among controls were having significant BPH.

IPSS_Score * GROUP Crosstabulation					
			GROUP		Total
			Case	Control	
IPSS_Score	Mildly symptomatic	Count	24	33	57
		% within GROUP	48.0%	66.0%	57.0%
	Moderately Symptomatic	Count	24	15	39
		% within GROUP	48.0%	30.0%	39.0%
	Severely Symptomatic	Count	2	2	4
		% within GROUP	4.0%	4.0%	4.0%
Total		Count	50	50	100
		% within GROUP	100.0%	100.0%	100.0%

Chi square value= 3.48; P value 0.174



Among those scored ≥ 8 in IPSS scores 60.47 were cases and 39.53% were controls. However the difference is not statistically significant.

PROSTATE VOLUME:

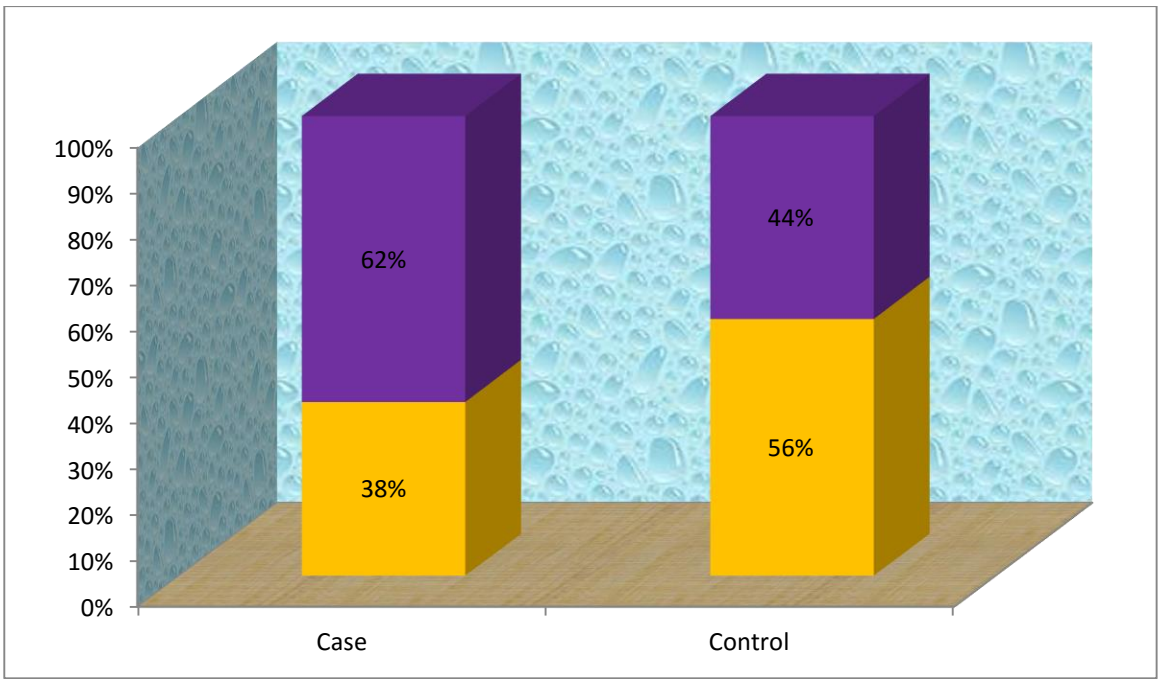
Among cases, 31 subjects were having prostate volume >20cc and 19 were having prostate volume less than equal to 20cc. Therefore 31 of 50 cases were taken as having significant Benign Prostatic Enlargement.

Among controls, 22 subjects were having prostate volume > 20cc and 28 were having prostate volume less than equal to 20cc. Therefore 22 of 50 controls were taken as having Benign Prostatic Enlargement.

Pr_volume_score * GROUP Cross tabulation

			GROUP		Total
			Case	Control	
Pr_volume_score	≤20	Count	19	28	47
		% within GROUP	38.0%	56.0%	47.0%
	>20	Count	31	22	53
		% within GROUP	62.0%	44.0%	53.0%
Total		Count	50	50	100
		% within GROUP	100.0%	100.0%	100.0%

Chi square value = 3.252 p value = 0.071



Among those who had a prostate vol >20 ng/ml, 58.5% were cases and 41.5 % were controls. However, the difference is not statistically significant.

