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Review Article

DETECTION OF PROSTATE CANCER: A REVIEW

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ABSTRACT

Prostate cancer is the most commonly diagnosed non-cutaneous cancer in men. Prostate-specific antigen (PSA) is the biomarker used for the screening of prostate cancer and other prostate-related problems. Not only the genetic factors are involved dietary factors, environmental factors but also responsible for the development of prostate cancer. Risk factors such as family history, age, chemical exposure, infection, and smoking are at the peak point for the development of prostate disease. Advanced age is one of the main risk factors. Radical prostatectomy is the most common therapy for small group of patients with high-grade tumors. Early screening of PSA reduces the incidence rate of prostate cancer. Mostly prostate abnormalities are seen in among male patients above the age of 50 years or older. In worldwide population, the epidemiology of prostate cancer is high in western countries and less in Asian countries.

Keywords: Prostate cancer, Radical prostatectomy, Epidemiology and infection.

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INTRODUCTION

Prostate gland

Anatomy

The human prostate is a male accessory sex gland. It is chestnut-shaped like structure, located in the basement of the pelvis and surrounds the neck area of the bladder and urethra [1]. Urethra plays a role in two main purposes urination and ejaculation. The healthy prostate weight is approximately 11 g, ranging between 7 and 16 g [2]. A thin vascularized fibrous sheath with encloses prostate gland along with a fibromuscular layer continues smooth muscle that surrounds the bladder. This fibromuscular layer extends and divides the prostate gland into different zones [3,4]. It allows running the prostatic fluid into the urethra during ejaculation. The milky color fluid that is secreted is rich in citric acid, fibrinolysin, and enzymes, especially acid phosphatase. These prostatic secretions are responsible for liquefying semen and trigger the sperm motility [5]. The proteins rich prostatic secretions change the environment of the vagina and hold the sperm in the female reproductive part for survival. The main hormone which is secreted by the male reproductive organ is testosterone which is responsible for synthesizing dihydrotestosterone (DHT) in the peripheral tissue. DHT is responsible for supervising the prostate gland [6] (Fig. 1).

Prostate function

The main function of the prostate gland is to store the seminal fluid. The prostate gland secretes small amount of alkaline fluid that makes 25% seminal fluid which allows the sperm to swim freely. Due to the alkalinity, it changes the vaginal tract environment which is acidic in nature and allows the sperm to stay viable in female reproductive part. The rich constituents present in prostatic secretions are prostate-specific antigen (PSA), along with citrate (18.7 mg/ml), zinc (488 µg/ml), spermine (243 mg/ml), and cholesterol (78 mg/ml) [7].

Prostate structure

The structure of prostate is divided into two different regions, zones or lobes. The zones are further divided into four different regions. Peripheral zone (PZ): About 70% part of the prostate are developed in this zone that encircles the urethra. In the PZ, there are 80% chances to develop prostatic cancer. 25% part of the prostate are formed in the central zone that surrounds the ejaculatory ducts and only 2.5% chances of prostatic cancers develop in this region. Cancers that establish here are more intrusive [8]. Remaining 20% of prostatic cancers develop in transition zone (TZ) which encircles the proximal urethra. Sometimes, enlargement of the TZ arises benign prostatic hyperplasia (BPH). Anterior fibromuscular zone is the final zone consists of muscle and fibrous tissue only [9].

Prostate carcinogenesis

Cancers are defined as uncontrolled production and subsequent spreading of cells to other parts of the body. All types of cell in the body that sustains such malignant changes and develops into cancers. Due to the unregulated cell division, the normal cell converts into the tumor cell that invades first into the localized area, then spread into the surrounding tissue, then spread through lymphatic system and vascular system to various other parts of the body [10,11]. Balance between the proliferation and cell death cycle is disturbed by unregulated division of cancer cells. This process is disrupted by mutation in DNA that causes cell to divide rapidly and multiply at higher rate. The arising mass can either be benign or malignant.

Prostatic intraepithelial neoplasia (PIN) is the possible precursor of prostatic carcinoma. It is responsible for the abnormal growth of epithelial cells that line the prostate gland. The irregular spaced of epithelial cells is characterized low-grade of PIN. Nuclei become hyperchromatic (with elevated chromatin) and pleomorphism (variation in size and shape). Higher level of hyperchromatism and pleomorphism is found when the PIN is in high grade. Cluster round cells simulating a raspberry shape that distinguish the PIN from adenocarcinoma [12]. The significant risk for adenocarcinoma due to presence of PIN however adenocarcinoma can stand up to 10 years before prostate carcinoma presents. [13].

