Male Urinary Incontinence

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This article will describe the diagnosis and management of different causes of male urinary incontinence, with a focus on the most common type of male incontinence, post-prostatectomy incontinence.

Urinary incontinence can be broadly divided into **stress urinary incontinence (SUI)**, **urge incontinence (UI)** and overflow incontinence. SUI is loss of urine during physical exertion that increases intra-abdominal pressure, overcoming urethral sphincteric resistance. UI is associated with an overactive bladder, in which an urge to void or involuntary bladder contraction occurs at a volume less than capacity. UI is simply involuntary loss of urine associated with this urge to urinate (sensory UI) or involuntary bladder contraction (motor UI). Motor UI is seen in detrusor instability (caused by bladder outlet obstruction, bladder mucosal inflammation or tumor, or pelvic nerve injury) or detrusor hyperreflexia (caused by an upper motor neuron lesion, such as a brain lesion or spinal cord injury). Sensory UI is associated with increased bladder sensation but no actual detrusor contraction and can be seen in all types of overactive bladder, especially intersititial cystitis. Overflow incontinence is associated with urinary retention secondary to bladder outlet obstruction or detrusor failure and occurs when bladder capacity is exceeded. Mixed incontinence, usually a combination of SUI and UI is a common clinical finding.

Most often, significant male urinary incontinence is iatrogenic SUI directly related to surgery of the prostate and is known as **post-prostatectomy incontinence (PPI)**. By far, the most common surgical procedure associated with PPI is radical removal of a cancerous prostate (**radical retropubic prostatectomy or RRP**). Much less commonly, PPI can occur following surgical relief of prostatic obstruction, such as **transurethral resection of the prostate (TURP)** or open prostatectomy. Non-iatrogenic causes of male incontinence include UI or overflow incontinence as described above. In elderly males, the most common causes of UI include detrusor instability secondary to chronic bladder outlet obstruction, and less commonly detrusor hyperreflexia secondary to stroke or Parkinsonís disease.

Post-Prostatectomy Incontinence

According to physician-reported data, 0.3-87% of men have significant urinary incontinence after RRP.[1-8] Technical advances in the surgery are thought to have dramatically decreased severe incontinence,[3,4] but patient-reported data continue to show high rates of PPI (63%).[9] One per cent of men who have TURP have significant postoperative incontinence.[10]

Common causes of PPI include urethral sphincteric insufficiency, preexisting bladder dysfunction, anastomotic urethral stricture or **bladder neck contracture (BNC)** or a combination of these factors.[11-14,18]

Urethral Sphincter Insufficiency

The most common cause of incontinence from RRP and TURP is direct damage to the male urinary sphincter. The male urinary sphincter has two basic components: voluntary and involuntary. The voluntary component is located predominantly at the urogenital diaphragm but can extend in a limited fashion proximally to the bladder neck. It consists of striated, somatic muscle fibers that contract volitionally for short durations but not continuously. Although it does not contribute significantly to overall urinary continence, the voluntary component may be damaged during RRP and TURP, contributing to incontinence.

The involuntary component extends from the bladder neck distally to the urogenital diaphragm and consists predominantly of autonomic smooth muscle fibers, although some striated muscle is present. This component provides almost all of passive, involuntary, continous urinary continence and is commonly damaged during RRP, resulting in urethral sphincter insufficiency. Possible contributory factors include: 1) excessive operative time or bleeding resulting in ischemia and scarring of the urethral anastomotic site, 2) excessive urinary leakage from the urethral anastomosis resulting in scar, 3) BNC requiring dilation or incision followed by sphincteric insufficiency. The end-result of this sphincteric damage is a shortened functional urethral length with scarred mucosa lacking elasticity and coaptation. Stress incontinence associated with urethral sphincter insufficiency is also known as type III stress incontinence or **intrinsic sphincter deficiency (ISD)**.

Certain risk factors may predispose to PPI following RRP. Younger men undergoing RRP have been reported to achieve continence sooner than older men,[15] possibly because of age-related muscle atrophy and the increased incidence of detrusor instabiity with normal aging. Nerve-sparing RRP has been observed to be associated with higher continence rates that non-nerve sparing RRP.[6] Salvage radical prostatectomy after radiation therapy has higher incontinence rates, which may be attributed to radiation damage to the bladder and urethra as well as the technical difficulty of this operation.