Post - Void Residual Urine Volume

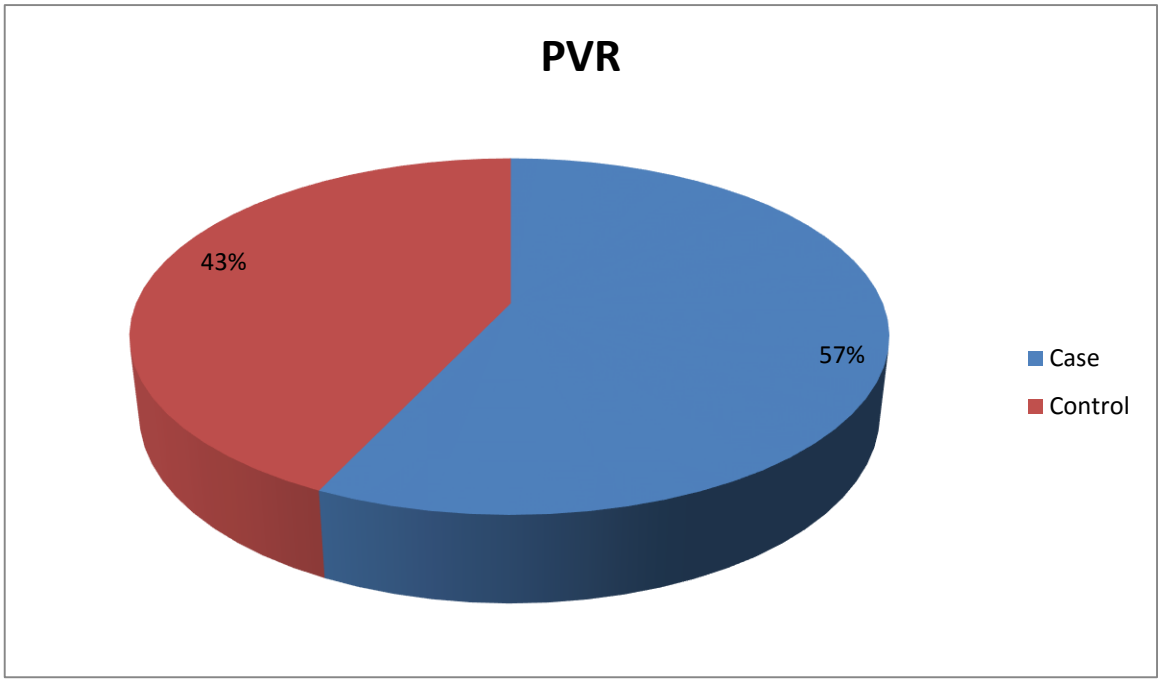
The measurement of Post – void residual urine volume is done by Ultra sonogram of abdomen. A volume of > 50ml is taken as significant. But there was no significant difference in the distribution of the number of patients with Post – void residual urine volume >50ml between cases and controls (17 and 13 respectively).

Distribution of Post - Void Residual Urine Volume among Cases and Controls:

PVR * GROUP Cross tabulation

			GROUP		Total
			Case	Control	
PVR	No	Count	33	37	70
		% within PVR	47.1%	52.9%	100.0%
	Yes	Count	17	13	30
		% within PVR	56.7%	43.3%	100.0%
Total		Count	50	50	100
		% within PVR	50.0%	50.0%	100.0%

Chi square value = 0.762; p value =0.383



Among those who had a PVRU > 50 ml, 56.7% were cases and 43.3 % were controls. However, the difference is not statistically significant.

PROSTATE SPECIFIC ANTIGEN:

