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A Review on Most Common Male Malignancy - Prostate cancer

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Review Article

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ABSTRACT

Paper contains the study on prostate cancer, which is now very common in males, males with age above 60 are more prone to this cancer due to multiple reasons, the study includes the reason for the occurrence of the cancer and also a detail study about the diagnosis and the treatment. The study includes a detailed study of treatment for the prostate cancer, varying from stage to stage.

ETIOLOGY AND EPIDEMIOLOGY

The etiology of prostate malignancy is not completely caught on. It is thought to be caused due to many factors, which may also include hereditary and ecological triggers ^[1].

On a fundamental level, prostate growth is brought about by changes in the DNA of a prostate cell. Researchers have gained awesome ground in seeing how certain progressions in DNA can make typical prostate cells become strangely and structure growths ^[2-6]. DNA is the compound in each of our cells that makes up our qualities, the guidelines for almost everything our cells do. We generally resemble our guardians in light of the fact that they are the wellspring of our DNA. Nonetheless, DNA influences more than what we look like ^[7,8].

A few qualities control when our cells develop, separate into new cells, and pass on ^[9]. Certain qualities that help cells develop, gap, and stay alive are called oncogenes. Others that regularly ease off cell division, repair botches in DNA, or reason cells to bite the dust at the ideal time are called tumor silencer qualities. Growth can be created to some extent by DNA changes (transformations) that turn on oncogenes or kill tumor silencer qualities ^[10-15].

DNA changes can either be acquired from a guardian or can be procured amid a man's lifetime.

Acquired DNA mutations

Acquired DNA changes in specific qualities appear to bring about around 5% to 10% of prostate growths. A few changed qualities have been connected to a man's acquired propensity to create prostate tumor, including:

RNASEL (once in the past HPC1): The typical capacity of this tumor silencer quality is to help cells pass on when something turns out badly inside them. Acquired transformations in this quality may let strange cells live more than they ought to, which can prompt an expanded danger of prostate disease.

BRCA1 and BRCA2: These tumor silencer qualities typically help repair botches in a phone's DNA (or reason the phone to pass on if the oversight can't be settled). Acquired changes in these qualities all the more ordinarily cause bosom and ovarian tumor in ladies. Anyway, acquired BRCA changes additionally represent a little number of prostate tumors.

DNA confuse repair qualities, (for example, MSH2 and MLH1): These qualities ordinarily help fix botches (bungles) in DNA that are made when a phone is get ready to separation into 2 new cells. (Cells must make another duplicate of their DNA every time they isolate.) Men with acquired changes in these qualities have a condition known as Lynch disorder, and are at expanded danger of colorectal, prostate, and some different diseases.

Other acquired quality changes may represent a few instances of inherited prostate tumor, albeit none of these is a noteworthy reason. More research is being done on these qualities ^[16,17].

DNA Transformations Gained amid a Man's lifetime

Most DNA transformations identified with prostate growth appear to create amid a man's life as opposed to having been acquired.

Each time a cell plans to separation into 2 new cells, it must duplicate its DNA. This procedure is not immaculate, and here and there blunders happen, leaving defective DNA in the new cell. It is not clear how regularly these DNA changes may be arbitrary occasions, and how frequently they are affected by different variables (diet, hormone levels, and so on). When all is said in done, the all the more rapidly prostate cells develop and separate, the more risks there are for transformations to happen. Subsequently, anything that accelerates this procedure may make prostate malignancy more probable ^[18-20].

The advancement of prostate disease may be connected to expanded levels of specific hormones. Large amounts of androgens (male hormones, for example, testosterone) advance prostate cell development, and may add to prostate disease chance in a few men ^[21].

A few specialists have noticed that men with elevated amounts of another hormone, insulin-like development variable 1 (IGF-1), are more inclined to get prostate growth. IGF-1 is like insulin, however it influences cell development, not sugar digestion system. Be that as it may, different studies have not discovered a connection between IGF-1 and prostate tumor ^[22-30]. Further research is expected to comprehend these discoveries.