Development and progression of prostate cancer

In developed countries, prostate cancer is the second most common analyzed cancer and the third most common cancer leads to death in men [14]. In 2006, it was reported that near about 1 in 8 men at the age of 75 years and 1 in 5 at the age of 85 years will develop prostate cancer accordingly [15]. A study conducted in 2007 by Collins *et al.* described the origin of prostate cancer from the glandular epithelium

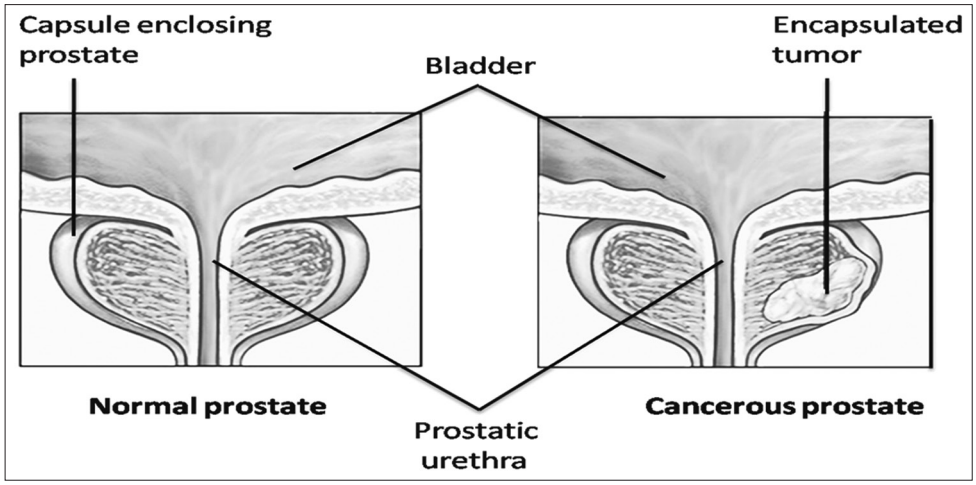

Fig. 1: Diagram of normal and localized prostate cancer

and the origin of tumor cells from luminal although both are dependent on androgens and represents luminal cell marker but increasing evidence from the research studies depict the derivation of cancer cells is less from differentiated stem cells [16,17]. Most prostate tumors are heterogeneous and multifocal, suggesting that multiple neoplastic foci have emerged and evolved independently [18]. Development and progression of prostate cancer is a multistep process. Malignant cells develop due to the genetic alteration. PIN, premalignant lesions considered as intermediary phase from benign epithelium to carcinoma, and it is quite comparable to prostate cancer with the exclusion that the basal layer is irregular but still presents. Due to furthermore changes the primarily malignant tumors rises in prostate, after that enter into prostate capsule and overthere attack on surrounded tissues of metastases [19].

Molecular changes

Cancer always generates from single somatic cells and by the action of many genetic changes it leads to a change in both phenotype and genotype [20]. Cancer leading to mutations mostly rise in the genes that are associated in the cell growth or cell death regulation [21]. Complexity or more than 100 types of cancer and their difference subtypes make it more difficult to point out the origin of the disease. Extensive research done from the past two decades on molecular, biochemical, and cellular process depicts how normal cells transforms or changes into the malignant cells. The enumerable majority of cancerous cell comprises entirely six different capabilities, namely, self-sufficiency in growth signals, insensitivity to antigrowth signals, evasion of apoptosis, infinite replication ability, sustained angiogenesis, and ability to invade tissue and metastasis [22]. Cell divisions of normal cells is under limitation, primarily observe the suitable external environment, then undergo cell division and if necessities, the cancerous cell have their own signals which set them free from the growth limitation of normal cells, so they divide and grow abnormally. The second capability is somewhat same as the previous stage; these cancerous cells have antigrowth signals, i.e., they do not receive signals to inhibit or to stop growth. The third feature or character of cancerous cell is the capability of assisting the growth since normal cells after complete cell division stop replicating, this phenomenon is controlled by telomeres. Telomeres are the segment of DNA or shortened by each round of DNA replication. This shortening of DNA does not allow cell to undergo further cell division and finally leads to cell death (apoptosis), but this phenomenon cannot be found in cancerous cell because of the ability to maintain the length of telomere. This allows them to replicate infinitely. The next capability is the evasion of apoptosis done by gene P53. Gene P53 is often found mutated in cancer cells, thus does not leading to normal apoptosis. Angiogenesis, which is the formation of new blood vessels, has the role for supplying or providing oxygen and nutrients to the tumor cells and the last but not the least capability is invasion in tissue and metastasis, in this cancerous cell gets attached to other cells and spreads throughout the body [23]. Cancer genes can be categorized into three main types: Oncogenes, tumor suppressor gene and cells involved in DNA repair.