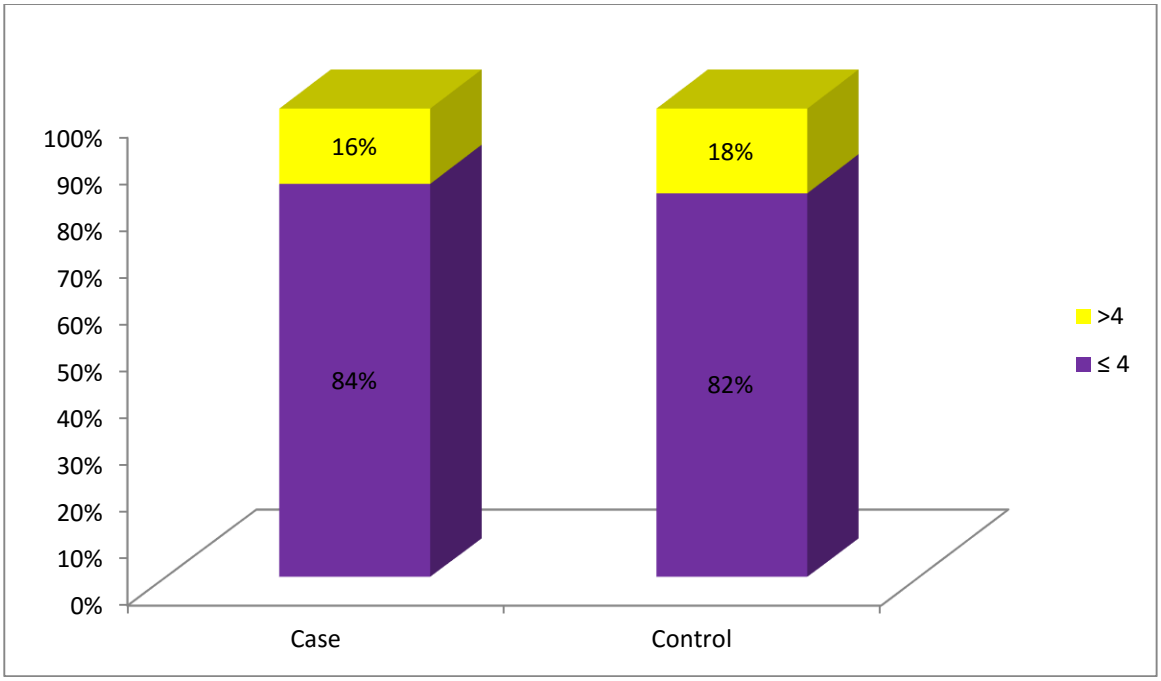
Among the cases 8 subjects were having PSA > 4ng/ml and 42 were having PSA less than or equal to 4ng/ml. Therefore 8 out of 50 cases were taken as having significant Benign Prostatic Enlargement.

Among the controls 9 subjects were having PSA > 4ng/ml and 41 were having PSA less than or equal to 4ng/ml. Therefore 9 out of 50 controls were taken as having significant Benign Prostatic Enlargement.

PSA_score * GROUP Cross tabulation

			GROUP		Total
			Case	Control	
PSA_score	≤ 4	Count	42	41	83
		% within GROUP	84.0%	82.0%	83.0%
	>4	Count	8	9	17
		% within GROUP	16.0%	18.0%	17.0%
Total		Count	50	50	100
		% within GROUP	100.0%	100.0%	100.0%

Pearson Chi-Square = 0.071; p value = 0.790

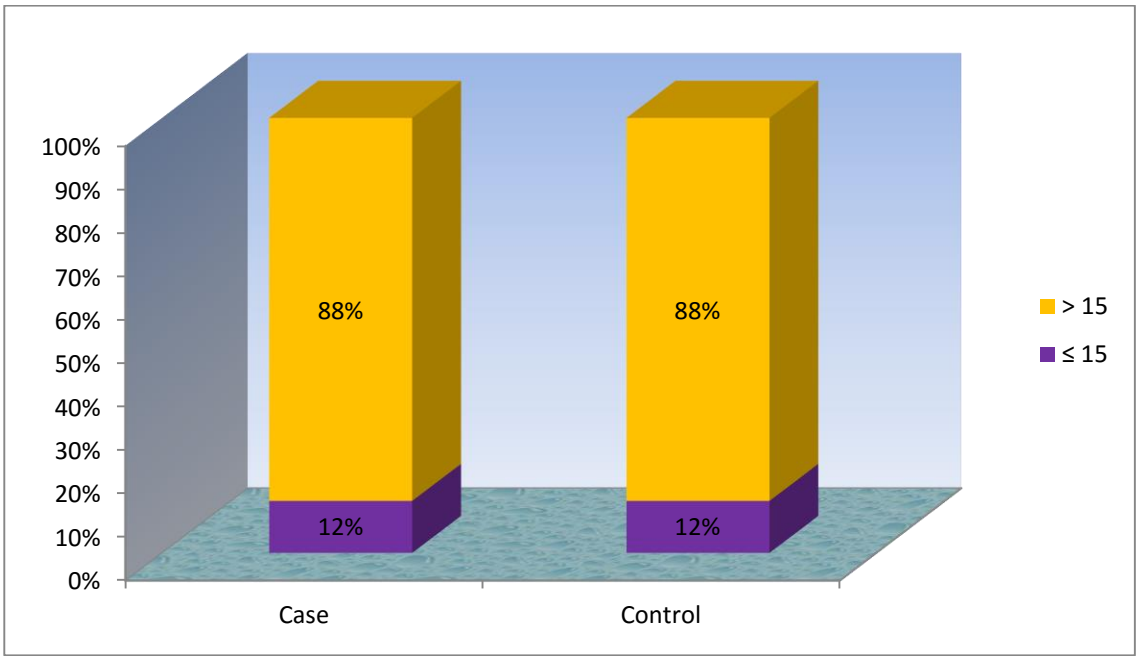


Among those who had PSA value > 4ng/ml, 47.5% were cases and 52.5% were controls. However, the difference is not statistically significant.