Preexisting neurologic disease is a risk factor for PPI. The risk of PPI after TURP in patients with Parkinson's disease has been reported as high as 20%,[16] because of the high rate of detrusor hyperreflexia and abnormalities of external sphincter control in this disease.

Diagnosis of Urethral Sphincter Insufficiency

Urodynamic (UDs) will rule out detrusor instability as a cause of UI,

detrusor failure as a cause of overflow incontinence and bladder outlet obstruction as a cause of both UI and overflow incontinence. Valsalva leak point pressures is a urodynamic measurement of abdominal or valsalva pressures required to cause SUI. Valsalva leak point pressure <50 cm H2O confirms ISD, although ISD can be demonstrated simply by eliciting severe SUI by coughing or valsalva maneuvers. Fluoroscopy (videourodynamics or voiding cystourethrography) can identify a BNC and evidence of preexisting bladder dysfunction (bladder trabeculation or vesicoureteral reflux).

Cystoscopy may reveal signs of urethral sphincter insufficiency, including a very short posterior urethra with scarred or blanched urethral mucosa. A fixed, damaged external sphincter that opens and closes incompletely may be seen following TURP or open prostatectomy. BNC should be ruled out, especially if an artificial urinary sphincter is contemplated (see below). Severely scarred urethral mucosa would argue against the use of transurethral bulking agents as a treatment for ISD because of the very low success rate. Therefore, cystocopy and UDs with or without fluoroscopy are very helpful in the diagnosis and management of PPI.

Treatment of Urethral Sphincter Insufficiency

Conservative Options

PPI can result in tremendous morbidity, including limitation of usual activities, skin rashes, odor, need for anti-incontinence devices and corrective surgery. Usually, non-surgical management is recommended for at least one year because of the possibility for spontaneous improvement. After this, further improvement is unlikely. Prior to surgery, many men try Kegel exercises, empiric medications, bulky pads, external catheters and even penile clamps. Kegel exercises may improve mild PPI but are of minimal benefit in severe SUI because only the voluntary component of the urinary sphincter is exercised.

Bulky pads are inconvenient, uncomfortable, expensive, may carry an odor and facilitate rashes. External catheters can cause frequent **urinary tract infections** (**UTIs**), allergic reactions, rash and soft tissue injury of the penis. In addition , they may not stay on the penis, especially if uncircumcized. Penile clamps are uncomfortable and can cause ischemic injury to the penile shaft and urethra.If urodynamics reveal motor UI, anticholinergics may be helpful, but do have common side-effects including dry mouth and constipation. Patients with closed-angle glaucoma may have blurry vision, which is reversible. Anticholinergics are unlikely to improve sensory UI.

The most effective treatment of severe PPI is an artificial urinary sphincter.

Treatment of Urethral Sphincter Insufficiency: Surgical Options

Conservative measures often are of minimal benefit, and surgical intervention is the next step. Surgery for PPI include transurethral injection of bulking agents, artificial urinary sphincter and the male sling. Transurethral bulking agents include bovine collagen, autologous fat, teflon and carbon coated beads. Collagen is injected at the bladder neck in a submucosal location, usually at the 5 and 7:00 or 4 and 8:00 positions. This results in coaptation of the urethral mucosa. Possible complications include urinary retention, infection, bleeding and allergic reaction to collagen. Urinary retention is almost always temporary and commonly resolves after overnight catheter drainage. A test dose of collagen is injected in the forearm prior to collagen injection to identify allergy to collagen, and the injection site is observed for at least one month. Allergic reactions occur < 1%. Collagen injection for PPI have very poor results, most likely because the injection is usually into scarred mucosa and submucosa. It is not commonly done for PPI after TURP.

