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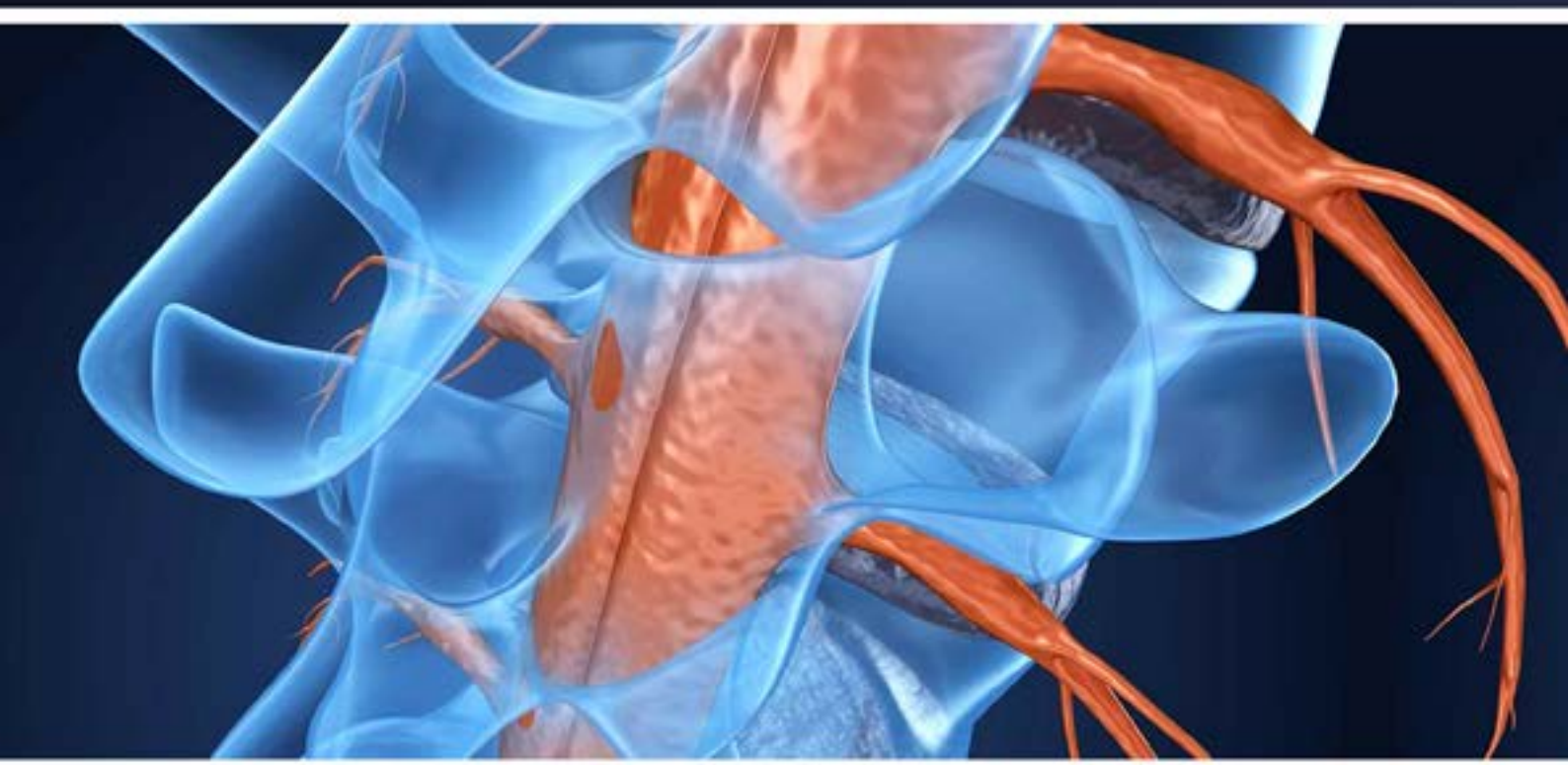

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FROM THE EDITORS DESK...

Here's the second issue of spinal cord rehabilitation.

Dr Ann is our invited author is discussing sexual rehabilitation after SCI. We welcome our newest authors Dr. Nitha and Bineesh who write about what patients have taught them. Dr. Unni contributes by telling us what he has done to improve spinal cord rehabilitation. Quizmaster Dr. Bineesh's second quiz is here.

Our next release will be themed Auditory, Vestibular, and Visual rehabilitation. Until then, enjoy this issue and stay safe.

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Contents

(The included articles are the responsibility of each author)

Sexual Function And Fertility In Men After Spinal Cord Injury

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Introduction

Sexuality is an important part of life. Sexual and reproductive health is important for quality of life and has a significant influence on patient satisfaction. Spinal cord injury (SCI) results in alterations in physiological sexual responses and has negative impacts on sexual interest, satisfaction and can affect fertility in couples. Spinal cord pathology occurs from trauma, deformity, and degenerative disease processes may be leads to damage sexual function and reproductive system. SCIs are usually located in discrete areas of the neuraxis and based upon which descending spinal motor and autonomic pathways are disrupted, it can result in predictable impacts on sexual function[1-2].

Spinal cord injury (SCI) has been well studied in both genders. The type of sexual dysfunction the patient will ultimately experience depends in large part upon the spinal cord level and the degree of completeness of the injury [3]. Multiple studies have shown that the frequency of sexual activity and the level of sexual satisfaction decreases in both men and women after sustaining a SCI [4]. As men are more commonly affected than women with regard to SCI[5] my article focuses on sexual rehabilitation and fertility in males after a spinal cord injury.

Sexual and reproductive health in Males with SCI

SCI can be a risk factor for abnormal lower levels of testosterone [6,7]. In one study, 46% of men with SCI were identified to have low serum total testosterone concentrations (total testosterone <11.3 nmol/l), when compared with the age-related decline in total serum testosterone concentrations found in the general population [6]. Furthermore, the prevalence of testosterone deficiency was significantly greater in SCI men with motor complete (AIS A and B) injuries compared with those with motor incomplete (AIS C, D, and E) injuries, and in those taking narcotic medications for pain management [7] .

Erection in Men with SCI

There are two control systems situated in the spinal cord associated with erection; parasympathetic and sympathetic nervous systems. The

parasympathetic center is in sacral segments S2–S4 and is responsible for reflexogenic erection and the sympathetic center is placed in T11–L2 segments of the spinal cord, which is responsible for psychogenic erection[2] . Upper motor neuron (UMN) SCI allows for reflexogenic erection to occur in men, because the parasympathetic sacral reflex arc is intact. Psychogenic erection, however, is usually not possible unless the injury is incomplete. Ejaculation is difficult for these patients because it is sympathetically mediated from T11-L2. Lower motor neuron (LMN) SCI has preserved integrity of the thoracolumbar sympathetic outflow tract, and thus psychogenic erections and ejaculation are theoretically more likely to be intact but reflexogenic erections are not[4] . However, these erections are often unreliable or inadequate for sexual intercourse with difficulties experienced in maintaining an erection[8] .