Oncogenes are the first cancer-causing gene which causes unregulated cell growth [24]. They arise from proto-oncogenes which, in turn, responsible for normal cell growth. They usually remain dominant and cause mutations like increase in protein activity or loss of regulation, this increase in protein concentration or chromosomal translocation causes gene expression of different cell types. Some examples of oncogenes are RAS mutated in about 15% of cancer [25]. Antioncogenes (tumor suppressor gene) are inactivated by loss of function mutation. Knudson, in 1971, studied sporadic and familial retinoblastoma and based on his studies, he formulated two-hit model of carcinogenesis which gives brief explanation of the loss of function changes. In familial retinoblastoma, there is 50% chance to a child of inheriting this condition from one of the affected parents, but in sporadic retinoblastoma, no additional chance can be found [26].

The inherited form cannot cause predisposition of tumor development because of the germline mutations in one of the copies of tumor suppressor gene [27]. In the second copy of tumor suppressor gene, there occurs somatic mutation that will cause tumor progressions. Sporadic retinoblastoma is involved in two different hit models that are required for the same cell to develop into a tumor P53 gene being one of the most important tumor suppressor genes is found to be involved in key cancer control pathways such as cell cycle control apoptosis (cell death), angiogenesis (formation of new blood vessels), and genetic stability. The last category comprises the genes responsible for DNA repair mechanisms for normal DNA replication. In a single mutation in the whole process can result in genetic instability, thus leading to abnormal chromosome number or breaks. Moreover, certain other mutations in oncogenes and tumor suppressor genes, leading to other type of cancers are very rare in primary prostate cancer, but specific mutation for prostate cancer is yet to be discovered and therefore needs further research [28].

BPH

Benign prostatic hyperplasia also called BPH, a condition in which the prostate gland becomes enlarged. It is the most common prostate problem, BPH is not the cancer, but the symptoms of BPH are quiet similar to those of prostate cancer. Due to the overgrowth of epithelial nodules and stroma tissue in the TZ of the prostate, this leads the gland enlarged and the condition called BPH [29]. The two risk factor advanced age and circulating hormone (androgen) are responsible for developing for BPH. Due to the enlargement of prostate gland, the urethra becomes compress and pressure will increase within the bladder causing frequently contraction and less amount of urine is present, by this the bladder is not able to emptying itself and causes many other problems [30].

Androgen and estrogen are two sex steroid hormones are responsible for regulation of prostate and these are important for the prostate cell growth. Estrogen is originated through the stromal aromatization of androgen and estrogen: Androgen ratio increases in BPH patients [31,32]. By the advanced age of men, testosterone conc. becomes lower and estrogen becomes high in blood. According to the studies, higher concentrations of the estrogen increase the smooth muscle cell proliferation and differentiation; this may lead to BPH [33]. Aromatase inhibitors such as testolactone are antiestrogens, which are used for the treatment of BPH patients. These antiestrogens have a role for preventing the androgen to estrogen [34]. Androgenic steroid testosterone is converted into DHT which is important for the function of secretory epithelial cells; this conversion is catalyzed by 5α-reductase isoenzyme type 1 and 2. 5α-reductase type 1 is most often found in the liver and skin but less amount in prostate. Type-2 is most prevalent in prostate. 5α-reductase type-2 is responsible for converting the androgenic steroid testosterone to DHT in prostate gland. In BPH condition, higher activity of 5α-reductase has been demonstrated as compared to normal tissue [35]. Finasteride, a 5α-reductase type 2 inhibitor, has been used for the treatment of BPH patients [36,37].