Fat as a bulking agent is even less successful than collagen. Teflon is not widely used because of the risk of migration of teflon to distant organs. Carbon-coated beads do not require a test dose and have been reported by its manufacturers to remain in location in a durable fashion. It is unclear whether clinical experience supports this finding. The superficial injection of carbon beads may be even more difficult in a scarred urethra than collagen. A recent report described migration of carbon beads to local and distant lymph nodes,[17] which raises reservations about the use of carbon beads similar to those regarding teflon.

The most effective treatment of severe PPI is an **artificial urinary sphincter** (**AUS**). The AUS, made of silicone, consists of a cuff that wraps around the bulbar urethra, a regulating balloon that is placed in a prevesical location, and a scrotal pump. The device is fluid filled and works hydraulically: the pressure of the regulating balloon provides the occlusive force of the cuff. The intraabdominal location of the regulating balloon allows transmission of increased intraabdominal pressure during abdominal straining or stress maneuvers to the cuff to prevent leakage. The cuff is emptied by squeezing the scrotal pump, which transfers fluid to the regulating balloon, allowing voiding. Resistors in the AUS device delay cuff refill for 2-3 minutes, allowing time for voiding.

Preoperative UDs will identify impaired bladder contractility, which may require longer voiding times. The cuff can be emptied more than once. Men with completely acontractile bladders with large-volume urinary retention are poor candidates for AUS placement because of the risk of UTIs, which may infect the AUS. Catheterization is possible with an AUS, especially in children with neurogenic bladder, but this is not an optimal situation and may increase the risk of UTI and urethral trauma, both of which may lead to infection and erosion of the AUS.

UDs will also identify patients with impaired bladder compliance and high-pressure involuntary bladder.

Children with myelodysplasia with abnormal bladder parameters in whom AUS was placed have developed silent upper tract deterioration. Hostile bladder parameters such as impaired compliance or high-pressure contractions will worsen in the face of an inflated cuff, resulting in silent renal deterioration as well as urge incontinence. Therefore, an AUS should not be placed until these hostile parameters are adequately treated, which in itself may reduce incontinence by treating the urge component.

Preoperative cystoscopy will also identify a BNC. This should be treated prior to the AUS and observed for a minimum of 6 months for a recurrent contracture. The urethra around which the cuff is placed may be atrophic or narrowed secondary to ischemic damage during cuff placement. This increases the risk of damage at this site with subsequent passage of a large caliber instrument or urethral dilator. Therefore, a BNC that recurs after AUS placement may be difficult to incise or dilate without an increased risk of urethral injury and subsequent cuff infection or erosion.

Adequate parenteral antibiotics should be given before and after AUS placement. Strict sterile technique should be observed with minimal traffic in and out or the operating room to prevent contamination. Meticulous dissection around the urethra is necessary to prevent ischemic damage to the urethra or urethral injury. If urethral injury occurs, the urethra should be repaired primarily and the AUS should not be placed because of the high risk of infection associated with urinary extravasation. After AUS placement, the cuff should be deactivated in an open position for at least 6 weeks to allow urethral healing.

Three possible problems can occur after AUS placement: infection, erosion and malfunction. In an infection, the patient may have swelling, tenderness or erythema of a component or the entire device. A prolonged course of antibiotics may treat the infection. More commonly, a portion or the entire device is removed, followed by antibiotics and subesequent AUS replacement several months later.

Signs and symptoms of an erosion include recurrent incontinence, AUS infection, gross hematuria, urethral discharge, perineal pain and edema. Early erosion usually results from urethral trauma at the time of insertion. Delayed erosions can occur because of infection or catheter trauma, especially when a catheter is inserted and kept in without deactivation of the cuff. Erosion can be confirmed by urethroscopy or retrograde urethrography and is treated by removal of the entire AUS and replacement after the erosion heals.

Mechanical malfunction usually occurs because of lack of attention to detail during insertion and usually presents as recurrent incontinence or failure to activate the cuff. Recurrent incontinence is caused by leaks in any component due to injury to the device during insertion or by detachment of tubing due to improper assembly during surgery. This is treated by replacement of the leaking component or by reattachment of the detached tubing. Cuff atrophy and improper positioning of the cuff can also cause incontinence. Cuff atrophy occurs because of ischemic trauma to the cuff during insertion or by pressure atrophy of the urethra from improper sizing. This results in a loose cuff. This is treated by cuff resizing, replacing in a different location, or adding a second cuff, preferably more proximally. If the initial cuff is not placed proximally enough, the patient can sit on the cuff, causing both perineal discomfort and deactivation of the cuff.