UROFLOWMETRY STUDY

QMax (Maximum Flow rate) Crosstab

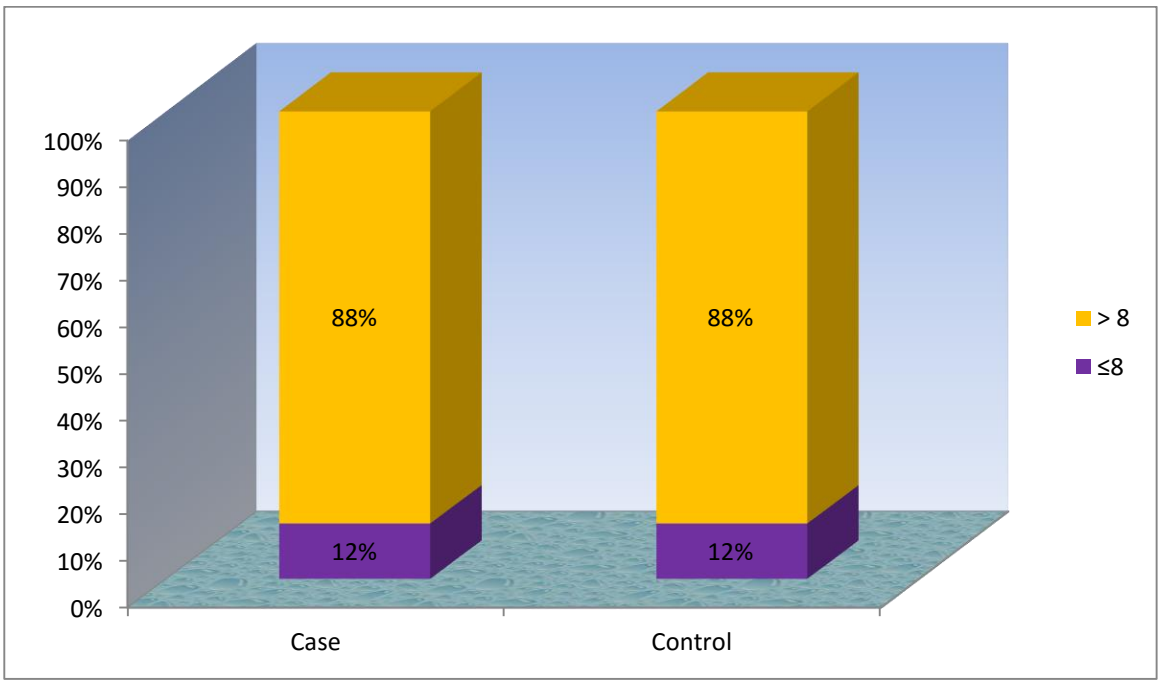
			GROUP		Total
			Case	Control	
Qmax_Score	<15	Count	6	6	12
		% within GROUP	12.0%	12.0%	12.0%
	>15	Count	44	44	88
		% within GROUP	88.0%	88.0%	88.0%
Total		Count	50	50	100
		% within GROUP	100.0%	100.0%	100.0%



Chi square value = 0; p value =1

Average Flowrate Crosstab

			GROUP		Total
			Case	Control	
afr_score	<8	Count	6	6	12
		% within GROUP	12.0%	12.0%	12.0%
	>8	Count	44	44	88
		% within GROUP	88.0%	88.0%	88.0%
Total		Count	50	50	100
		% within GROUP	100.0%	100.0%	100.0%



Chi square value = 0; p value = 1

Among those who had Qmax < 15 ml/sec , 50% were cases and 50% were controls. Among those who had APR < 8 ml/sec , 50% were cases and 50% were controls. However, the difference is not statistically significant.

CONCLUSION

- Among the cases 84 were having unilateral hernia and 16 were having bilateral hernias.
- Right sided hernia was slightly more common than the left sided hernias (48vs 36).
- Univariate analysis of association between Inguinal Hernia and Prostate Gland Enlargement (Benign Prostatic Hyperplasia) using International Prostate Symptom Score showed no statistically significant association between the two.
- Univariate analysis of association between Inguinal Hernia and Prostate Gland Enlargement (Benign Prostatic Hyperplasia) using Serum Prostatic Specific Antigen showed no statistically significant association between the two.
- Prostatic size also showed no statistically significant association between Inguinal Hernia and Prostate Gland Enlargement (Benign Prostatic Hyperplasia).
- There was no significant difference in the distribution of the number of patients with Post – void residual urine volume >50ml between cases and controls.