As said in the "What are the danger variables for prostate growth?" area, a few studies have found that irritation in the prostate may add to prostate disease ^[31,32]. One hypothesis is that irritation may prompt cell DNA harm, which may thusly push a cell closer to getting to be malignant. More research around there is required.

Introduction to radiation or disease bringing about chemicals can bring about DNA changes in numerous organs, however these elements have not been ended up being imperative reasons for transformations in prostate cells.

The danger of prostate disease is firmly connected with seniority. Just 1% of determinations in 2011 were in patients less than 50 years old ^[33-40]. Modifiable danger variables ought not be disregarded in illness avoidance and administration. A late meta-investigation showed tobacco smoking as a noteworthy danger variable for lethal prostate tumor and a late efficient survey exhibited great confirmation that being overweight or corpulent expands the danger of creating propelled prostate disease. Patients of dark African or Caribbean ethnic source have one in four lifetime frequency of prostate growth. Those with a family history (more than one influenced first-degree relative) convey up to 3.5 times the RR of adding to the infection. In spite of the fact that a critical extent of men beyond 80 years old are thought to exhibit histological proof of prostate growth, its high predominance does not make an interpretation of specifically into infection death rates - there were 9,632 passings from prostate disease in 2010. The expanding malady load hence speaks to a developing test to the medicinal services framework, especially with vast quantities of long haul survivors of the condition.

In the UK, there are around 41,700 new victims of prostate cancer for every year. It is normal that one in eight men will get the analysis in their lifetime and this is anticipated to increment.

CLASSIFICATION OF PROSTATE CANCER

Prostate malignancy is classified as localized, mainly progressed or metastatic. At present, ailment characterization demonstrates condition-particular death rates and illuminates treatment standards. Then again, ideal administration of the condition remains the subject of consistent assessment, as our comprehension of its pathogenesis and treatment adequacy over the long time moves forward.

SCREENING

Screening asymptomatic men for prostate growth stays questionable. A late investigation of the European Randomized Study of Screening for Prostate Cancer trial exhibited a huge lessening in prostate growth mortality more than a 13 year study period.

On the other hand, information recommend a screening populace of 781 patients is obliged to make one new prostate growth analysis and 27 new analyses are expected to maintain a strategic distance from one passing from prostate cancer.

Periodically, clinical elements of prostate growth might just get to be obvious with the immediate or aberrant improvement of metastatic illness.

This may identify with systemic elements of threat, for example, general disquietude / weight reduction, or the site of auxiliary growth association, for instance, hard agony or neurotic cracks.

Without UTI, all patients with LUTS ought to be offered a computerized rectal and outer genitalia examination and advised around a PSA test. If irregular, these patients ought to be alluded for a urological pro feeling.

The complete determination of prostate tumor is made histologically through the utilization of focused on biopsies.

Diagnosis

The slippery way of prostate growth implies numerous patients with on time infection are asymptomatic. Indications of generally propelled sickness may be hard to recognize from those connected with BPH. Lower urinary tract indications (LUTS, for example, expanded urinary recurrence, nocturia, poor stream and terminal spilling, prevail in these regularly existing together conditions.

PSA

Prostate specific antigen, it is used in the diagnosis of the malignancy of prostate. It is made by the cells of prostate gland.

Analysis may be coincidental, with a high record of suspicion created most ordinarily from a raised PSA estimation, or every so often from disconnected imaging.

A PSA < 3 ng/mL in patients matured under 50 ng/ml, and < 4ng/mL for men aged 60-69, is ordinary. In men more than 70, the typical extent is stretched out to 5 ng/mL. A PSA < 10 ng/mL is generally connected with Benign Prostatic Hyperplasia and / or unending prostatic irritation, with just 20% of men testing positive for growth histologically. The probability of threat increments up to 50% in men with a PSA > 10 ng /mL.