Expression of peptide growth factor in BPH and prostate cancer

Prostate cancer poses a significant clinical challenge both in terms of its prevalence and its complexity [38]. Several prostate cancer-associated genes including c-myc, insulin-like growth factor-I, P27, and peptide growth factor are highly expressed across all of the tumor types [39]. The cellular growth, differentiation, and programmed cell death (apoptosis) are regulated with some peptide growth factor and proteins. Most important families such as epidermal growth factor (EGF) family, insulin growth factor (IGF) family, the transforming growth factor beta (TGFB) family, and the vascular endothelial growth factor (VEGF) family, these families are involved for the progression of prostate cancer [40,41]. EGF family has further two members EGF and TGFA. EGF responsible for promoting the proliferation of cell and also involved in embryogenesis, angiogenesis, and cellular differentiation [42]. This EGF protein is overexpressed in benign and malignant [43,44]. Fibroblast growth factor (FGF) has several members FGF (bFGF or FGF2), acidic FGF (aFGF or FGF1), and keratinocyte growth factor (KGF or FGF7), these families are highly expressed in varying levels by prostatic cells [45]. FGF2 families are overexpressed in mRNA and its presence implicated the development of BPH [46,47]. TGFB belong to TGFB family [48], the level of TGFB increase in prostatic neoplasia [48,49] and this finding associated with the development of tumor and progression [50,51] because TGFB biological activities are exploited by cancer cells. TGFB promotes angiogenesis [52] along with this TGFB is immunosupressor [53-55]. TGFB protects the cancer cells from the host immune system [56], it plays a key role in extracellular matrix by enhancing the invasiveness and metastatic ability of malignant cells [57,58]. VEGF is also involved for developing tumor and metastasis [59]. VEGF expressed in BPH and prostate cancer epithelial cells [60,61] and its appearance plays a role for its tumor growth, inducing angiogenesis [62]. IGF1 productions in epithelial cells of the prostate have a role for the development of prostate adenocarcinoma [63]. In prostate malignancy, the level of IGF1 is increased in blood but not with BPH [64,65].

Biomarkers for evaluating prostate cancer

PSA

Prostate belongs to human kallikrein gene family is a serine protease with chymotrypsin-like activity. PSA is a single chain glycoprotein made of 237 amino acids containing oligosaccharide side chain. PSA molecular weight is approximately 30,000 Dalton [66-69]. The glandular epithelium of prostate gland formed major portion of PSA. The breast cancer, salivary gland neoplasm, periurethral and anal gland, cells of male urethra, breast milk, blood, and urine originate PSA [70,71]. The prostate gland contains acinar cells that formed glycoprotein also known as PSA [72]. Main function of PSA is to dissolve the seminal clot that is formed after ejaculation and helps to transport the spermatozoa in female reproductive tract. PSA has two types:

1. Complex PSA formed by complex combination of serum protein.
2. Free PSA is made by free combination of serum protein.

When these two combines together, they formed total PSA. Seminal fluid contains high percentage of PSA (0.5–2.0 mg/ml), whereas in blood, PSA concentration is quite less, i.e., 1000 times. The changes in concentration are independent of other protein; its changes depend on serum testosterone levels [73]. If the leakage of PSA from the prostate gland, the level of PSA becomes low and high level of PSA is allied with prostatic pathology including prostatitis, BPH, and prostate cancer [74-78]. In 1982, PSA was used to describe as prostate cancer marker, PSA first screening report came into existence in 1991 [79,80]. The occurrences of prostate cancer have had a less increase in western countries over last 30 years [81]. The high grades of PSA level are found in 50 years of age or in older males (Table 1).

Table 1: PSA reference ranges (Oesterling *et al.*, 1993)

Age (years)	Reference ranges (ng/dl)
50–59	0–3.5
60–69	0–4.5
70–79	0–6.5

PSA: Prostate-specific antigen

Digital rectal examination (DRE)

DRE is the other method that is used for the examination of prostate abnormalities [82,83]. Enlargement of prostate gland can be found in BPH patients. DRE is performed through rectum by finger to feel the hardness of the gland or irregular or hard lump indicates the presence of tumor. This method is not able to examine all prostate abnormalities through rectum, so DRE method was not the best method for diagnosis of all prostate problems [84].

Transrectal ultrasound (TRUS)

TRUS can be used for guidance of needle biopsies of the prostate gland. This method examines the enlargement of gland, cancer nodules, and tumor invasion to the seminal vesicles [85]. Other molecular markers which are used for finding prostate abnormalities such as prostate stem cell antigen, early prostate cancer antigen, hepsin, enhancer of zeste homolog gene 2, human glandular kallikrein 2, TGF-1, and chromogranin A have been suggested as potential promising biomarkers for prostate cancer [86].