- Univariate analysis of association between Inguinal Hernia and Prostate Gland Enlargement (Benign Prostatic Hyperplasia) using Uroflowmetry Study also showed no statistically significant association between the two.

- Although both Inguinal Hernia and Benign Prostatic Hyperplasia are seen with increased frequency in the aged male population, this study showed no statistically significant association between the two. Their occurrence together can be considered a chance co-existence rather than cause and effect.

LIMITATIONS OF THE STUDY

- Age standardization could not be done in this study because of the method of selection of cases and controls.
- Trans Rectal Ultrasonographic assessment of prostatic size could not be done in this study as it was not available in our hospital.

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PROFORMA – CASES

Basic Details

Name: Age/Sex

IPNO: DOA:

Hernia

Right O Left O Bilateral O Recurrent O

Direct O Indirect O

IPSS:

In the past month:	Not at All	Less than 1 in5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
Incomplete Emptying: How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
Frequency: How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
Intermittency: How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency: How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
Weak Stream: How often have you had a weak urinary stream?	0	1	2	3	4	5	
Straining: How often have you had to strain to start urination?	0	1	2	3	4	5	

	Nil	1 **Time**	2 **Time**	3 **Time**	4 **Time**	5 **Time**	
Nocturia: How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total IPSS Score							

Prostate Volume :

Post – void Residual Urine Volume :

PSA :

Uroflowmerty Study					
V.V (ml)	QMax (ml/sec)	AFR (ml/sec)	VT (sec)	FT (sec)	TMF (sec)

PROFORMA CONTROLS

Basic Details

Name: Age/Sex

IPNO: DOA:

Diagnosis:

IPSS:

In the past month:	Not at All	Less than 1 in 5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
Incomplete Emptying: How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
Frequency: How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
Intermittency: How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency: How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
Weak Stream: How often have you had a weak rinary stream?	0	1	2	3	4	5	
Straining: How often have you had to strain to start urination?	0	1	2	3	4	5	

	Nil	1 Time	2 Time	3 Time	4 Time	5 Time	
Nocturia: How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total IPSS Score							

Prostate Volume :

Post – void Residual Urine Volume :

PSA :

Uroflowmerty Study					
V.V (ml)	QMax (ml/sec)	AFR (ml/sec)	VT (sec)	FT (sec)	TMF (sec)

KEY TO THE MASTER CHART

A	:	Age
IPNo	:	Inpatient number
Rt	:	Right sided
Lt	:	Left sided
B/L	:	Bilateral
Rc.	:	Recurrent Hernia
D	:	Direct Hernia
ID	:	Indirect Hernia
Ic. Em	:	Incomplete Emptying
Fr.	:	Frequency of Urination
In.Em	:	Intermittent Emptying
Urg.	:	Urgency
Wk.St.	:	Weak Stream
Str.	:	Straining
Noct.	:	Nocturia
IPSS	:	International Prostate Symptom Total Score
Pr.V	:	Prostate volume in cc
PVR	:	Post – void residual urine volume >50ml
PSA	:	Prostate Specific Antigen
n/ml	:	nanograms/litre

V.V	:	Voided volume in ml
QMax	:	Maximum flowrate in ml/sec
AFR	:	Average Flow ratr in ml/sec
VT	:	Voiding time in sec
FT	:	Flow time in sec
TMF	:	Time to Maximum flow in sec