Failure to prevent blood or debris from entering the tubing at time of surgery may result in inability to activate the cuff by squeezing the pump. Using improper filling fluid during insertion may also result in clogging of the tubing. This also requires removal or revision of the AUS.

Careful attention to detail, meticulous surgical technique and testing the device for proper function, leaks, secure tubing connections and debris or blood in the tubing at the time of surgery can reduce the likelihood of later revision. However, despite all this, most clinicians are aware of the limitations of this device. The male sling has been proposed as a surgical alternative to the AUS. A strip of cadaveric, autologous or synthetic material is placed underneath the bulbar urethra and tied over the rectus fascia or secured to the ischial rami with bone anchors after enough tension is placed upward to create urethral occlusion pressures as high as 90 cm H2O.[18,19]

Longterm results are not available. There are several additional concerns about this procedure. Unlike the female sling in which as little tension as possible is placed and the mechanism of continence is increased passive support to the bladder neck and proximal urethra, continence appears to be achieved by urethral occlusion. This may result in pathologic obstruction, bladder decompensation, upper tract damage, future urgency and UI. Adverse effects of cadaveric and synthetic materials and bone anchors have yet to be defined. Long-term follow up will hopefully address these concerns.

Conclusion

Male urinary incontinence is multifactorial but is most often iatrogenic following prostatectomy. Careful diagnostic evaluation will allow the clinician to determine the most effective treatment. If a surgical option is selected, meticulous surgical technique and attention to detail will optimize results. However, treatment for male incontinence can be suboptimal and frustrating. More effective treatment options are eagerly awaited.

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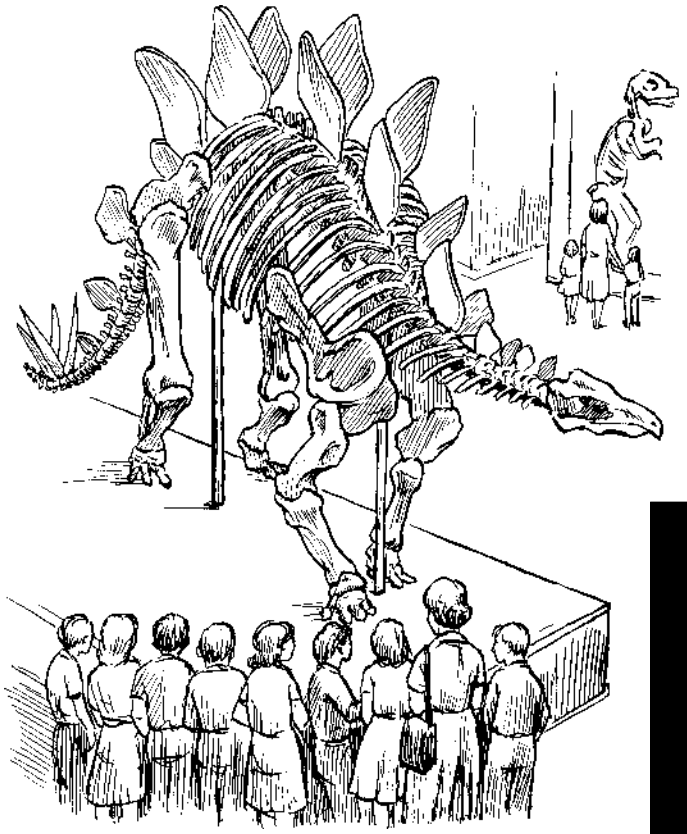