Treatment for erectile dysfunction in males include Oral medications such as the oral phosphodiesterase 5 inhibitors [PDE5i] ,Intracavernosal injectable medications such as prostaglandin E1 penile injections] and other injectable combinations of papaverine and phentolamine), Topical agents for penile smooth muscle relaxation (prostaglandin, minoxidil, papaverine and nitroglycerine),Intraurethral preparation of prostaglandin E1 (MUSE), Mechanical methods, such as vacuum devices and penile rings and Surgical penile implants.

All methods except surgical penile implants are reversible[9].

Effect of Phosphodiesterase 5 inhibitors on Erection in Men with SCI

Erection is initiated by smooth muscle relaxation of the corpora cavernosa (erectile bodies) of the penis and depends on the nitric oxide-cyclic guanosine monophoshpate [cGMP] pathway. The PDE5i are selective inhibitors of type 5 (cGMP specific) phosphodiesterase, which delay breakdown of cGMP, prolonging and enhances the erectile response. Most clinicians recommend that men with SCI, regardless of their level of injury, be offered a trial of PDE-5 inhibitors [10,11,12] . The recommended dosage of sildenafil is 50 mg 45 minutes before start of sexual intercourse, increased to100mg if not effective[13]

Headache (8-15%) and flushing (3-14%) were seen to be the most common side effects for men with SCI using PDE5i, followed by dyspepsia, nasal congestion, dizziness, visual disturbances and infrequently back pain([10,12]. Urinary tract infection was also found to be a common adverse event, along with headache, in one sildenafil study [14] .Priapism and symptoms of dysreflexia are not reported in the SCI population after PDE5i use [15] .However, PDE5i are contraindicated in SCI patients taking nitrates for angina or coronary heart disease as it causes potentiates the hypotensive effects of nitrates [16] .

Effect of Intracavernosal Injections (ICI)

An intracavernosal (or intracavernous) injection is an injection into the base of the penis. This is often used to administer medications including alprostadil (prostaglandin E1 [PGE1]), Bimix (a combination of papaverine and phentolamine), and Trimix (a combination of papaverine, phentolamine and PGE1), to treat erectile dysfunction in adult men. This treatment can generate an erection in patients with SCI by direct cavernosal vasorelaxation, therefore bypassing the neurotransmission signals (release of nitric oxide from the nerve endings) requisite to initiate erection.([17,18,] .Self-administration is an issue for the subset of SCI patients with poor hand function [19] .

The most common side effects of ICI are transient, such as pain and swelling at the injection site[20]. The more serious side effect of priapism (or prolonged erection) has

typically been reported with use of papaverine and can be treated with aspiration of blood from corpora with injection of an alpha-adrenergic medication [21] or with oral midodrine [22]. Rarely, long-term complication of ICI is fibrosis due to scarring of the tunica albuginia; the risk of which can be reduced by lowering the frequency of injections and minimising medication dose [23].

Effect of Topical Agents

Topical agents aimed to treat erectile dysfunction are applied to the penis or perineal regions and have included hormone-derived medications, as well as vasodilators. Topical agents that cause vasodilation, such as minoxidil,PGE1, papaverine and nitroglycerine, are generally safe yet found to be effective only in a minority of patients (providing an erection sufficient for vaginal penetration in 22-29%), most likely due to inadequate absorption through the tunica albuginia. Use of topical agents to treat ED seem to have little if any role to play in the SCI population, particularly in view of the efficacy and reliability of PDE5i and ICI [24,25]

Intraurethral Preparations and other Medications

The application of intraurethral prostaglandin E1 (alprostadil) can be done by a urologist or self administered to the distal male urethra via a drug deliver system. It appears to produce cavernosal vasodilation to initiate erection. The MUSE study group reported response rates in 60% to 70% range [26] However, in further studies, it was found that the use of intraurethral alprostadil (PGE1) was ineffective in sustaining an adequate erection, and undesirable outcomes such as pain in urethra,patients experiencing hypotension from the medication were documented[27].

Mechanical Methods: Vacuum Erection Devices and Penile Rings

Vacuum device is a pump machine consists of a plastic cylinder with an aperture at one end that is placed over the penile shaft, the other end is a pump mechanism that is used to generate negative pressure within the cylinder to draw venous and arterial blood into the erectile tissue. The erection is maintained by placing a constricting ring around the base of the penis and can slow down the speed at which blood leaves the penis. The constriction ring should not remain on for more than 30 – 45 minutes. The vacuum erection device (VED) is an acceptable alternative for ED therapy in men with SCI who may not tolerate other methods[28]. Unwanted side effects such as premature loss of rigidity, petechiae and penile skin edema, lack of spontaneity, uncomfortable erections and a 'cold penis" were reported. For safe practice, it is recommended that the maximum vacuum pressure should not exceed 250 mmHg (to prevent petechiae and ecchymosis) and the penile ring placed at the base of the penis to trap blood does not remain on for more than 30-45 minutes.[28,29]. Although fairly safe and effective, this device detracts already compromised spontaneity of sexual relations, not suitable for men with poor hand functions and patients who are insensate in the genital area must be cautious with its use. [30].

Surgical Penile Implants

Surgical implantation of a penile prosthesis is one option for erectile dysfunction which involves inserting an implant into the erectile tissue. Different types exist including malleable (semi-rigid) and inflatable (hydraulic). Penile prostheses have been used for over 25 years to treat ED, penile retraction (or a combination of both) or for improvement in urinary management in selected men with SCI[31] .

Generally, for ED, this option is only considered after failure of more

conservative treatments, including ICI of vasoactive substances, vacuum devices and, more recently, oral PDE5i. Inflatable prostheses (although more expensive) are often preferred over semi-rigid malleable prosthesis as the semi-rigid prosthesis is more difficult to conceal due to the permanence of the erection.[32,33]. Serious complications may occur in about 10% of patients, including infection (4-8%) and perforations depending on implant type (9-18% of semi-rigid devices vs 0-2.7% of semiflexible. Under these improved circumstances, implantation of a penile prosthesis appears to be a relatively safe and viable option, perhaps best reserved for when reversible ED therapies have failed or for those men (or their partners) who find other alternatives, such as intracavernosal injections unacceptable [34].

Ejaculatory function in Men with SCI

Ejaculation is a complex process involving coordinated activity of the sympathetic (smooth muscle) and somatic (striated muscle) nervous system controlling prostate and seminal vesicles, bladder neck/ sphincter, pelvic floor and urethra. Ejaculation is primarily a sympathetic phenomenon (involving the spinal cord segment between T10-L2). Internally, there is a pathway for sperm to be transported from the testicles with accessory fluids before being expelled out the end of the penile urethra (antegrade ejaculation) [35]. Ejaculatory disorders (most often the lack of both seminal emission and antegrade ejaculation called anejaculation) are highly prevalent (reported at over 90%) so fertility can be a major issue for men with SCI [36].Ejaculation is most likely to occur naturally in men with incomplete conus or cauda equina lesions and it is least likely to occur naturally in men with complete supraconal lesions[3,19]. Methods that are commonly used for assisting ejaculation include Penile Vibratory Stimulation (for sexual pleasure), Electroejaculation, Prostatic Massage, Surgical Sperm Retrieval[37].