MASTER CHART CASES

S.No.	Name	A	IP NO	Hernia						International Prostate Symptom Score								Pr. V	PVR	PSA	Uroflowmetry Study					
				Rt	Lt	B/L	Rc.	D	ID	Ic.Em	Fr.	In.Em	Urg.	Wk.St.	Str.	Noct.	IPSS				V.V	Q_{Max}	AFR	VT	FT	TMF
1	Kannan	67	37278	*					*	0	1	0	1	1	1	0	4	19		0.5	250	23	12	21	20	10
2	Santhanam	73	39268		*			*		0	0	0	1	1	1	1	4	24		1.8	300	27	14	23	22	11
3	Gandhi	71	40526	*					*	1	3	1	4	2	3	2	16	30	*	5.1	200	15	8	27	26	13
4	Kuppusamy	54	40616		*				*	0	2	0	0	1	0	0	3	18		0.9	300	26	14	22	21	11
5	Balan	62	41585		*			*		2	2	0	2	2	2	1	11	33	*	5.8	220	14	8	26	26	13
6	Baskar	68	41868	*					*	0	1	0	1	1	0	0	3	17		3.2	280	30	16	20	18	10
7	Alex	74	42432		*			*		3	4	2	4	5	4	3	25	33	*	4.6	200	20	11	20	19	10
8	Jamal Khan	66	44616		*			*		0	0	0	1	1	1	1	4	16		1.3	330	28	15	23	22	12
9	Saravanan	56	45809	*					*	1	1	1	3	2	3	1	12	25		4.0	250	25	13	20	19	10
10	Murugan	61	46302	*				*		0	1	0	1	1	0	0	3	19		3.2	270	24	12	24	23	12
11	Amaran	62	47305	*					*	0	1	1	0	1	0	0	3	23		0.8	280	26	13	23	22	12
12	Manickam	53	48509			*			*	0	1	0	0	1	0	0	2	24		1.9	250	19	10	26	25	13
13	Peralan	75	49101	*				*		1	1	1	2	1	1	1	8	15		0.6	300	27	14	22	21	11
14	Jahir Hussain	53	50201	*				*		0	1	0	0	0	1	0	2	16		0.9	320	28	14	24	23	12
15	Palani	70	50415		*			*		1	2	2	3	2	5	2	17	30	*	4.3	270	19	10	28	26	14
16	Thangapandi	55	51121	*					*	0	1	0	1	0	0	0	2	18		2.2	250	25	12	22	21	11
17	Suresh	61	52548		*			*		2	2	1	3	4	4	2	18	35	*	6.2	210	13	7	30	28	15
18	Santhosh	54	52988	*				*		1	2	1	1	2	2	0	9	25		2.0	220	22	11	21	20	10
19	Ganesan	59	53144			*		*		0	1	0	1	1	1	0	4	20		1.1	260	21	11	24	23	12
20	Chinnakannan	64	54137		*			*		0	1	2	3	3	2	1	12	30		2.8	300	29	15	21	20	10
21	Gunasekar	65	55782	*				*		0	0	1	1	1	2	0	5	18	*	1.5	280	25	13	23	22	12
22	Jeyakaran	72	56123			*			*	2	2	2	4	2	4	3	19	40	*	6.2	220	13	7	32	30	16
23	Sivaraman	76	57179		*			*		1	2	1	2	1	2	0	9	20		2.2	250	21	11	23	22	12
24	Wasif Khan	64	58583		*			*		0	1	1	3	3	3	3	14	30	*	1.5	300	26	14	22	21	11
25	Kamarajan	57	59035	*					*	0	1	1	1	0	0	0	3	23		3.3	300	30	16	20	19	10

S.No.	Name	A	IP NO	Hernia						International Prostate Symptom Score								Pr. V	PVR	PSA	Uroflowmetry Study					
				Rt	Lt	B/L	Rc.	D	ID	Ic.Em	Fr.	In.Em	Urg.	Wk.St.	Str.	Noct.	IPSS				V.V	Q_{MAX}	AFR	VT	FT	T Q_{MAX}
26	Thandapani	56	60884			*		*		0	1	1	0	0	1	0	3	18		1.2	250	25	13	20	19	10
27	Madhan	61	60902	*				*		0	1	1	0	1	1	0	4	18		2.9	270	28	14	20	19	10
28	Balasamy	63	61784		*				*	2	2	0	4	3	4	2	17	25		2.1	300	25	13	23	22	12
29	Palraj	61	63267			*		*		0	1	1	1	1	1	0	5	15		3.2	220	23	12	20	19	10
30	Rajan	62	64555	*				*		0	2	2	0	2	1	1	8	30	*	3.1	240	20	10	25	24	12
31	Sethu	52	68121	*				*		0	1	1	0	1	0	0	3	16		0.4	320	24	13	25	24	13
32	Krishnakanth	59	1114	*				*		1	1	2	3	3	3	1	14	36	*	4.0	250	17	9	29	28	15
33	Senthil	64	1149		*				*	1	2	1	2	2	3	1	12	33		3.9	290	26	14	21	20	10
34	Balamurugan	62	1259		*				*	0	1	0	1	2	0	0	4	18		1.1	300	31	16	20	19	10
35	Ayyakannu	65	4462	*				*		2	3	1	3	3	3	3	18	30	*	2.2	260	23	11	25	24	13
36	Shanmugham	62	4833			*		*		0	1	0	0	1	1	0	3	20		1.1	270	26	13	22	21	11
37	Anand	61	5562	*				*		2	2	1	3	2	4	2	16	40	*	3.8	200	20	9	24	23	12
38	Udhayan	61	8254	*				*		0	1	1	0	1	1	1	5	20		1.2	280	23	12	25	24	12
39	Ramesh	71	8834		*				*	1	1	1	2	2	2	2	11	30	*	4.2	300	19	10	32	31	16
40	Ambalam	64	9920		*				*	0	1	1	1	1	1	1	6	17		1.3	240	20	10	24	23	12
41	Karuppan	70	10212	*				*		0	1	0	1	0	0	1	3	16		0.8	300	21	11	28	27	14
42	Rajamani	58	11583	*				*		0	1	1	1	1	1	1	6	15		0.8	340	28	15	25	23	13
43	Raja	61	13898		*			*		0	1	1	2	2	2	2	10	36	*	3.2	280	19	10	30	29	15
44	Velmurugan	67	15896		*				*	0	1	1	2	2	2	0	8	21		2.3	260	14	8	32	31	16
45	Rangan	61	18329	*					*	1	2	2	2	3	3	2	15	38	*	2.2	200	15	8	25	24	12
46	Singaraj	68	23481			*		*		0	1	1	3	3	3	3	15	30	*	1.6	240	18	9	27	26	14
47	Suryan	61	25167			*		*		1	1	1	2	2	1	0	8	18		1.6	300	26	14	22	21	11
48	Arulmuthu	70	28353		*			*		1	1	1	1	2	1	1	8	20		1.5	320	33	16	21	20	11
49	Durai	78	30101	*				*		3	4	4	4	5	4	4	28	36	*	5.1	240	21	10	25	24	12
50	Shankarlingam	52	31203	*					*	1	0	1	1	1	0	0	4	15		1.0	300	25	13	24	23	12