Osteoporosis and the Aging Male

Barry Stein, MD, and Seetharaman Ashok, MD

Osteoporosis in the male is a newly appreciated problem. Long associated with the aging woman, osteoporosis is now known to affect more than 2,000,000 American males over the age of 50, with another pool of 3,100,000 men who are at risk for developing it.[1-5] About one out of every 8 men over the age of 50 will at some point have an osteoporosis related fracture.[6-7] Every year about 100,000 men will suffer an osteoporosis related hip fracture; one third of them will die within the year. In addition, tens of thousands of men will have a fracture of the wrist, spine or rib. Physicians who treat men need to be increasingly sensitive to this problem. Thirty-six percent of the osteoporosis in men is due to low androgen levels, which can occur due to hypogonadism, either congenital, as part of the aging process, or due to acute androgen deprivation, such as the treatment of advanced carcinoma of the prostate.[8] In the latter case, given the usually advanced age of the patient, the acute loss of androgens is occurring in addition to the bone density loss of aging.[7] This can lead to a higher incidence of osteoporosis and its complications. Considering that hormone therapy is initiated earlier today due to the PSA monitoring of prostate cancer patients, a longer duration of androgen deprivation therapy can be anticipated, and the incidence of osteoporosis may yet increase.

At this time, there is no accurate measurement of overall bone strength, so we use the measurement of bone mineral density as its proxy. Bone mineral density accounts for 70-85% of bone strength, and the measurement of bone mineral density correlates with the load-bearing capacity of the skeleton.[5] **DEXA (Dual Energy X-Ray Absorption)** scans, which measure bone density, are our best gauge of bone strength. This technology utilizes X-rays of 2 different energy levels so as to distinguish between bone and the surrounding soft tissue structures. A two-dimensional image is produced, which can be used to measure bone density.[3] A number of prospective studies have been performed, most, but not all, in women, which show that DEXA scans can predict the future risk of fracture. Each decrease in bone mineral density of 1 **standard deviation (SD)** in the hip translates into a 1.5-3.0 fold increased risk of fracture.[5]

In reading DEXA scans, the measurement of the patient's bone mineral density is compared to one of two cohorts of men, whose results are already in the computer's data bank. The z-score compares the patient's DEXA results to an age and ideally race-matched cohort of men, and will compare that man to this cohort by both percent of normal and by SD readings. The cohort is based on the Caucasian male, because insufficient data exist at this time relative to other races. The t-score compares the patient's bone mineral density to young healthy men between the ages of 30-45. This represents the optimum age for peak bone mineral density, and the t-score is the more important measurement for the determination of bone density loss. Thus all results presented in this article will focus on the t-score for determination of the bone density status. In interpreting DEXA scans, a bone mineral density between 1 and 2.5 SD less than the cohort is read as osteopenia, while a bone mineral density loss of >2.5 SD is defined as osteoporosis.[2] When a DEXA scan reading is >2.5 SD less than the cohort of young healthy men, and the patient has a fracture, this is termed severe osteoporosis.

The Scope of the Problem in Prostate Cancer

When three of the author's patients suffered hip fractures within the space of a year, we began to evaluate our patients who were being treated with **androgen deprivation therapy (ADT)** with DEXA scans. Of 75 patients studied to date, 70 were on leuprolide therapy, and 5 had undergone bilateral orchiectomy.[9] All patients underwent an in-office screening DEXA scan utilizing the fourth digit of the non-dominant hand. When this proved abnormal, a full table body scan of the hip and spine was performed for comparison. The patients' ages ranged from 46-98, with a mean of 76.4. The patients' duration of ADT ranged from just beginning treatment, to 13 years on therapy, with an average of 3.35 years. Of the entire series, only 34 (45.4%) had a normal bone mineral density. Of the 41 patients with abnormal DEXA scans, 25 (61%) had osteopenia, while 16 (39% or 21.3% of the entire group of 75 men) had osteoporosis.

When we examined the men by age, we found that those over age 70 had a greater risk of loss of bone mineral density than men younger than 70. Overall, 68.4% of the men younger than 70 years of age had a normal bone mineral density, compared to only 37.5% of the men over 70. Of the 25 men with osteopenia, 19 (76%) were older than 70, while all of the 16 men with osteoporosis were over 70. Duration of therapy also appeared to be important. We examined the 34 men who were on treatment for less than 2 years and compared their DEXA results to the men on ADT for more than 2 years. Of the less than 2 year group, 58.8% had a normal DEXA scan, while only 34% of the men on treatment for more than 2 years had a normal DEXA scan. We further examined the 34 men on therapy for less than 2 years, and found that those with normal bone mineral density, had an average age of 72.4 years, while those with abnormal DEXA scans, had an average age of 81.3. We examined the men by the number of years on therapy and as the duration of therapy became longer, the bone mineral density decreased. From our own work we conclude that the older the man is and the longer he is on ADT, the likelier he is to experi-

ence a loss in bone mineral density. Other studies have confirmed our results.