Symptoms and diagnosis

Symptoms are not found for many years in prostate tumor because it is usually slow-growing disease. In early stage of prostate cancer, symptoms are not found. Its symptom mostly affects the urination because its location surrounding the urethra. In prostate cancer, symptoms like frequent urination. Nocturia , hematuria, difficulty in maintain a steady stream of urine and dysuria are observed. These symptoms also found in other prostate disease like BPH. It also affects the sexual function, difficulty in achieving erection or painful ejaculation. If the prostate cancer is in late stage, it can spread to other organs such as bone, lymph nodes, and causing bone pain in pelvis or ribs region. Diagnosis of the prostate cancer must be proofed by the needle biopsy. The international classification of diseases version 10 classifies malignant neoplasm of the prostate as code c61 [87].

Age

According to the ages, the risk of developing prostate disease is increased. Age is the one of the most common risk factors. Prostate cancers will significantly increase by the advanced ages and by the age of 70 years; approximately 65% men have at least microscopic evidence of prostate cancers. There is a positive correlation between time and prostate cancer progression [88].

Family history

It has been suggested that heredity may play a role in prostate cancer. Men with a family history of prostate cancer may have a higher risk of having this disease [89-91].

Ethnicity

In developed countries such as Africa and America, men of these countries have a high risk of developing prostate cancer [92,93] but in Asian countries, lower risk of developing prostate cancer and if they move to North America risk will increases [94].

Dietary factors

Dietary factors play an important role in African and American males and have a higher risk of prostate cancer in comparison to Asian males. High-fat intake may increase the risk of prostate cancer. According to the researchers study, they suggest that fat elevated the production of hormone testosterone that may promote the prostate cancer cell growth. Specific subtypes of fat are responsible for influencing the prostate cancer cell growth. Heterocyclic amines), a group of carcinogens known as 2-amino-1-methyl-6-phenylimidazo [4, 5-b] pyridine (PhIP), are found in grilled beef, pork, chicken, lamb, and fish. It has been indicated that PhIP relates to prostate cancer incidence [95].

Smoking

Cigarette smoking is one of the risks of prostate cancer. The circulatory level of steroidal hormone is altered by smoking [96]. A few hypothetical mechanisms were proposed to enhance the risk of prostate cancer. It has been suggested that smoking can increase the circulating levels of bioavailable testosterone and lower levels of bioavailable estradiol in men [97]. There are significant positive correlations between cigarettes smoked/day and serum total androstenedione as well as total and free testosterone in men [98]. Data from a population-based case–control study suggest that smoking is a risk factor of prostate cancer. Current smokers appear to be at moderately increased risk for this disease compared with non-smokers [99].

Exposure to chemicals

Prostate cancer and chemical exposure relation are not fully understood, but it is reported that males who were indulge with heavy labor and work with certain metals and chemicals, including cadmium, dimethylformamide, and acrylonitrile, may be at higher risk for prostate cancer. Some studies indicate farmers are at higher risk of prostate cancer [100].

Infection and inflammation

Genetic factors that affect the body's response to viruses can also associate with inherited prostate cancer. Some relation between the prostate cancer and bacterial or viral infections is seen in such infections such as herpes virus, human papillomavirus, and cytomegalovirus. It implied that genetic susceptibilities in men could develop a chronic inflammatory condition in the prostate by viral infection and possibly initiate cancerous changes. It has been suggested that exposure to environmental factors such as infectious agents and dietary carcinogens, and hormonal imbalances could lead to prostate injury and develop chronic inflammation and regenerative risk factor lesions, referred to as proliferative inflammatory atrophy, which could progress to PIN and eventually invasive carcinoma [101]. However, some recent studies have shown that there is no link between viral infections and prostate cancer development [102].

Treatment

Surgery

Radical prostatectomy is the surgery procedure in which the entire prostate gland and surrounding tissue such as seminal vesicles are removed. Some amount of removed organ like lymph nodes is performed for biopsy to examine the cancer has metastasized or not. This therapy is used in early stage of prostate cancer and after surgery the patient can survive for 10–20 years. The main purpose of this therapy is to remove the whole cancer and prevents its spread to other parts of the body. There is some risk after surgery such as impotence, heart attack, stroke, blood clot, and infection [103].