Penile Vibratory Stimulation

PVS is performed using a specialized vibrator placed on the dorsum or frenulum of glans penis to induce reflex ejaculation[38]. Mechanical stimulation produced by the vibrator recruits the ejaculatory reflex to induce ejaculations [39]. This method is more effective in men with incomplete injury or men with an intact ejaculatory reflex , that is , men with a level of injury T10 and above compared to men with a level of injury T11 and below, those men with SCI who had a reflex hip flexion with scratching the soles of the feet and with injuries over 6 months to less than 3.5 years in duration[39,40]. The goal of PVS treatment is to activate the ejaculatory reflex in the thoracolumbar area of the spinal cord. The dorsal penile nerve must be intact (S2-4) for ejaculatory success [41]. The application of a specialized vibrator, with settings of approximately 70-100 Hz with 2.5-3.5 mm amplitude for period of 2-3 minutes on the penis (usually frenulum) followed by rest period of 1-2 minutes. It produces antegrade, retrograde, and some mixed semen samples[39]. After each interval, it is important to examine the penile skin for early detection of skin abrasions or edema, which will determine termination of the stimulation session [42]. If patient is unable to ejaculate with vibrator, additional methods such as application of two vibrators [40], use of abdominal electrical stimulation in addition to PVS [43]

or oral administration of sildenafil prior to PVS[44]. Vibrostimulation has been shown to also induce pronounced levels of autonomic dysreflexia especially in men with tetraplegia [45].

Electroejaculation

Individuals who cannot respond to PVS are often referred to Electroejaculation. This procedure must be administered by a physician trained in this procedure. This method involves placement of a probe in the rectum , after which electric current delivered through the probe stimulates nerves that lead to emission of semen[46,47] . EEJ is contraindicated for patients with inflammatory bowel disease involving the rectum and patients on anticoagulation therapy. Precautions must be taken for SCI who has a pacemaker [45]

Immediately prior to EEJ, bladder is prepared for retrograde ejaculation, which is common with EEJ [48, 49] .Because the acidic pH is toxic to sperm, bladder is emptied by urinary catheterisation. 12-24 ml of buffered medium such as sperm wash medium, may then be instilled through catheter into the bladder. Catheterisation is performed using a plastic catheter to avoid sperm adherence.

The patient is placed in lateral decubitus position to allow insertion of rectal probe and collection of semen from the urethra . The probe is inserted so that the electrodes are just completely inside anal verge. The electrodes are placed in such a way that it faces anteriorly towards prostate and seminal vesicle. Electricity is delivered in a wave like fashion. Recommended procedure is to peak at 5 V for first stimulation , hold at that level for 5 seconds and then abruptly turn off the current. A pause of 10-20 seconds is then allowed inorder to cease the muscle contraction. The next stimulation proceeds after complete cessation of contractions. Voltage is increased tin 2.5 V to 5V increments every 1to 2 stimulations. The procedure is stopped when further stimulations result in no more seminal emissions, when probe temperature exceeds 38.5 degrees or if patient does not tolerate the procedure. The voltage and currents that have been reported to be produce successful ejaculation range from 5 to 25 V and 100 to 600 mA respectively. Ten to twenty stimulations are necessary for complete emptying of the system [50]

Prostatic massage

This technique is mainly used to collect semen for use of insemination [51,52] . This procedure is done by a physician by pressing both seminal vesicle and prostate with a finger inserted in to the patient's rectum. Sperms are stored in the ampulla of the vas deferens and are sequestered in the seminal vesicles in men with SCI. The practitioner attempts to mechanically push the sperm out through the ejaculatory ductal system. [52]

Surgical Sperm Retrieval

SSR is method used for retrieving sperm from reproductive tissue. Techniques such as testicular sperm extraction, testicular sperm aspiration, microsurgical Epididymal sperm aspiration, percutaneous epididymal sperm aspiration and aspiration of sperm from vas deferens are used. These methods are not used to treat anejaculation. These methods are more commonly used to treat infertility in men with SCI[53]

Fertility In Males with SCI

Male fertility after SCI is often compounded by the difficulties of erectile dysfunction, as well as

retrograde ejaculation or anejaculation. Semen quality is also noted to decline after SCI [54]. Semen quality in men with chronic SCI is reported to have decreased motility and viability, although total numbers of sperm tend to remain high. Sperm DNA damage, which is another method of assessing semen quality, has also been shown to be higher among men with SCI when compared to able-bodied controls [55] .There is also some evidence to suggest that vibratory stimulus seems to produce better semen quality samples than electroejaculation in terms of motility [56]. Semen quality appeared to improve with repeated ejaculations in some series [57,58], and not in others [59]. Once-weekly vibrator stimulation resulted in an increase of semen volume and of fructose and acid phosphatase levels in the seminal plasma (suggesting improved function of the seminal vesicles and prostate) [60]. Unfortunately, after SCI, semen quality declines necessitating assistive reproductive technologies to compensate for the alterations [61]. Pregnancy rates are lower than the general population but have been much improved since the advent of in vitro fertilization (IVF) and intracytoplasmic injection (ICSI). Pregnancy rates of partners of men with SCI, although somewhat dependent on sperm motility, are improved consistently with higher levels of reproductive assisted technology. With the use of assisted reproductive technology such as Intrauterine insemination, In vitro fertilization (IVF), IVF + intracytoplasmic sperm injection (ICSI) , there has been improvement in the fertility rate among men with SCI. [46,54,62]. Cycle fecundity rate (chance of pregnancy per cycle) for intrauterine insemination is <15%, whereas for IVF/ICSI it is between 25- 40% [62]. While fresh semen samples were preferred, cryopreserved semen samples were used successfully for IVF technology. Cryopreservation of embryos to be replaced at a later date is also useful. Multiple gestations were more frequent with IVF/ICSI[63]. Pregnancy rates among couples using testicular aspirated sperm from males with SCI were comparable to the rates among couples using the same procedure from able-bodied controls with obstructive azoospermia. [64,65]. The use of retrograde vasal sperm aspiration has also shown to be a reliable method for consistent sperm recovery such that a high pregnancy rate and cryopreservation of excess sperm for future use was possible(66).

Men with SCI stand a good chance (>50%) of becoming biological fathers when they have access to specialized clinics and care. Studies have found that fertilization rates of 55-57%, embryo cleavage rates of 90-93%,and pregnancy rates of 50-75% when using antegrade ejaculation or surgical sperm retrieval (SSR) [67].

Conclusion.

Both men and women desire sexual contact and fertility even after a spinal cord injury, although SCI – related physiological changes may interfere with enjoyment of sexual activity and reproduction. Men with SCI experience erectile dysfunction, ejaculatory dysfunction and semen abnormalities. Erectile dysfunction can be treated with oral PDE5i, intracaverous injections, vacuum erection devices and penile implants. Ejaculation for sexual pleasure and fertility can be improved with penile vibratory stimulator. This method and other methods such as electroejaculation , prostatic massage can be used to obtain semen in anejaculatory men with SCI. Methods to assist conception in couples with male SCI partners such as IUI, IVF can be performed with or without ICSI. Ejaculates of men should be evaluated for sperm count, motility and viability prior to using ART.