Inflammation of prostate gland alluded as "prostatitis" is normally seen in the prostate biopsies or prostatectomy and benevolent prostatic hyperplasia examples. Men with perpetual prostatitis frequently encounter perineal or pelvic torment with or without irritative voiding manifestations. Prostatitis is likewise a settled reason for raised PSA. As indicated by National Institutes of Health, prostatitis has been ordered into four clinical classifications: Category I, intense bacterial prostatitis; Category II, endless bacterial prostatitis; Category III, ceaseless pelvic torment disorder either incendiary (IIIa) or noninflammatory (IIIb); and Category IV, asymptomatic provocative prostatitis. Past study from our gathering has shown that men having both prostatitis and moderate to extreme periodontitis have higher PSA levels contrasted with those having either condition alone. Gram-negative microscopic organisms have been proposed as etiologic operators for periodontitis and classification I and II prostatitis. A bacterial etiology for classes III and IV prostatitis, on the other hand, has not yet been recognized. Cytokine awkwardness and modified levels of ace and calming cytokines has been embroiled in the pathogenesis of both endless periodontitis and prostatitis. Given the closeness in etiopathogenesis of prostatitis and periodontitis, it is conceivable that a relationship between the two conditions exists, that may show hoisted PSA levels in the circling blood. The motivation behind this study was to survey whether non-surgical periodontal treatment has an impact on prostate indication score and serum PSA and levels of proinflammatory cytokines, viz., IL-1 β and CRP in men with hoisted serum PSA and perpetual periodontitis

The highest level is the transrectal ultrasound-guided (TRUS) biopsy. On the other hand, this is connected with critical dangers of contamination, dying, agony and infrequently, intense urinary maintenance.

An option biopsy system is by means of the format guided transperineal methodology. This is not yet routine practice in the UK, but rather may be considered for patients with beforehand uncertain / negative TRUS biopsies and hoisted PSA results. The parts of both strategies are under assessment.

The Gleason score is utilized to report the histological evaluating of prostate biopsies. The aggregate score may go from six (non-forceful malady) to 10 (profoundly forceful). This evaluating framework is utilized as a precise indicator of infection movement in conjunction with PSA estimation and radiological organizing (TNM).

TREATMENT

Minimal disease (stage 0/a1)

No randomized controlled trials have been performed contrasting treatment with no treatment in patients with Stage 0/A1 prostate tumor. On the off chance that arrangement, rates of ailment movement of 5 to 16 percent have been accounted for with an interim to movement of six to nine years. Notwithstanding, the survival of men with Stage 0/A1 prostate tumor is practically identical to the normal survival of men of comparative ages in the all-inclusive community. Since the medicines for confined prostate growth are connected with noteworthy dreariness and survival does not seem, by all accounts, to be influenced in Stage 0/A1 ailment, our proposed quality pointer obliges that no treatment be offered to men age 60 and more established with Stage 0/A1 infection. Since sickness movement increments with time, a few specialists do suggest treating more youthful men (under age 60) with Stage 0/A1 ailment. In any case, in light of the fact that there is no agreement with respect to the administration of Stage 0/A1 sickness in men more youthful than 60, we have constrained our quality marker to men 60 and more established.

Localised disease (stage i & ii / a2 & b)

