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News Letter

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EDITORIAL

MEN OR WOMEN. WHO IS STRONGER?

Ask anyone, he will tell you that men are the stronger sex. His reasoning is obvious: in general, men are bigger and more muscular than women. They can run faster and lift more and so on and so forth. When it comes to health, man are the weaker sex. All over the world, women live longer than men. Men die younger than women, they fall ill at a younger age and have more chronic illnesses than women. Although women see doctors more often than men; men cost our society much more for medical care beyond the age of 60. Men die at a faster rate than women. The overall mortality rate is 41% higher for men than for women. The health disparities between male and females begin during fetal life and continue from cradle to grave. About 115 males are conceived for every 100 females, but males are much more likely to die before birth. Boys are about 60% more likely to be born prematurely.

Why do men lag? Males and females are different from the very moment of conception. Some of the genes on Y chromosomes may be linked to diseases that contribute to the excess male mortality throughout life. Hormones also contribute to the gender gap. Estrogens raise HDL (Good cholesterol) levels, perhaps explaining why heart disease typically begins about 10 years later in women than men. On the other hand, testosterone may contribute to risky, aggressive behavior that causes problems for many young men. Medical problems like Diabetes, hypertension and cardiac problems are more common in men than women. Pot belly of men (abdominal obesity) is much riskier than pear shaped obesity of women interms of heart attack and stroke.

Type A behavior, work stress and hostility have all been implicated as heart disease risk factors in males. It is well known that good people are good medicine. Women have much larger and more reliable social networks than men. A study by the New England Research Institute found that 28% of women but only 9% of men report that they can rely on friends for support. Men are 2.5 times more likely than women to lack social supports. Aggression and violence are extreme forms of risky behavior which threaten the health and well being of males. Smoking, unsafe sex, alcohol and substance abuse are self-destructive habits which are more common in males. Men are fond of meat & potatoes which are bad for health. Women do household and office work including field jobs. Most men don't come close to getting the exercise they need for health!

Men have ostrich's mentality e.g. skip tests and treatments and disregard medical advice. Men make poor patients. Is it nature or nurture, the chromosome and testosterone, or daredevil role models and cultural norms of men; answers are not clear.

How can we close the gap?

Men cannot change their chromosomes or genes and few would change their hormones. Should they go girly? Should they change their behavior? Here are 10 tips to help men to bridge this gap.

(i) Avoid Tobacco in all forms
(ii) Eat well – eat whole grains, vegetables, legumes, nuts, seeds
(iii) Exercise regularly
(iv) Stay lean
(v) Alcohol in moderation
(vi) Reduce stress-enough sleep, build social ties.
(vii) Avoid risky behavior e.g. drug abuse, unsafe sex, dangerous driving
(viii) Reduce exposure to toxins, radiation
(ix) Screening and regular check-ups
(x) Seek joy and share it with others – laughter is the best medicine. Fun and optimism improve health.

Men are from Mars and Women are from Venus. But males who get their planets aligned correctly can enjoy the best if both worlds and good health are right here on Earth.

Dr. Dewan AK
Medical Director

NEW PET TRACERS IN RGCIRC : THE GLOW BEYOND FDG & DOTANOC

Cancer treatment has become individualized over the years with more disease specific options. There is thus a growing demand of more specific functional imaging to serve this purpose in the bed side. Couple of year back, Fluorodeoxyglucose (18F-FDG) Positron Emission Tomography-Computed Tomography (PET-CT) became a game changer in oncology practice. Down the years few disease specific tracers have gone through the rigorous standardization in the laboratory and are being now available in Rajiv Gandhi Cancer Institute and Research Centre (RGCIRC).

68Ga-Prostate Specific Membrane Antigen (PSMA)

Prostate specific membrane antigen (PSMA) is the specific prostate epithelial cell membrane antigen. In-vitro studies have indicated that virtually all prostate cancer cells express PSMA. PSMA is also expressed in normal prostate, benign prostatic hypertrophy, small intestine, proximal renal tubular and salivary glands cells. Fortunately PSMA expression in these cells is 100-1000 times less than prostate cancer cell. Moreover, its expression increases with higher grade and hormone resistance in prostate cancer cell. Due to non secreting nature and internalization after ligand binding endocytosis (via clathrin coated pits), PSMA has received worthy attention for theronostics.