Smith and associates initially reported on 41 men with locally advanced prostate cancer, without metastatic disease, and who had not yet undergone ADT.[10] These men then underwent DEXA scan of the hip and spine. The mean age in this series was 68. Of this group, 66% had normal bone densities, while 29% had osteopenia, and 5% had osteoporosis. This compares very favorably to our findings in men on therapy for less than 2 years.

Stoch et al studied 3 groups of men: group 1 consisted of controls solicited via a newpaper ad, group 2 were men with cancer of the prostate, but not on ADT, and group 3 were men on ADT for at least 6 months, with a mean of 41 months of therapy.[11] The men underwent evaluation of bone mineral density by a variety of techniques including finger, spine and hip DEXA scans. They found that the normal rate of bone loss due to aging is 0.5-1.0% per year, but that LHRH analogue therapy was associated with more than a decade increase in this loss. They also reported an incidence of osteoporotic fractures similar to other groups.

Daniell performed 2 studies on osteoporosis and ADT. In the first paper, he reviewed the records of 235 men with prostate cancer, and from this culled the names of 17 men who had undergone orchiectomy between 1983-1990, and were still alive in 1995.[12] He then performed DEXA scans of the femoral neck and compared the results to 23 controls. He found 10 osteoporotic fractures in the larger group, 8 of which were found in the 17 orchiectomy patients. Of the 16 men who survived for >60 months, 6 had osteoporotic fractures and reduced bone mineral density on DEXA scans. The incidence continued to increase over time. In a follow up study, Daniell evaluated 26 men prior to orchiectomy or LHRH analogue therapy and followed them for 6-42 months, comparing them to 12 controls.[13] They found that bone mineral density in the ADT patients fell about 4% per year for years 1-2, and 2% per year every year thereafter. The loss continued at a pace of 1.4-2.6% per year from years 3-8. Both orchiectomy and LHRH analogue therapy were likely to cause this loss.

Eriksson et al compared 2 groups of men on hormone therapy for prostate cancer: group 1 (11 pts) were treated with orchiectomy alone, while group 2 (16 pts) underwent orchiectomy plus estrogen therapy (IM or PO).[1] They then measured BMD of the femoral neck, trochanter and ward's triangle. There was a decrease in BMD in the orchiectomy only patients, not seen in the orchiectomy+estrogen patients. Statistical significance was achieved only in the forearm.

Given the high incidence of bone mineral density loss in men with prostate cancer undergoing hormone therapy, we recommend baseline evaluation with a DEXA scan.

Recommended Evaluation and Treatment

Given the high incidence of bone mineral density loss in men with prostate cancer undergoing hormone therapy, we recommend baseline evaluation with a DEXA scan. If the baseline scan is normal, no further evaluation is necessary at that time, and a follow up scan should be performed in 1-2 years. If the scan is abnormal, then treatment should be discussed with the patient, explaining the risks and benefits of treatment.

Men with abnormal scans should first be provided with counseling on nutrition and lifestyle issues. They should be instructed to eat a balanced, healthy diet, especially high in calcium content. They should stop smoking, moderate alcohol consumption, and begin a regimen of physical exercise. Exposure to sunlight is also suggested, providing that they do not have skin cancer.

Our initial medical treatment for osteopenia is bisphosphonate therapy at osteopenic doses, Vitamin D and Calcium. Currently we use Alendronate 35mg once a week; and Vitamin D 400IU or more, and Calcium Carbonate or Citrate 1000mg daily. The Vitamin D and Calcium are often available as a combination marketed specifically for osteoporosis. The initial treatment for osteoporosis is identical except for increases in the dose of bisphosphonate, in this case to Alendronate 70mg weekly.