Treatment of confined prostate disease stays disputable. The best seek after curing prostate growth is with radical prostatectomy or radiation treatment while it is still confined. The main randomized controlled trial of radical prostatectomy with no treatment neglected to exhibit a survival point of preference with radical prostatectomy. In any case, the dependability of this outcome is regularly addressed on the grounds that the example size was just 142, and just 111 of 142 patients included in the trial were accessible for investigation. A few non-randomized investigations of hopeful administration ("watchful holding up") of patients with confined prostate malignancy have exhibited ten year sickness particular survival rates of pretty nearly 85 percent and ten year general survival rates of more or less 60 percent. These outcomes are practically identical to those got with radical prostatectomy and radiation treatment. The main randomized controlled trial contrasting radical prostatectomy and radiation treatment utilized time to first treatment disappointment as its essential endpoint and demonstrated leeway for radical prostatectomy. Be that as it may, the study has been censured in light of the fact that the patients treated with radiation were not surgically organized. At the 1987 NIH Consensus meeting on Prostate Cancer, no agreement with respect to treatment was come to, and none has been come to since. Still, most American specialists suggest complete treatment for restricted prostate malignancy for men with a future more noteworthy than ten years. Radical prostatectomy is normally performed through a retropubic methodology and more up to date surgical strategies permit saving of the neurovascular package all together to reduction the rate of incontinence and feebleness. As a rule, a pelvic lymphadenectomy is performed before the prostatectomy, and the specialist just continues if the lymph hubs are negative for metastatic sickness on solidified segment. Post-agent difficulties incorporate incontinence, urethral stricture, rectal harm, ineptitude, and the grimness and mortality connected with general anesthesia and a noteworthy surgical system (30 day mortality of two percent in one investigation of 10,600 radical prostatectomies). Reports in the writing of inconvenience rates after radical prostatectomy are truly fluctuated. In one expansive contextual analysis of men experiencing the nerve-saving radical prostatectomy, huge incontinence happened in six percent of men, while 35 to 60 percent of men who were sexually powerful before surgery got to be feeble after the technique. On the other hand, in a national review of Medicare patients who experienced radical prostatectomy in 1988-1990, more than 30 percent of men reported the requirement for cushions or braces for incontinence, and around 60 percent reported having no erections since surgery, with 90 percent reporting no erections adequate for intercourse amid the month before the study.

While radioactive inserts are utilized to treat prostate malignancy, the most well-known method right now being used today is outside shaft radiation. Utilizing a straight quickening agent 67 Gy to 70 Gy is conveyed to the prostatic quaint little inn tissues more than six to seven weeks, with the pelvic lymph hubs getting pretty nearly 50 Gy. In the event that radiation treatment is picked as authoritative treatment, lymphadenectomy is generally not performed, bringing about those cases which are clinically Stage I or II / An or B yet pathologically Stage III or IV / C or D not being distinguished.

This makes challenges when attempting to analyze the results of clinical trials of patients treated with radiation treatment with those treated with radical prostatectomy. The entanglements of radiation treatment, however rare, incorporate the runs, proctitis, cystitis, hematuria, rectal dying, butt-centric stricture, urethral stricture, rectal ulcer, inside obstacle. These entanglements are normally reversible and once in a while get to be constant. Sexual strength is for the most part protected in the fleeting with radiation treatment, yet may reduce over the long haul. Given the absence of clear proof for a specific treatment for limited prostate malignancy, the variable confusion rates after radical prostatectomy and radiation treatment, and the requirement for patients to have the choice of a therapeutic treatment when giving growth at a remedial stage, we propose a quality pointer indicating that men under age 65 with Stage II / A2&B ought to have been offered radical prostatectomy or radiation treatment.

Locally advanced disease (stage iii / c)

The ideal treatment for patients with generally propelled prostate tumor is even less clear than that for confined illness. The consequences of radical prostatectomy in Stage III / C patients are incredibly substandard compared to the outcomes in confined illness. As surgical evacuation of the organ is regularly troublesome in Stage III / C prostate tumor, radiation treatment is by and large chosen for patients with clinical Stage C prostate tumor. The ten year general survival with both radical prostatectomy and radiation treatment for Stage III / C prostate tumor is around 35 percent. Neoadjuvant androgen removal treatment has had some accomplishment in "downstaging" patients so that PSA levels get to be imperceptible and the staying tumor is organ bound in additional patients at surgery. Keeping in mind one randomized investigation of radiation treatment with and without androgen removal demonstrated leverage in movement free survival at five years for the arm that got androgen removal, to date, neoadjuvant androgen removal has not been demonstrated to give leverage in general survival. Another treatment choice for Stage III/C is early androgen removal treatment (which will be examined in the Advanced Ailment area); yet there is no proof that it drags out survival. Still another choice is eager administration and treatment when important to alleviate manifestations.