References

1. Previnaire JG, Soler JM, Alexander MS, Courtois F, Elliott S, McLain A. Prediction of sexual function following spinal cord injury: A case series. Spinal cord series and cases. 2017 Dec 13;3(1):1-7.
2. Toksoz S. Sexual Rehabilitation of Patients with Spinal Cord Injury. J Spine S. 2016;7:2.
3. Monga M, Bernie J, Rajasekaran M. Male infertility and erectile dysfunction in spinal cord injury: a review. Archives of physical medicine and rehabilitation. 1999 Oct 1;80(10):1331-9.
4. Braddom 5th Edition, Chapter 22, page 473
5. Wu JC, Chen YC, Liu L, Chen TJ, Huang WC, Cheng H, Su TP. Effects of age, gender, and socio-economic status on the incidence of spinal cord injury: an assessment using the eleven-year comprehensive nationwide database of Taiwan. Journal of neurotrauma. 2012 Mar 20;29(5):889-97.
6. Bauman WA, La Fountaine MF, Spungen AM. Age-related prevalence of low testosterone in men with spinal cord injury. The journal of spinal cord medicine. 2014 Jan 1;37(1):32-9.
7. Durga A, Sepahpanah F, Regozzi M, Hastings J, Crane DA. Prevalence of testosterone deficiency after spinal cord injury. PM&R. 2011 Oct;3(10):929-32.
8. Courtois FJ, Goulet MC, Charvier KF, Leriche A. Posttraumatic erectile potential of spinal cord injured men: how physiologic recordings supplement subjective reports. Archives of physical medicine and rehabilitation. 1999 Oct 1;80(10):1268-72.
9. Abramson CE, McBride KE, Konnyu KJ, Elliott SL. Sexual health outcome measures for individuals with a spinal cord injury: a systematic review. Spinal Cord. 2008 May;46(5):320-4.
10. Rizio N, Tran C, Sorenson M. Efficacy and satisfaction rates of oral PDE5is in the treatment of erectile dysfunction secondary to spinal cord injury: a review of literature. The journal of spinal cord medicine. 2012 Jul 1;35(4):219-28
11. Elliott S, McBride K. Sexual and reproductive health following spinal cord injury. Spinal cord injury rehabilitation evidence, Version. 2014;5:1-84.
12. Derry F, Hultling C, Seftel AD, Sipski ML. Efficacy and safety of sildenafil citrate (Viagra®) in men with erectile dysfunction and spinal cord injury: a review. Urology. 2002 Aug 1;60(2):49-57.
13. Khorrami MH, Javid A, Moshtaghi D, Nourimahdavi K, Mortazavi A, Zia HR. Sildenafil efficacy in erectile dysfunction secondary to spinal cord injury depends on the level of cord injuries. International journal of andrology. 2010 Dec;33(6):861-4.
14. Ergin S, Gunduz B, Ugurlu H, Sivrioglu K, Oncel S, Gok H, Erhan B, Levendoglu F, Senocak O. A placebo-controlled, multicenter, randomized, double-blind, flexible-dose, two-way crossover study to evaluate the efficacy and safety of sildenafil in men with traumatic spinal cord injury and erectile dysfunction. The journal of spinal cord medicine. 2008 Jan 1;31(5):522-31.
15. Giuliano F, Hultling C, El Masry WS, Smith MD, Osterloh IH, Orr M, Maytom M, Sildenafil Study Group. Randomized trial of sildenafil for the treatment of erectile dysfunction in spinal cord injury. Annals of neurology. 1999 Jul;46(1):15-21.
16. Webb DJ, Muirhead GJ, Wulff M, Sutton JA, Levi R, Dinsmore WW. Sildenafil citrate potentiates the hypotensive effects of nitric oxide donor drugs in male patients with stable angina. Journal of the American College of Cardiology. 2000 Jul 1;36(1):25-31.
17. Yildiz N, Gokkaya NK, Koseoglu F, Gokkaya S, Comert D. Efficacies of papaverine and sildenafil in the treatment of erectile dysfunction in early-stage paraplegic men. International Journal of Rehabilitation Research. 2011 Mar 1;34(1):44-52.
18. Zaslau S, Nicolis C, Galeap G, Britanico J, Jonathan VM. A simplified pharmacologic erection program for patients with spinal cord injury. The journal of spinal cord medicine. 1999 Jan 1;22(4):303-7.
19. Ibrahim E, Lynne CM, Brackett NL. Male fertility following spinal cord injury: an update. Andrology. 2016 Jan;4(1):13-26.
20. Tang SF, Chu NK, Wong MK. Intracavernous injection of prostaglandin E1 in spinal cord injured patients with erectile dysfunction. A preliminary report. Spinal Cord. 1995 Dec;33(12):731-3.
21. Bodner DR, Leffler B, Frost F. The role of intracavernous injection of vasoactive medications for the restoration of erection in spinal cord injured males: a three year follow up. Spinal Cord. 1992 Feb;30(2):118-20.
22. Soler JM, Previnaire JG, Mieusset R, Plante P. Oral midodrine for prostaglandin e1 induced priapism in spinal cord injured patients. The Journal of urology. 2009 Sep;182(3):1096-100.
23. Ismail M, Abbott L, Hirsch IH. Experience with intracavernous PGE-1 in the treatment of erectile dysfunction: dose considerations and efficacy. International journal of impotence research. 1997 Mar;9(1):39-42.
24. Kim ED, McVary KT. Topical prostaglandin-E1 for the treatment of erectile dysfunction. The Journal of urology. 1995 Jun 1;153(6):1828-30.
25. Watanabe T, Chancellor MB, Rivas DA, Hirsch IH, Bennett CJ, Finocchiaro MV, Razi S, Bennett JK, Green BG, Foote JE, Killorian RW. Epidemiology of current treatment for sexual dysfunction in spinal cord injured men in the USA model spinal cord injury centers. The journal of spinal cord medicine. 1996 Jan 1;19(3):186-9.
26. Padma-Nathan H, Hellstrom WJ, Kaiser FE, Labasky RF, Lue TF, Nolten WE, Norwood PC, Peterson CA, Shabsigh R, Tam PY, Place VA. Treatment of men with erectile dysfunction with transurethral alprostadil. New England Journal of Medicine. 1997 Jan 2;336(1):1-7.
27. Bodner DR, Haas CA, Krueger B, Seftel AD. Intraurethral alprostadil for treatment of erectile dysfunction in patients with spinal cord injury. Urology. 1999 Jan 1;53(1):199-202.
28. Brison D, Seftel A, Sadeghi-Nejad H. The resurgence of the vacuum erection device (VED) for treatment of erectile dysfunction. The journal of sexual medicine. 2013 Apr 1;10(4):1124-35.
29. Brackett NL, Lynne CM, Ibrahim E, Ohl DA, Sønksen J. Treatment of infertility in men with spinal cord injury. Nature reviews urology. 2010 Mar;7(3):162.
30. Kirshblum 2nd Edition , Chapter 23 page 415.
31. Hellstrom WJ, Montague DK, Moncada I, Carson C, Minhas S, Faria G, Krishnamurti S. Implants, mechanical devices, and vascular surgery for erectile dysfunction. The journal of sexual medicine. 2010 Jan 1;7(1):501-23.
32. Henry G, Houghton L, Culkin D, Otheguy J, Shabsigh R, Ohl DA. Comparison of a new length measurement technique for inflatable penile prosthesis implantation to standard techniques: outcomes and patient satisfaction. The journal of sexual medicine. 2011 Sep 1;8(9):2640-6.