Radiation therapy

Radiation therapy is one of the treatments for prostate cancer for several decades. Prostate cancer is a radiation-sensitive neoplasm that determines a classic sigmoid dose-response curve to X-rays. Higher volume tumors need higher radiation doses. Bladder and rectum are at risk when radiation is performed [104].

Hormone therapy

Hormone therapy causes blocking hormonal action due to which the growth of cancer cells stops, for example, luteinizing hormone producing some others hormones, which are able to inhibition of gonadotropin secretion. Following an early stimulation of gonadotropin, chronic administration of leuprolide acetate causes suppression of testicular steroidogenesis. This proves and shows that luteinizing hormone-releasing hormone agonists and results inhibition of the growth of certain hormone which promote tumors (such as prostatic tumors). Examples are leuprolide, goserelin, and buserelin. Antiandrogens exert its action by inhibiting androgen uptake and/or by inhibiting nuclear binding of androgen to the androgen receptors on prostatic cells such as flutamide and nilutamide.

Studies are still being carried on to find the ideal therapy for localized prostate cancer. Currently, the two most common therapies used in the United States to treat prostate cancer remain radical prostatectomy and radiation therapy [105]. The newer focal therapy consists of cryoablation techniques and heat energy-based treatments high intensity focused ultrasound, radiofrequency interstitial tumor ablation, and thermal brachytherapy. Radioactive seeds were first used by Dr. Anthoy D'Amico at Harvard Medical School to treat early stages of prostate cancer. Magnetic resonance imaging was used to place 100 radioactive seeds into tumors inside prostate to destroy cancer cells. For some patients, it may be superior to the usual methods of surgical removal of the gland [106].

Epidemiology

In western populations, prostate cancer is the most common cancer in men. In 2005, the new cases were expected beside 232,090 [38]**.** From the early 1990s, the incidence rates were greatly increased worldwide [107]. In western countries, due to the increased detection of BPH with surgical procedure, particularly TURP, this may lead the incidence rate was high in late 1970s and early 1980s [108]. The sharpened incidence rate was rise in between 1986 and 1992 by the more use of PSA [109]. In the US, the incidence rate was usually dropped down in mid 1990s, but recently, the rate was started and slowly rises again. In general, in Asian countries, the incidence rate is low but recently found that the rate was raised more than the western countries [110].

Incidence and mortality

The variation in incidence rate and mortality rate, by the differently appearance of prostate cancer, was directly impact on world's population, while the examination of these rates, it provides initiation about the disease and also helpful for the generation of hypothesis for further research [111].

Incidence

Prostate cancer considers incidence rate is variable in whole world. African and American countries rates are at highest in the world (185.4/100,000 person-years), displacing by Caucasian-Americans (107.8/100,000 person-years) (Fig. 2). The Caribbean and in Brazil rate fall on 92–96/100,000 person-years. Rates are much lower (28–