Given the poor ten year survival with provincially propelled ailment, numerous specialists would prescribe more forceful treatment in more youthful men (not exactly age 60). On the off chance that pathologic organizing affirmed Stage III / C sickness, numerous specialists would prescribe radical prostatectomy, if actually practical, or radiation treatment with corrective purpose. As there is little agreement on the most proficient method to treat asymptomatic patients with Stage III / C prostate disease, we don't prescribe a quality pointer for the treatment of this gathering of patients.

Advanced disease (stage iv / d)

The most widely recognized manifestations of cutting edge prostate growth start from the urinary tract or from bone metastasis. Generally, more than 50 percent of patients present with bone metastases (preceding the approach of PSA screening). Patients with bone torment, instinctive association, approaching rope pressure, obstructive urinary manifestations or hydronephrosis ought to get androgen removal treatment for concealment. Specialists likewise by and large suggest treating patients with asymptomatic propelled prostate tumor with androgen removal treatment; on the other hand, the information for this are not indisputable. In randomized controlled trials, androgen removal treatment seems to moderate ailment movement in Stage IV / D prostate malignancy, and may enhance general survival; then again, it is not clear if beginning androgen removal treatment early, while patients are still asymptomatic, has favorable position over holding up until patients create side effects.

There are different ways to deal with androgen removal treatment including orchiectomy alone, monotherapy with a luteinizing hormone-discharging hormone (LHRH) simple, monotherapy with non-steroidal antiandrogen therapy, or maximal androgen bar (either orchiectomy or a LHRH simple and antiandrogen treatment). The significant reactions of all androgen removal medicines incorporate barrenness (all around), bosom delicacy, and hot flashes. Moreover, with LHRH analogs, numerous patients encounter a flare of bone agony what's more, different indications subsequent to starting treatment. Since 1941, orchiectomy has been viewed as the standard removal treatment for cutting edge prostate growth; then again, it has not been contrasted with no treatment in a randomized trial, nor has it been demonstrated to delay survival. The main randomized placebo-controlled trial of androgen removal contrasted DES and placebo. The VACURG study demonstrated an abating of illness movement in Stage IV / D patients treated with DES 5 mg/day contrasted and placebo, yet general survival was more regrettable in the gathering treated with DES (diethylstilbestrol), to a great extent because of an increment in cardiovascular mortality. As treatment with DES in this study was connected with an increment in cardiovascular entanglements and heart mortality, DES has been generally supplanted by the more up to date drugs (LHRH analogs and antiandrogens). Randomized controlled trials of two-sided orchiectomy, the LHRH simple goserelin, and DES have demonstrated to every one of them to be just as viable regarding moderating illness movement. Then again, none of these studies answer the particular inquiry of whether prompt treatment has a survival point of preference over conceded treatment with androgen barricade for cutting edge prostate growth. A randomized trial is presently in advancement to attempt to answer this inquiry.