33. Selph P, Carson C. Penile implant surgery. Male Sexual Dysfunction: A Clinical Guide. 2017 Jan 10:258-69.

34. Trost L, Wanzek P, Bailey G. A practical overview of considerations for penile prosthesis placement. Nature Reviews Urology. 2016 Jan;13(1):33.

35. Krassioukov A, Elliott S. Neural control and physiology of sexual function: effect of spinal cord injury. Topics in spinal cord injury rehabilitation. 2017 Dec;23(1):1-0.

36. Elliott S. Ejaculation and orgasm: Sexuality in men with SCI. Topics in Spinal Cord Injury Rehabilitation. 2002 Jul 1;8(1):1-5.

37. Kirshblum, Chapeter 23 Page 416-418.

38. Brackett NL. Semen retrieval by penile vibratory stimulation in men with spinal cord injury. Human Reproduction Update. 1999 May 1;5(3):216-22.

39. Sønksen J, Ohl DA. Penile vibratory stimulation and electroejaculation in the treatment of ejaculatory dysfunction 1. International journal of andrology. 2002 Dec;25(6):324-32.

40. Brackett NL, Kafetsoulis A, Ibrahim E, Aballa TC, Lynne CM. Application of 2 vibrators salvages ejaculatory failures to 1 vibrator during penile vibratory stimulation in men with spinal cord injuries. The Journal of urology. 2007 Feb 1;177(2):660-3.

41. Wieder JA, Brackett NL, Lynne CM, Green JT, Aballa TC. Anesthetic block of the dorsal penile nerve inhibits vibratory-induced ejaculation in men with spinal cord injuries. Urology. 2000 Jun 1;55(6):915-7.

42. Sinha V, Elliott S, Ibrahim E, Lynne CM, Brackett NL. Reproductive health of men with spinal cord injury. Topics in spinal cord injury rehabilitation. 2017 Dec;23(1):31-41.

43. Kafetsoulis A, Ibrahim E, Aballa TC, Goetz LL, Lynne CM, Brackett NL. Abdominal electrical stimulation rescues failures to penile vibratory stimulation in men with spinal cord injury: a report of two cases. Urology. 2006 Jul 1;68(1):204-e9.

44. Giuliano F, Rubio-Aurioles E, Kennelly M, Montorsi F, Kim ED, Finkbeiner AE, Pommerville PJ, Colopy MW, Wilkins HJ, Wachs BH. Efficacy and safety of vardenafil in men with erectile dysfunction caused by spinal cord injury. Neurology. 2006 Jan 24;66(2):210-6.

45. Brown R, Stolzenhein G, Engel S, Macefield VG. Cutaneous vasoconstriction as a measure of incipient autonomic dysreflexia during penile vibratory stimulation in spinal cord injury. Spinal Cord. 2009 Jul;47(7):538-44.

46. Brown DJ, Hill ST, Baker HW. Male fertility and sexual function after spinal cord injury. Progress in brain research. 2006 Jan 1;152:427-39.

47. Ohl DA, Quallich SA, Sønksen J, Brackett NL, Lynne CM. Anejaculation and retrograde ejaculation. Urologic Clinics of North America. 2008 May 1;35(2):211-20.

48. Brackett NL, Padron OF, Lynne CM. Semen quality of spinal cord injured men is better when obtained by vibratory stimulation versus electroejaculation. The Journal of urology. 1997 Jan;157(1):151-7.

49. SØNKSEN J, OHL DA, WEDEMEYER G. Sphincteric events during penile vibratory ejaculation and electroejaculation in men with spinal cord injuries. The Journal of urology. 2001 Feb 1;165(2):426-9.

50. Ohl DA. Electroejaculation. The Urologic clinics of North America. 1993 Feb;20(1):181-8.

51. Taylor Z, Molloy D, Hill V, Harrison K. Contribution of the assisted reproductive technologies to fertility in males suffering spinal cord injury. Australian and New Zealand journal of obstetrics and gynaecology. 1999 Feb;39(1):84-7.

52. Craft I, Tsirigotis M. More experiences with ICSI continued: Simplified recovery, preparation and cryopreservation of testicular spermatozoa. Human Reproduction. 1995 Jul 1;10(7):1623-6.

53. Kirshblum 2nd Edition , Chapter 23 page 418.

54. DeForge D, Blackmer J, Garritty C, Yazdi F, Cronin V, Barrowman N, Fang M, Mamaladze V, Zhang L, Sampson M, Moher D. Fertility following spinal cord injury: a systematic review. Spinal cord. 2005 Dec;43(12):693-703.

55. Brackett NL, Ibrahim E, Grotas JA, Aballa TC, Lynne CM. Higher sperm DNA damage in semen from men with spinal cord injuries compared with controls. Journal of andrology. 2008 Jan 2;29(1):93-9.

56. Le Chapelain L, Van Tam PN, Dehail P, Berjon JJ, Barat M, Mazaux JM, Joseph PA. Ejaculatory stimulation, quality of semen and reproductive aspects in spinal cord injured men. Spinal Cord. 1998 Feb;36(2):132-6.

57. Giulini S, Pesce F, Madgar I, Marsella T, Volpe A, de Aloysio D, Battaglia C. Influence of multiple transrectal electroejaculations on semen parameters and intracytoplasmic sperm injection outcome. Fertility and sterility. 2004 Jul 1;82(1):200-4.

58. Patki P, Woodhouse J, Hamid R, Craggs M, Shah J. Effects of spinal cord injury on semen parameters. The journal of spinal cord medicine. 2008 Jan 1;31(1):27-32.

59. Das S, Dodd S, Soni BM, Sharma SD, Gazvani R, Lewis-Jones DI. Does repeated electro-ejaculation improve sperm quality in spinal cord injured men?. Spinal Cord. 2006 Dec;44(12):753-6.

60. Siösteen A, Forssman L, Steen Y, Sullivan L, Wickström I. Quality of semen after repeated ejaculation treatment in spinal cord injury men. Spinal Cord. 1990 Feb;28(2):96-104.

61. Elliott SL. Sexual dysfunction and infertility in men with spinal cord injury. InSpinal cord medicine: Principles and practice 2010 Mar 19 (pp. 409-428). Demos Medical Publishing, New York.

62. Shieh JY, Chen SU, Wang YH, Chang HC, Ho HN, Yang YS. A protocol of electroejaculation and systematic assisted reproductive technology achieved high efficiency and efficacy for pregnancy for anejaculatory men with spinal cord injury. Archives of physical medicine and rehabilitation. 2003 Apr 1;84(4):535-40.

63. Chung PH, Yeko TR, Mayer JC, Sanford EJ, Maroulis GB. Assisted fertility using electroejaculation in men with spinal cord injury—a review of literature. Fertility and sterility. 1995 Jul 1;64(1):1-9.

64. Kanto S, Uto H, Toya M, Ohnuma T, Arai Y, Kyono K. Fresh testicular sperm retrieved from men with spinal cord injury retains equal fecundity to that from men with obstructive azoospermia via intracytoplasmic sperm injection. Fertility and sterility. 2009 Oct 1;92(4):1333-6.