According to the NIH consensus conference 2000, both alendronate and risendronate are bisphosphonates, and have been shown to reduce the risk of vertebral fractures by 30-50% in randomized, clinical trials, although the majority of such trials involve female osteoporosis.[3] Orwoll et al reported on a randomized, double blind trial in men with osteoporosis, evaluating alendronate 10mg daily (n=146) vs placebo (n=95), with all men receiving calcium carbonate 500mg and vitamin D 400IU daily.[8] All men underwent DEXA scans of the lumbar spine, hip and total body up to 24 months. In the placebo group bone density remained unchanged, while in the alendronate arm the bone density increased, particularly in the lumbar spine. These changes were not related to testosterone or estradiol levels. The incidence of vertebral fractures in the placebo group was 7.1%, while in the alendronate group it was only 0.8% (p=.02). Frediani and associates also performed a placebo controlled study of alendronate 10mg daily (n=30) vs placebo (n=30), with all men receiving calcium 500mg daily. These men were followed up to 24 months with DEXA scans of the hip and spine. The men on placebo in this study had a loss of bone mineral density of 2.8-3.6%, while those on alendronate had a bone mineral density gain of 3.4-6.3%.

Smith et al recently published a new series of 43 men treated either with leuprolide alone (22) or leuprolide plus

IV pamidronate (a bisphosphonate) in an attempt to prevent bone loss.[14] All the men were placed on Vitamin D and Calcium supplements. The patients were evaluated up to 48 weeks with repeat DEXA scans. The authors found that in the patients on leuprolide and supplemental therapy alone the bone mineral density decreased by 3.3% in the spine, 2.1% in the trocanter and 1.8% in the total hip. By contrast, those patients whose treatment included bisphosphonate therapy experienced no change in their bone mineral density.

Conclusion

Osteoporosis is a major health threat to the aging male population, but especially to men with prostate cancer on ADT. This can be diagnosed easily with DEXA scans, and can be successfully treated. The men at highest risk are those >70 years of age, and on ADT for >2 years. Successful treatment can be undertaken with bisphosphonates, vitamin D and calcium. This diagnosis and treatment in men is just as important as diagnosing women with osteoporosis.

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ACP ASIM

THE CREATIVE CLINICIAN CASE

"The practice of medicine is an art, not a trade; a calling, not a business ..." – WILLIAM OSLER, Aequanimitas

Borderland

James E. McLennan, MD

Ms. J.D., a 29 year old computer analyst, reported mental fogginess and headache to her primary physician around Christmas 1986. When, in May 1987, I saw her in my office, the studies revealed a large bifrontal tumor. I hoped it would be meningioma. The craniotomy, however, disclosed an extensive, poorly encapsulated mass that was removed mostly by suction. Frozen section pathology showed malignant glioma. The tumor passed through the falx and involved both frontal poles; it was grossly, completely, resected including the falx and anterior sagital sinus. The final diagnosis was Grade III malignant glioma, signaling a poor prognosis.

Judi had a stable post operative course. Because of her youth and the total gross resection of the tumor, the oncology team and I discussed with her and her family very aggressive treatment.

A few weeks after discharge, with radiotherapy already underway, Judi and her mother attended a Catholic Mass during which they received communion. When the other parishioners left, they went to the side altar where they prayed before a visiting statue of Our Lady of Fatima that was on special exhibit. A priest appeared and, without saying anything, opened the tabernacle, the repository for Communion provisions, removed a single host and placed it on Judi's tongue. At that moment, she later told her mother, the Lord revealed to her that she would be healed.

Over the next several months, Judi indicated that her thinking seemed slightly fuzzy but, otherwise, she was functioning well in her daily life. Her gait and balance were good. She completed twenty-five radiation treatments. Since she responded so well to the treatments, she received an additional coned-down, radiation boost to both frontal lobes, for a cumulative tumor dose of 5980 cGy. On her final day of radiotherapy, her treating physician noted that she was "animated and alert" and had a normal examination.

Without telling anyone, Judi attended a Healing Mass where, her family learned later, the Blessed Mother appeared to her on the altar.