MASTER CHART CONTROLS

S.no	Name	A	IP NO	DIAGNOSIS	International Prostate Symptom Score								Pr.V	PVR	PSA	Uroflowmetry Study					
					Ic.Em	Fr.	In.Em	Urg.	Wk.St.	Str.	Noct.	IPSS				V.V	Q_{MAX}	AFR	VT	FT	T Q_{MAX}
1	Sundharam	68	37286	Diabetic Foot	2	2	1	2	3	3	1	14	33	*	4.1	230	18	9	26	25	13
2	Parthiban	62	37392	Hemorrhoids	0	0	0	1	1	0	1	3	18		2.2	250	25	13	20	19	10
3	Jaganathan	55	40112	Hydrocele	0	0	0	1	0	1	0	2	16		1.8	300	29	15	21	20	10
4	Mani	67	41510	Ca.Rectum	1	0	1	2	0	0	0	4	16		1.9	330	30	16	23	21	11
5	Shankar	71	41624	Chronic Pancreatitis	1	3	1	3	4	4	2	18	40	*	3.4	220	18	9	25	24	12
6	Palpandi	63	44195	Acute Gastritis	0	0	0	0	0	0	0	0	16		1.2	280	24	12	24	23	12
7	Senthil	52	45211	Gastric Outlet Obstruction	0	1	0	1	0	0	0	2	18		2.1	250	20	10	26	24	13
8	Anakutty	75	47322	Diabetic Foot	0	2	2	3	2	2	2	13	30	*	4.6	220	14	8	30	28	15
9	Murugan	58	48110	Varicose veins	0	1	1	1	0	1	0	4	16		1.8	300	28	14	22	21	11
10	Muniyandi	64	49197	Liver abscess	0	1	0	1	1	0	0	3	18		2.1	290	26	13	23	22	12
11	Ibrahim	67	50003	Chronic Pancreatitis	0	1	1	1	1	0	0	4	18		1.9	300	27	14	22	21	11
12	Pasupathy	80	51219	Acid Peptic Disease	1	1	1	1	2	2	2	10	26		2.8	260	22	11	24	23	12
13	Syed Sultan	52	50993	Hemorrhoids	2	2	3	3	3	3	4	20	35	*	6.1	200	13	7	29	28	14
14	Bala	62	52218	Ca.Stomach	0	0	0	0	0	0	0	0	16		1.3	250	21	10	25	24	12
15	Nayakam	59	54784	Malignant melanoma	0	1	1	1	0	1	0	4	20		3.2	280	23	12	25	23	13
16	Periyakomban	65	55763	Fissure in ano	1	2	1	3	3	3	3	16	30	*	4.6	220	15	8	28	27	14
17	Ayyankalai	73	55901	Ca.colon	1	1	1	1	1	1	0	6	25		3.5	280	24	12	25	24	12
18	Natchiappan	63	56402	Hemorrhoids	0	0	0	0	0	0	0	0	15		1.2	260	25	12	22	21	11
19	Chinna	65	57107	Ca. Rectum	1	1	1	1	2	2	2	10	18		3.8	230	20	10	25	23	13
20	Kannan	57	59525	Cellulitis	0	0	0	0	0	0	0	0	16		1.2	280	26	13	22	21	11
21	Sakkarai	60	60257	Diabetic Foot	1	1	1	0	0	0	1	4	17		1.3	330	28	14	25	24	14
22	Asker	72	61399	Lipoma back	1	1	2	1	3	2	4	14	20	*	4.8	200	14	7	28	27	14
23	Tamil	62	62865	Fissure in ano	0	0	0	0	0	0	1	1	22		2.7	280	25	12	24	23	13
24	Madurai	61	63717	Hemorrhoids	0	2	1	2	2	2	1	10	22	*	5.1	230	18	9	27	26	14
25	Banthalu	64	64122	Ileocaecal TB	0	0	0	0	0	0	0	0	15		1.1	300	27	14	22	21	11