65. Raviv G, Madgar I, Elizur S, Zeilig G, Levron J. Testicular sperm retrieval and intra cytoplasmic sperm injection provide favorable outcome in spinal cord injury patients, failing conservative reproductive treatment. Spinal Cord. 2013 Aug;51(8):642-4.

66. Hibi H, Ohori T, Yamada Y, Honda N, Hashiba Y, Asada Y. Retrograde vasal sperm aspiration in anejaculatory patients with spinal cord injury. Reproductive medicine and biology. 2008 Sep 1;7(3):115-8.

67. Bechoua S, Berki-Morin Y, Michel F, Girod S, Sagot P, Fauque P. Outcomes with intracytoplasmic sperm injection of cryopreserved sperm from men with spinal cord injury. Basic and clinical andrology. 2013 Dec;23(1):14.

LIPOMA ARBORESCENS: A RARE CAUSE OF SWELLING OF THE KNEE

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A 38 year old female homemaker, presented to our department with complaints of multiple joint pain and swelling of 6 years duration & difficulty to walk for 1 year.

6 years back she developed right knee pain, which gradually progressed with swelling and no symptoms suggestive of inflammatory arthritis. It was treated in local hospital with analgesics and got relief. Subsequently she developed pain and swelling of both knee joint. She was investigated and diagnosed as chronic synovitis both knee with synovial biopsy proved pannus formation. Treated as rheumatoid arthritis with remission and exacerbation, by various specialties like orthopedics, rheumatology and alternative medicine for 5 years. Recently, she again developed the symptoms in both knees and ankle, associated with difficulty to walk, dressing and toileting. No h/o small joint involvement, morning stiffness. She came to our OPD and was admitted in our department for rehabilitation medicine intervention.

Past history

History of Chikungunya 8 years back

History of treatment for infertility 8 years back. Conceived after 2 years of treatment. No history of abortions.

No history of TB, UTI, eye infections, inflammatory bowel disease, skin infections

No other co-morbidities

Family history

No history of similar illness/ rheumatoid arthritis

No history of Diabetes mellitus/ hypertension/ CAD

Local examination – knee

Inspection

Bilateral suprapatellar fullness

Bilateral genu valgum

Prominent veins visible over left knee

Palpation

Local rise of temperature present

Medial and lateral joint line tenderness

Diffuse boggy swelling in suprapatellar region

Bilateral patellar tap present

Local examination – ankle

Inspection

Hyperpigmentation over the left ankle

Left hallux valgus

Bilateral diffuse ankle swelling

Palpation

Local rise of temperature present on both ankle

Tenderness present bilaterally

Diffuse boggy swelling at both ankle

Musculoskeletal exam- revealed all joints had full passive range of movement except for the knees and ankles. Knee RoM was 0-100°on the right and 0-110° on the left. There were 5 tender joints and 5 swollen joints. They were bilateral knees and ankle, and 1st MTP on the left.

After history and clinical examination, the differential

Diagnosis we considered were :

1. Inflammatory arthritis – probably rheumatoid arthritis with secondary osteoarthritis
2. Reactive arthritis
3. Post viral arthritis
4. Non inflammatory arthritis – degenerative
5. Pigmented villonodular synovitis

Other systems

Central nervous system
Respiratory system
Cardiovascular system
Gastrointestinal system
Breast and thyroid
} Normal

Course in the Hospital

Admitted and investigations done and NSAIDs were given.

Routine blood investigations and serological test for Rheumatoid arthritis were done.

Specific test

Blood routine	Renal function test	Liver function test
Hemoglobin – 11.1gm%	Urea – 17mg/dl	Bilirubin (T) – 0.2mg/dl
Total count – 8600/cmm	Creatinine – 0.5mg%	(D) – 0.1mg/dl
Differential Count – P50%, L46%, E4%		SGOT – 15U/ml
Platelet – 4.8 lakhs/cmm		SGPT - 12U/ml
ESR – 55mm/hr		ALP – 58 IU/l

Uric acid -	3.8mg%
S. Calcium -	9.6mg%
CRP -	1.5mg/dl
ANA -	Negative
Anti CCP -	1.2U/ml
Rheumatoid factor -	0.1 (neg)
IgM chikungunya -	Negative
IgM chikungunya -	Negative
Mantoux test -	Negative

X Ray

X ray showed soft tissue swelling and degenerative changes of both knee joints.

USG both knee

Ultrsonographic findings suggestive of bilateral asymmetric diffuse synovial thickening with papillary projections

Synovial fluid cytology

Synovial fluid aspiration done from both knee joint.

20ml of fluid was aspirated from right knee joint.

Cytology

Straw coloured viscous fluid.

TC- 0-1cells/hpf

DC-100% lymphocytes

Impression: Chronic inflammation

Steroid infiltration to both knee joints done. Still pain persisted and so MRI was considered

MRI both knee

Fat density fond like projections of synovium with mild effusion within the bilateral suprapatellar and infrapatellar bursa and knee joint with degenerative changes of knee joint – possibly lipoma arborescens-secondary type.

Synovectomy is the treatment of choice. So along with orthopedics department synovectomy done.

Biopsy

Lipoma arborescens with synovitis

Post-op Rehabilitation

Rehabilitation started from day 1

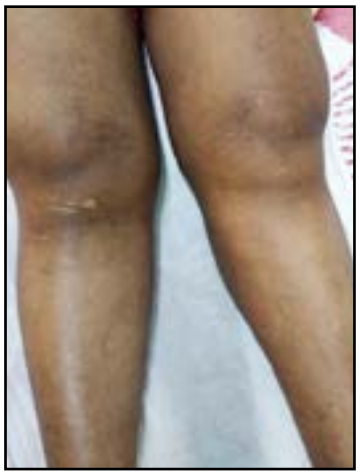
ROM and strengthening exercises started.

Gait training initiated in parallel bar

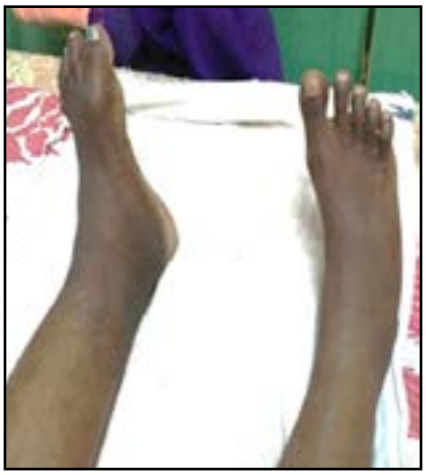
Then walker and achieved walking without assistance by 2 weeks.