After consulting with the oncologists, we treated Judi with a protocol of bone marrow harvesting and storage, followed by intensive chemotherapy and replacement of the marrow. To control the brain edema, she required prolonged significant doses of Decadron which caused Cushingoid side-effects of hirsutism and obesity. She experienced muscle weakness and visual changes. She began having severe hip and shoulder pain from steroid-induced avascular necrosis of the femoral and humeral heads. Judi suffered tremendously during this period but remained stoic. Throughout her treatment, she never asked me or her other doctors, "How long do I have?" (Nor did her family).

By Spring 1989, we succeeded in weaning her off Decadron. She stabilized with only cystic changes appearing on her brain scans. She described her memory and function as normal. Cranioplasty was performed. She resumed her active life style, retraining and working as a medical secretary. She even skied although she had difficulty getting up when she fell.

Followup reports from Judi's radiotherapist and oncologist expressed their increasing amazement at her recovery, as evidenced in this note from 1990: "for all intents and purposes, she has to be considered cured of her primary tumor."

In 1992, Judi consulted an orthopedist hoping for relief from her continual hip pain and difficulty in walking. Considering the expected prognosis for her disease, the orthopedist recommended conservative treatment. Judi, however, insisted on surgery and underwent bilateral total hip replacement. In 1995, her radiotherapist reported that her only problem was pain in the shoulders.

In the summer of 2001, Judi experienced new mental status changes. The scans eventually revealed massive recurrent tumor, a biopsy was performed, and palliative care was instituted.

During her last admission, Judi told her family about the Divine message she received while attending Mass with her mother, so many years ago. She told them about the Healing Mass where the Blessed Mother appeared to her. As she recalled the experience that she had kept secret until now, her eyes filled with tears: "The Blessed Mother was so lovely and She had a beautiful fragrance." (Judi's frontal fossa surgery and radiation had interrupted her sense of smell.)

In her final days, the family heard her arguing, "I don't know why he can't agree with me."

They asked her whom she was talking about.

She replied, "God, but I don't agree with Him."

Her family interpreted this as an argument with God

to cure her, as, she believed, He had before.

Judi's sister, who practiced a charismatic form of Catholicism, also prayed for a miracle. With her Bible in front of her, she received the instruction to randomly open it. On that page would be God's message. The Bible fell open to Isaiah, Chapter 38, verses 1-5, the story of King Hezekiah who was "sick unto death."

> And Isaiah, the prophet . . .came unto [Hezekiah],
> Thus saith the Lord: Set thine house in order:
> for thou shalt die, and not live.
> Then Hezekiah turned his face toward
> the wall, and prayed unto the Lord,
> And said, Remember now, O Lord, I beseech
> thee, how I have walked before thee in truth
> and with a perfect heart, and have done that
> which is good in thy sight. And Hezekiah wept sore.
> Then came the word of the Lord to Isaiah,
> saying,
> Go, and say to Hezekiah, Thus saith the
> Lord . . . I have heard thy prayer, I have seen
> thy tears: behold I will add unto thy days
> fifteen years.*
>
> * King James Version

Her sister realized Judi's fifteen years were up. Judi died soon thereafter.

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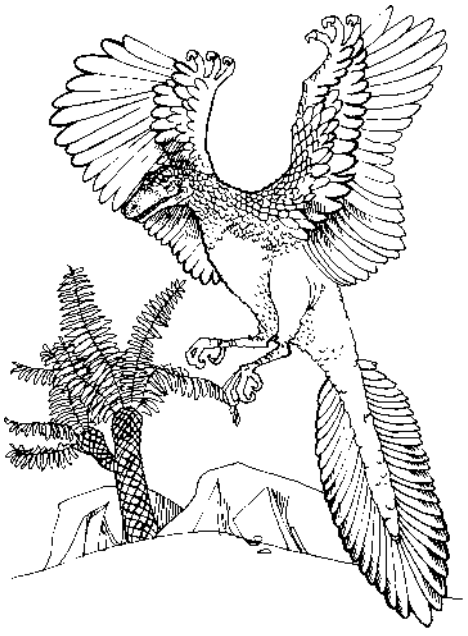

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