S.no	Name	A	IP NO	DIAGNOSIS	International Prostate Symptom Score								Pr.V	PVRU	PSA	Uroflowmetry Study					
					Ic.Em	Fr.	In.Em	Urg.	Wk.St.	Str.	Noct.	IPSS				V.V	Q_{MAX}	AFR	VT	FT	T Q_{MAX}
26	Pasha	58	66286	Cellulitis	2	2	2	2	3	3	1	15	32	*	3.1	250	18	9	28	27	14
27	Mayilvahanan	61	66392	Obstructive Jaundice	0	0	1	1	1	0	1	4	18		1.4	300	27	14	22	21	11
28	Pitchai	57	67112	Ca.Penis	0	0	1	1	0	1	0	3	15		2.8	280	24	12	24	23	12
29	Muniyandi	64	68510	Hemorrhoids	1	2	1	2	0	0	0	6	18		1.5	300	30	16	20	19	10
30	Annadurai	70	1624	Diabetic Foot	1	2	1	2	2	2	2	12	30	*	4.4	250	18	9	29	27	14
31	Manickam	66	1995	Varicose veins	0	1	0	0	1	0	0	2	15		1.3	220	17	9	25	24	12
32	Kathiravan	56	2211	Fissure in ano	0	1	1	1	0	0	0	3	18		1.1	280	22	11	26	25	13
33	Ramachandran	72	5322	Gastric Outlet Obstruction	0	2	2	2	2	2	2	12	20		2.6	300	27	14	22	21	11
34	Natarajan	55	7110	Cellulitis	0	1	1	1	2	1	0	6	15		1.6	320	24	12	28	27	14
35	Shanmugam	62	9197	Hemorrhoids	0	1	0	1	1	0	0	3	16		1.1	300	29	15	21	20	10
36	Arumugam	63	12003	Peripheral vascular disease	0	1	1	1	0	0	0	3	18		1.5	280	26	13	22	21	11
37	Palanivel	80	15219	Cellulitis	1	1	1	2	2	2	2	111	25		2.7	330	27	14	24	23	12
38	Ponnan	52	16993	Diabetic Foot	1	1	1	1	2	2	2	10	18		1.1	350	30	15	25	23	13
39	Periyakaruppan	65	19218	Diabetic Foot	1	1	1	1	1	1	1	6	16		1.4	260	24	12	22	21	11
40	Sundaram	57	21784	Ca.Penis	0	1	1	1	1	1	0	5	18		2.2	290	25	12	25	24	13
41	Chellaiah	63	22763	Acid Peptic Disease	1	2	1	2	3	3	3	15	32	*	5.6	200	12	7	30	28	15
42	Raja	71	25901	Cellulitis	1	0	1	1	1	1	0	5	20		3.0	230	20	10	25	24	13
43	Kumarvel	62	28402	Hemorrhoids	0	0	0	0	0	0	0	0	16		1.1	300	26	13	25	23	14
44	Sami	63	29107	Liver abscess	1	1	1	1	1	2	2	9	15		2.8	320	29	15	23	22	12
45	Sakthivel	55	30525	Hemorrhoids	0	0	0	0	0	0	0	0	18		1.1	330	32	17	20	19	10
46	Vadivel	66	31257	Fournier's Gangrene	1	1	1	1	0	0	1	5	20		1.8	290	26	13	23	22	12
47	Sundaram	77	32399	Fistula in ano	1	1	2	1	3	3	4	15	30	*	4.9	240	19	10	25	24	12
48	Kannan	64	33865	Cellulitis	0	0	0	0	1	0	1	2	22		2.8	290	25	13	23	22	11
49	Kannabiran	65	35717	Diabetic Foot	0	2	0	2	2	2	1	9	30	*	5.1	230	15	8	30	29	15
50	Veluchamy	62	38122	Ca.Stomach	0	1	0	1	0	1	0	3	20		2.1	300	28	15	21	20	10