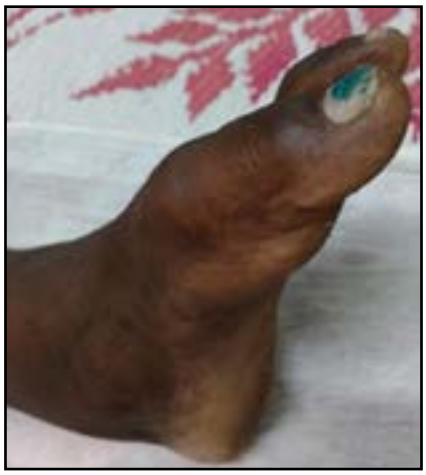
Conclusion

Lipoma arborescens is a benign indolent synovial proliferative disease and an uncommon cause of articular masses. It primarily involves knee joint. This entity can be confidently diagnosed by its characteristic features on various imaging modalities, particularly MRI. Awareness of its clinical and imaging findings and possible differential diagnosis is essential for early diagnosis and treatment, as well as to avoid misinterpretation of this benign condition with other aggressive articular masses.

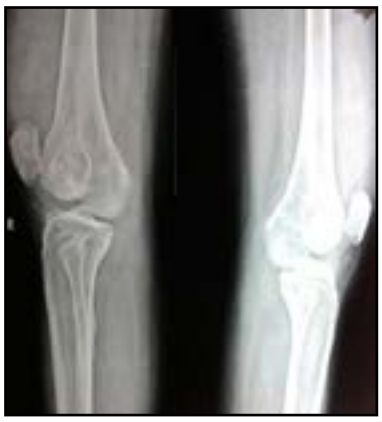


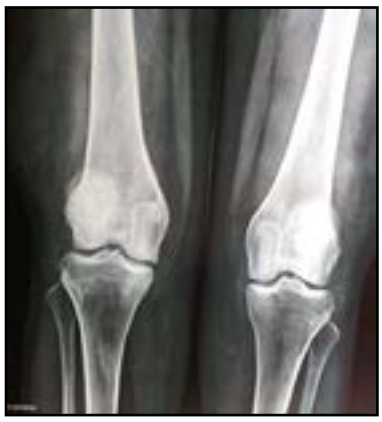
Inspection Knee Joint

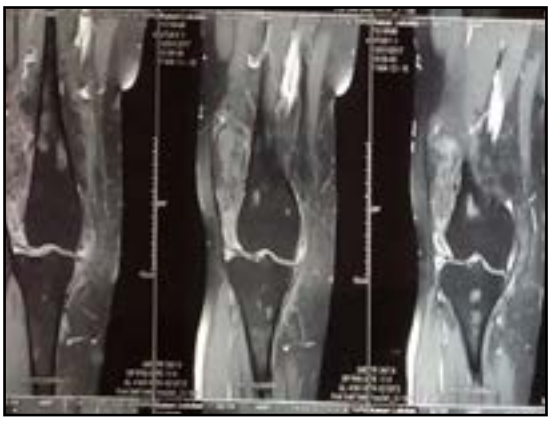


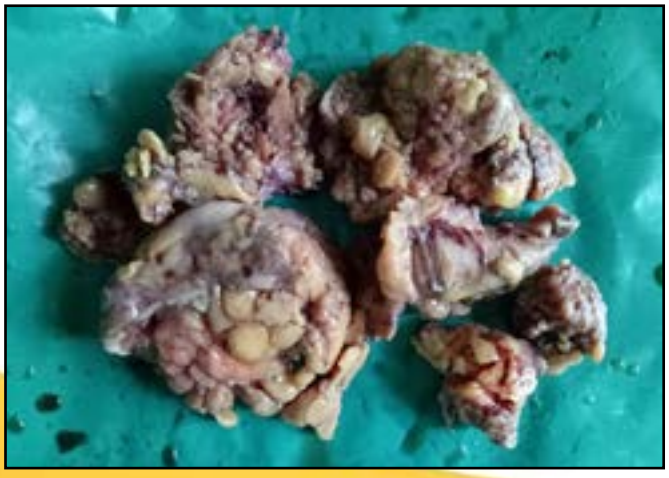


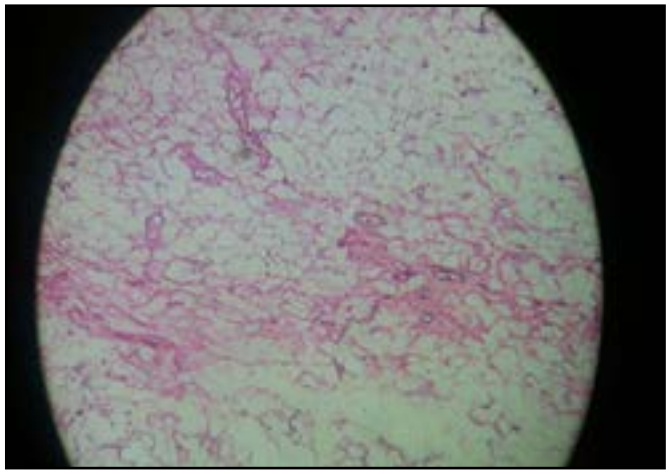
Inspection of Ankle Joint











Use Of Neuromuscular Ultrasound In PMR Practice-A Case Series

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High resolution ultrasound has become a bedside tool for the clinician. It has revolutionised diagnosis and treatment of musculoskeletal conditions. For the physiatrists it is more so important because most of the times they are dealing with conditions related to muscle and nerve. It has got important role in diagnosis, decision making and planning and implementation of rehabilitation.

Peripheral nerve ultrasound has been well described even from the mid 1980's 1 . Over the years it has been proved an effective tool in diagnosis of peripheral nerve pathology. It is complementary to electrophysiological examination, correlating well with electrophysiology. The two methods combined together give better result than electrophysiology alone 2 . USG can provide details about the anatomy of the involved nerve which is not possible by electrophysiology 3. Compared with MRI ultrasound has got the advantages of higher resolution for smaller nerves, ability to scan whole length of the nerve and ease of doing dynamic evaluation4.

Qualitative ultrasound of the muscle is also helpful in diagnosis of neuromuscular pathologies. Normally the muscle is seen with decreased echogenecity (darker) interspersed with bright connective tissue 5. In case of muscle pathologies the muscle becomes more echogenic (brighter) and the echogenesity increases with progression of pathology. In extreme cases the underlying bone which is brighter cannot be identified separately from the muscle because of the muscle echogenesity.

Heckmatt's visual rating scale for skeletal muscle ultrasound. 6	
Grade	Description
I	Normal
II	Increased muscle signal with preserved,distinct bone echo
III	Marked increase in muscle signal with reduced bone echo
IV	Very increased muscle signal with loss of bone echo

Heckematt's criteria is used to grade the abnormality of the muscle 6.

Given below is a case series consisting of 3 cases. Neuromuscular ultrasound played an important role either in the diagnosis or evaluation of these cases. All these cases were reported at the department of PM&R, Govt. Medical College, Kozhikode. USG was performed by a physiatrist having 5 years experience in musculoskeletal ultrasound. The examination was performed by USG machine with high frequency linear probe (5-13 MHz)

Case I

50 year old female patient was referred by a neurologist to the department of PM&R as a case of left sided PIN palsy for bracing and exercises. She had insidious onset of fingerdrop 2 months back. NCS showed features of axon loss lesion for PIN innervated muscle with a normal SNAP of the superficial radial nerve. She was on steroids for the last 2 months with no significant improvement. Because of the static nature of the problem we decided to re evaluate the case and performed USG evaluation of the left elbow and forearm. USG showed hypoechoic lesion compressing the PIN soon after the division (fig1). The supinator muscle on the affected side showed increased echo-texture and atrophy (fig 2). A contrast enhanced MRI was done and the lesion was suggestive of a ganglion cyst. Patient is posted for a decompression surgery.