REFERENCES

1. Mu P, et al. MicroRNAs in Prostate Cancer: Small RNAs with Big Roles. *J Clin Cell Immunol*. 2015;6:315.
2. Alwithanani N, et al. Periodontal Treatment Improves Prostate Symptoms and Lowers Serum PSA in Men with High PSA and Chronic Periodontitis. *Dentistry*. 2015;5:284.
3. Lindholm PF, et al. Monocyte-Induced Prostate Cancer Cell Invasion is Mediated by Chemokine ligand 2 and Nuclear Factor- κ B Activity. *J Clin Cell Immunol*. 2015;6:308.
4. Mahesh S, et al. Reconstruction of Gene Regulatory Network to Identify Prognostic Molecular Markers of the Reactive Stroma of Breast and Prostate Cancer Using Information Theoretic Approach. *IJIRCCCE*.
5. Tajbakhsh J, et al. Using 3D High-Content Analysis and Epigenetic Phenotyping of Cells in the Characterization of Human Prostate Tissue Heterogeneity. *Single Cell Biol*. 2015;4:i104.
6. Boucif A, et al. Chronic Prostatitis (CP) in Atlas Shepherd Dog: A Case-Control Study. *Clin Microbiol*. 2015;4:197.
7. Bottu S, et al. Recent Developments in Cancer. *J Dev Drugs*. 2015;4:r001.
8. Maria G, et al. Characterization of Kallireins and microRNAs in Urine Sediment for the Discrimination of Prostate Cancer from Benign Prostatic Hyperplasia. *J Cancer Sci Ther*. 2015;7:130-136.
9. Ravindranathan P. Targeting Protein Interactions: A Novel Therapeutic Strategy against Prostate Cancer. *J Blood Lymph*. 2015;5:e119.
10. Chiang EC, et al. Selenium Form-Dependent Anti-Carcinogenesis: Preferential Elimination of Oxidant-Damaged Prostate Cancer Cell Populations by Methylseleninic Acid is Not Shared by Selenite. *Vitam Miner*. 2015;4:126.
11. Dahlbeck S, et al. A Prospective Pilot Study of Single 19 Gy Fraction High-Dose-Rate Brachytherapy for Favorable-Risk Adenocarcinoma of the Prostate. *J Nucl Med Radiat Ther*. 2015;6:215.
12. Mobit P, et al. What do Dosimetric Errors Encountered in Prostate Implant Brachytherapy tell us about α/β ?. *J Nucl Med Radiat Ther*. 2015;6:214.
13. Wrzosek M, et al. Diagnostic Value of Serum Ghrelin Levels in Diabetic Men with Benign Prostate Hypertrophy. *J Diabetes Metab*. 2015;6:500.
14. Rastinehad AR, et al. Intraductal Carcinoma of the Prostate Diagnosed by Multi-Parametric Prostate Magnetic Resonance Imaging (MRI) and MRI / Ultrasound Fusion-Guided Biopsy. *Biomedical Data Mining*. 2014;3:106.