Radiolablled anti PSMA antibody (Capromab pendetide, ProstaScint) is Food and Drug Administration (FDA) approved for detection of soft tissue metastasis and recurrence in prostate cancer patients. Due to low accuracy and technical challenge it is not utilized in most places. Small molecules which can be labeled with better radionuclide and clear fast is the current necessity. One novel promising PSMA specific pharmacophore is Glutamate-Urea-Lysine. It binds with extracellular domain of PSMA, followed by internalization. Experience with Glu-NH-CO-NH-Lys-(Axe)-[68Ga(HBED-CC)] (68Ga-PSMA) is promising with better and early detectability. Ali Afshar Oromich, et al. has reported 100% detectability (at least one lesion) above 2.2 ng/ml PSA and 60% below it. Besides recurrence evaluation, 68Ga-PSMA PET can be utilized in advanced prostate cancer for nodal and distant metastasis (Fig. 1). Role in guiding biopsy and radiotherapy planning is in infancy stage.

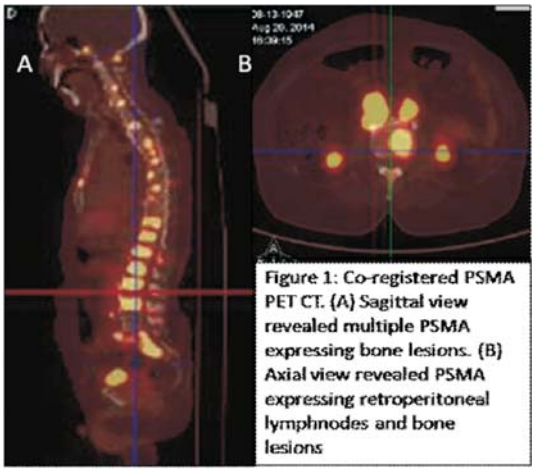

Figure 1: Co-registered PSMA PET CT. (A) Sagittal view revealed multiple PSMA expressing bone lesions. (B) Axial view revealed PSMA expressing retroperitoneal lymphnodes and bone lesions

16-α-(18F)-Fluoro-17-β-Estradiol (FES)

In RGCIRC, 68Ga-PSMA PET-CT is being done on regular basis. Results are promising for both staging and biochemical PSA recurrence.

Estrogens are involved in growth of both normal and cancerous breast tissues. Its activity is mediated by estrogen receptor (ER). ER-positivity in breast cancer cells has impact on treatment and patient outcome. Measurement of ER expression by biopsy at the time of primary diagnosis of breast cancer is standard care however it is known that ER expression may be heterogeneous and different in metastatic sites in approx. 20% of patients. In vitro data is available supporting good correlation between FES uptake and ER expression. Molecular radionuclide imaging with FES PET can potentially be use to see overall ER expression in all breast cancer sites and to predict response to therapy (Fig. 2 & 3). It can also be utilized in difficult cases, like treated case of dual malignancy with non assessable metastasis (Fig. 4). Good FES concentration in the lesion indicated good ER expression and can be treated as breast cancer metastasis only.

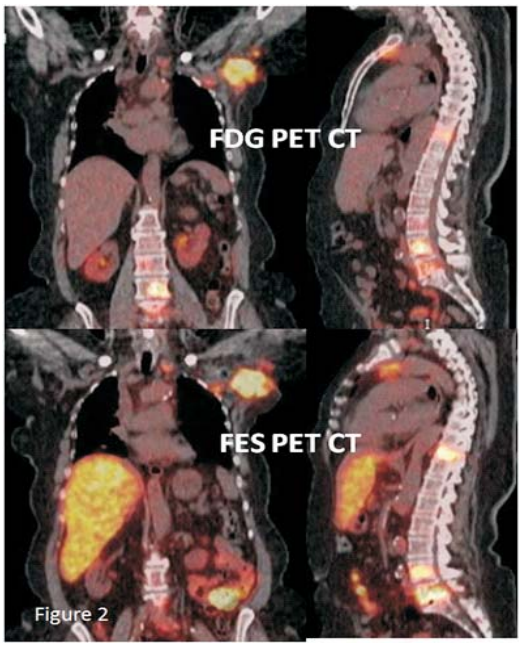

Figure 2: Recurrent ER positive breast carcinoma. FDG and FES Co-registered PET-CT: Coronal and Sagittal images revealed good FES uptake in all FDG positive metastatic sites (True positive).