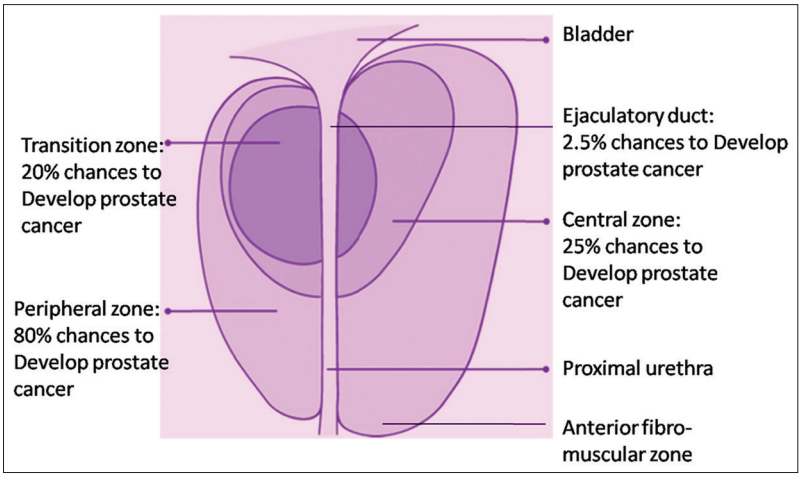

Fig. 2: Different zones of prostate gland

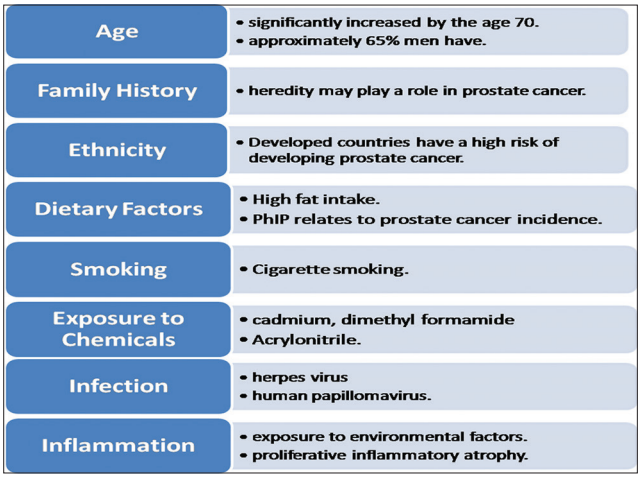

Fig. 3: Risk factors involved in prostate cancer

42/100,000 per years) in Central America and other parts of South America. In Europe, rates are 15–100/100,000 person-years, but Western Europe, rates are higher than Eastern Europe (15–36/100,000 person-years). The highest incidence rate reported in such countries; Australia, New Zealand, Northern and Western Europe, and Northern America.

Moderate rates were reported in South America and Eastern Europe and lowest rates were found from South-Central Asia. In Asian countries, the incidence rate found lower than the other countries. However, widely the western countries such as Japan, Israel, and the Philippines (22–47/100,000 person-years) were found more incidence rates than Thailand, India, Pakistan, Shanghai, and China (3–7/100,000 person-years) [112,113]. Part of the difference in worldwide incidence rates is related to the extent of prostate cancer screening, especially the less frequent use of prostate-specific antigen (PSA) testing in developing countries. However, screening practice differences alone are unlikely to explain the nearly 60-fold difference in prostate cancer risk between high- and low-risk populations.

Mortality

Only one in six American men diagnosed with prostate cancer will eventually die from it. Nevertheless, 30,350 prostate cancer deaths are expected in the U.S. in 2005, making prostate cancer the second leading cause of cancer death among U.S. men, after lung cancer [38]. Overall, the pattern of mortality worldwide reflects that of incidence, although the mortality rates show less variation between countries. Nevertheless, mortality rates are still higher in Western nations than in lower risk Asian countries. Interestingly, the world's highest mortality rates were seen in the Caribbean nations of Barbados, the Bahamas, and Trinidad and Tobago, where there are large populations of men of African descent. Mortality was higher in Scandinavian countries and parts of northern Europe than in the U.S. (18.7–23.6 vs. 14.0/100,000 person-years), and lowest of all in the Asian countries of South Korea, the Philippines, and Japan (1.6–4.4/100,000 person-years).The patterns of incidence and mortality worldwide provide a number of interesting leads [113]. The pronounced excess risk of prostate cancer in western nations suggests that factors associated with westernization, such as diet and obesity, may be positively associated with prostate cancer etiology. In addition, prostate cancer's disproportionate impact on African-Americans and Caribbean men suggests that factors associated with African ancestry may also play a role in prostate cancer etiology. While it is not known whether the risk factors explaining the observed patterns are environmental, lifestyle, or genetic, it is likely that a complex interplay of these factors is associated with prostate cancer development [114].

CONCLUSION

Prostate cancer is the third most leading cause of death from cancer in men after colorectal and lung cancer. PSA testing is used for the early detection of prostate cancer or many other prostate-related abnormalities such as BPH and prostatitis. PSA belongs to human kallikrein gene family, which is serine protease with chymotrypsin-like activity. PSA is secreted in the epithelial cells of the prostate gland and can be demonstrated in biopsy samples or other histological specimens using immunohistochemistry. The level of PSA rises in prostate abnormalities such as in BPH and prostate carcinoma. Since the level of PSA was comparatively higher in patients with a history of smoking as observed from the present study, the hazards of smoking (risk factor) cannot be ignored. Hence, by this study, we concluded that the prostate cancer is a major health problem and the incidence is gradually increased. Curative treatment of prostate cancer reduces the disease-specific mortality significantly. Early screening of disease by PSA marker reduces the mortality of prostate cancer. The prevalence of prostate cancer in the studied population was 15%. It is hoped that this work would help alert our adult male population on the need to go for early and routine screening for prostate disorders (from the age of 50 years). Early detection of BPH and prostate cancer makes management easy and lowers the impact of disease.

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