15. Qureshi ZP, et al. (2015) The Ethical Dilemma Surrounding Prostate Specific Antigen (PSA) Screening. *J Clinic Res Bioeth.* 2015;6:206.
16. Svensson MA, et al. Combination of Multiple Markers Predicts Prostate Cancer Outcome. *J Mol Biomark Diagn.* 2015;6:213.
17. Winand FJ, et al. GDF15 and Hhepcidin as Prognostic Factors in Patients with Prostate Cancer. *J Mol Biomark Diagn.* 2014;5:199.
18. Nair VJ, et al. Bone-Seeking Targeted Radio-Nuclide Therapy (BT-RNT) in Management of Metastatic Castration-Resistant Prostate Cancer (mCRPC): Shifting from Palliation to Improving Survival. *J Nucl Med Radiat Ther.* 2015;6:202.
19. Moran BJ, et al. Comparison of Health Related Quality of Life and Other Clinical Parameters between 20 g and 18 g Needles for Permanent Low-Dose-Rate Implantation in Localized Prostate Cancer. *J Nucl Med Radiat Ther.* 2015;6:199.
20. Mahoney S, et al. The Effects of Combination Treatment Using Phenoxodiol and Docetaxel; and Phenoxodiol and Secreted Frizzled-related Protein 4 on Prostate Cancer Cell Lines. *J Carcinog Mutagen.* 2014;5:204.
21. Sonohara F, et al. STEAP4 Inactivation Correlates Poor Prognosis and might be a Possible Cause of steatotic Change in Hepatocellular Carcinoma; Detected by Triple-Combination Array Analysis. *J Carcinog Mutagen.* 2014;5:201.
22. Gyamfi AM, et al. Efficacy of Supportive Histo-morphological Features in Prostate Cancer Diagnosis. *Med Surg Urol.* 2014;3:142.
23. Pavithra V, et al. Serum Free Prostate Specific Antigen in Indian Female Breast Cancer Patients. *Medical and Health Sciences.*
24. Gopalakrishna SM, et al. In- vitro Anti-Cancer Screening of Solanum indicum, Rhus succedanea, Rheum emodi and Gardenia gummifera Medicinal Plants in Cancer Cells. *Pharmacy and pharmaceutical sciences.*
25. Akinyemi RA, et al. Effect of the Methanolic Extract of Trichosanthes cucumerina Seed (Snake Gourd/Tomatoe) on Experimentally Enlarged Prostate Gland in Adult Wistar Rats. *Medical and Health Sciences.*
26. Obi AO, et al. Low Dose Spinal Saddle Block Anesthesia (With 1.5 mg Bupivacaine) For Transrectal Prostate Biopsy- Experience with 120 Cases. *J Anesth Clin Res.* 2014;5:469.
27. Schild MH, et al. Early Outcome of Prostate Intensity Modulated Radiation Therapy (IMRT) Incorporating a Simultaneous Intra-Prostatic MRI Directed Boost. *OMICS J Radiol.* 2014.
28. McClure P, et al. A Novel NMF Guided Level-set for DWI Prostate Segmentation. *J Comput Sci Syst Biol.* 2014;7:209-216.
29. Shen C, et al. Roles of Serine Protease Inhibitor Kazal type 1 (SPINK1) in Prostate Cancer. *Med chem.* 2014;4:725.
30. Aydogan F, et al. The Effect of BMI and Visceral Fat Percentage on the Development of Bone Metastases in Prostate Cancer. *J Nucl Med Radiat Ther.* 2014;5:193.
31. Dominello MM, et al. Target Volume Heterogeneity Index; a Potentially Valuable Metric in IMRT Prostate Cancer Treatment Planning. *J Nucl Med Radiat Ther.* 2014;5:192.
32. Mackenzie J, et al. Improved Outcomes for Prostate Cancer Using Hypofractionated Radiotherapy and Dose Escalation to 55Gy. *J Nucl Med Radiat Ther.* 2014;5:188.
33. Erklcedil E, et al. Mask Phenomenon Following Robot-assisted Prostatectomy: A Rare Complication due to Trendelenburg Position. *J Anesth Clin Res.* 2014;5:431.
34. Campbell C, et al. Advanced Prostate Cancer Survivors: Implications for Palliative Care. *J Palliat Care Med.* 2013;S3:003.
35. Albert HS. Potential Carcinogens from Steroid Hormones and Diethyl Stilbesterol (DES): Chemical Relationships between Breast; Ovarian and Prostate Cancers. *J Drug Metab Toxicol.* 2014;5:170.
36. Grizzi F. On the Multiscale Nature of Human Prostate Cancer. *Pharmaceut Reg Affairs.* 2014;3:e135.
37. Kamuf J. Cancer-Free or Overall Survival Rate Following Radical Prostatectomy is not Influenced by Perioperative Pain Management. *J Anesth Clin Res.* 2014;5:422.
38. Zhou X. Prospective Application of Lipidomics in Prostate Cancer. *Biochem & Pharmacol.* 2014;3:114.
39. Talwar GP, et al. Immunological Approaches for Treatment of Advanced Stage Cancers Invariably Refractory to Drugs. *J Clin Cell Immunol.* 2014;5:247.
40. Ghaderzadeh M. An Intelligent System Based on Back Propagation Neural Network and Particle Swarm Optimization for Detection of Prostate Cancer from Benign Hyperplasia of Prostate. *J Health Med Informat.* 2014;5:158.