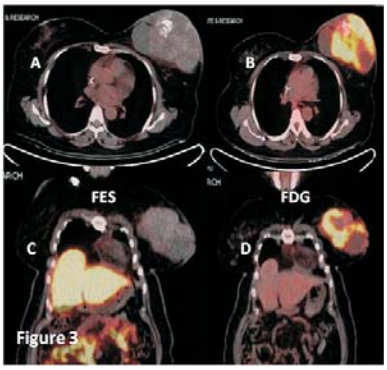

Figure 3: Left breast carcinoma with ER negative on IHC. FES Co-registered PET-CT: image A & C. Axial and Coronal view revealed no FES uptake in primary breast mass (True negative). FDG Co-registered PET-CT: image B & D. Axial and Coronal view revealed hypermetabolic left breast mass with necrosis.

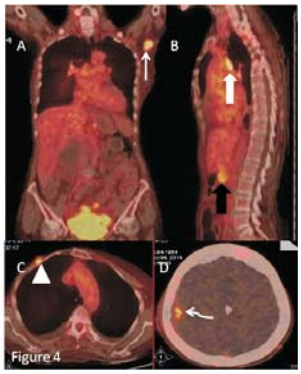

Figure 4: FES Co-registered PET-CT: A. Coronal view revealed ER expressing axillary lymph node (white arrow). B. Sagittal view revealed ER expressing mediastinal lymph node (white block arrow) and periamupullary deposit (black block arrow). C. Axial view revealed ER expressing chest wall deposit (white arrow head). D. Axial view revealed ER expressing brain deposit (curved white arrow).

18F-Fluoro-Ethyle-Tyrosine (FET)

Primary brain tumors constitute 1-2% of adult cancer. Often it is difficult to qualify a ring enhancing lesion on MRI as tumor (glioma, metastasis or other primary brain tumor) or non tumor (abscess, parasite, demylination, infarct or old hematoma) origin. Other issue of tumor recurrence in post surgery or radiotherapy setting is very troublesome. 18F-fluoro-ethyle-tyrosine (18F-FET) is an artificial amino acid and taken up by up regulated tumor cells. 18F-FET scan has proved its efficacy in brain tumor recurrence after surgery and radiotherapy (Fig. 3). 18F-FET shows lower uptake in inflammatory cells than 11C-Methionin or 18F-FDG, so it can be utilized in this diagnostic dilemma of tumor Vs inflammation. In other potential uses, it can be used for directing biopsy and radio-surgery.

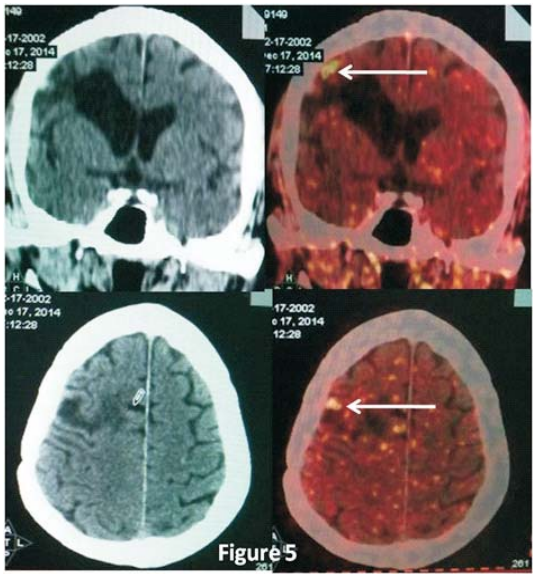

Figure 5: CT and Co-registered FET PET-CT axial and coronal images of brain: FET positive small focus (white arrow) along superior surface of post operative cavity in right fronto-parietal region in grade 2 oligodendroglioma patient.

Dr. Manoj Gupta/Dr. P S Choudhury
(Department of Nuclear Medicine)

CANCER SCREENING CAMP AT SKH METALS, MANESAR

In its continuous effort to increase Cancer Awareness, RGCIRC organized a Cancer Screening Camp and a small myth busting exercise for employees of SKH Metals, Manesar, Haryana. Around 75 employees were screened for cancer by Dr. J. G. Sharma, HOD – Preventive Oncology Deptt., RGCIRC.

WELCOME TO RGCIRC FAMILY

Dr. Shalini Mishra, Consultant – Pediatric Surgical Oncology trained at Christian Medical College, Vellore. She has a vast experience in the surgical management of infants and children with solid tumors. She also has more than 8 years pediatric surgical teaching experience at Maulana Azad Medical College, Delhi.

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