Case II

55 year old female with no history of Diabetes or previous trauma reported to the department of PM&R with insidious onset of weakness of left hand which was progressive. Clinically patient had features of high ulnar nerve palsy of left side. She had seen already by other physicians and NCS showed features of axon loss lesion of ulnar nerve with features of moderate CTS. USG showed compression of ulnar nerve on the affected side by an osteophyte with swelling of the nerve proximal to that (fig 3). X ray of the elbow also showed the osteophyte (fig4) . Patient was referred to the orthopaedic department for ulnar nerve transposition surgery.

Case III

10 year old boy , a known case of Duchenne Muscular dystrophy (DMD) reported to the PMR OPD with recent worsening of symptoms. Clinical examination showed weakness of different groups of muscles. USG was performed to check the condition of different muscles. The quadriceps muscle was found to be more involved (fig 5). The USG is useful in monitoring the status of muscle by serial examination and also in deciding which muscle is likely to yield findings form EMG.

Conclusion

These cases highlight the importance of neuromuscular ultrasound in PMR practice. Neuromuscular ultrasound is helpful in establishing the diagnosis in many cases which will be otherwise confusing. In many cases they are useful in evaluation of progress of the disease. USG guided interventions are also gaining popularity in day to day PMR practice. So residents must get adequate exposure and training in neuromuscular ultrasound.

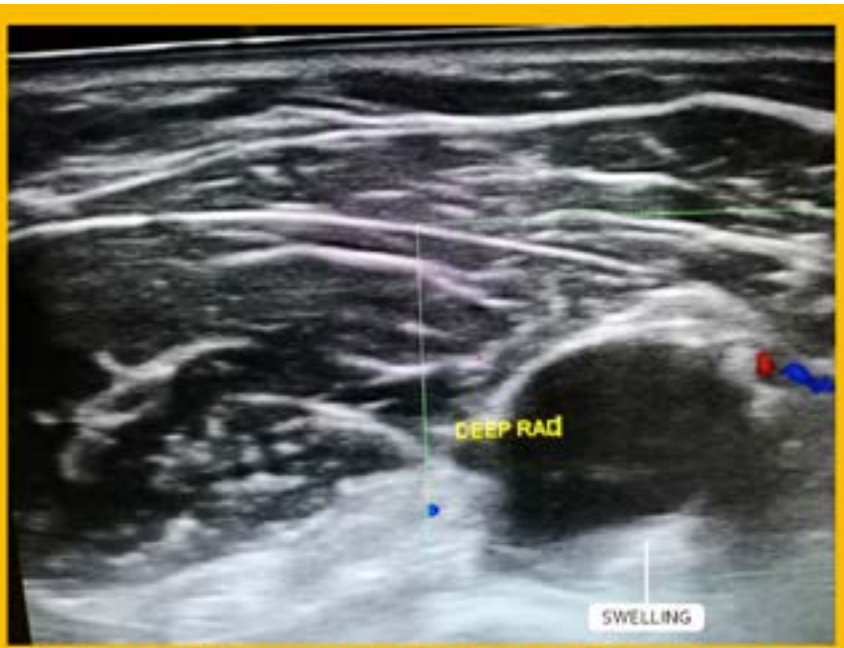

Figure 1. Swelling compressing the Posterior interosseous nerve

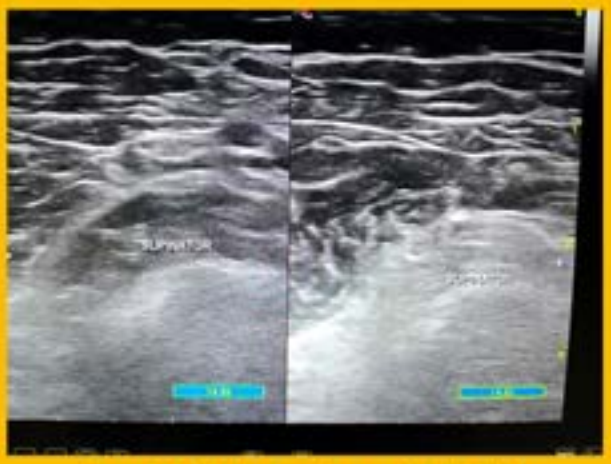

Figure 2. Affected supinator muscle (right side) is hyperechoic and reduced in size.

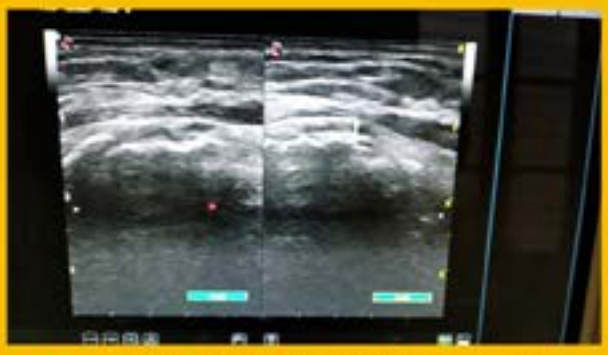

Fig 3. Arrow shows the compression of ulnar nerve. Left image shows normal nerve.

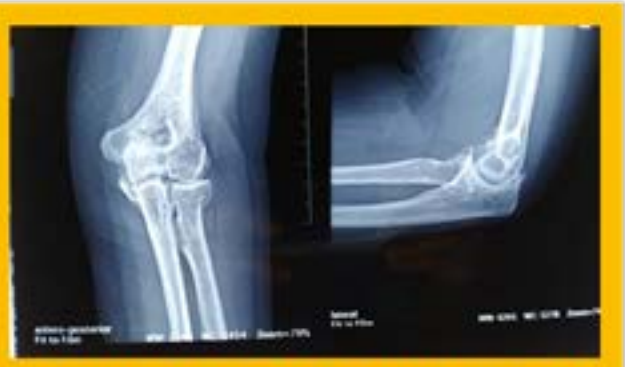

Fig4. X ray of the same patient shows osteophyte (arrow)

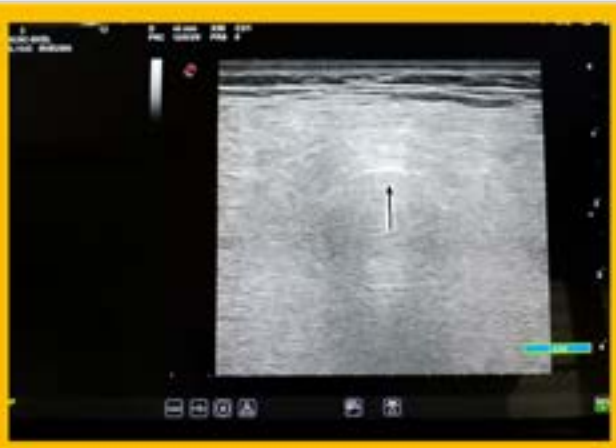

Fig 5. USG of quadriceps of a patient with DMD. The bony margin (arrow) is hardly visible.

References:

1. Solbiati L., De Pra L., Ierace T., et al: High-resolution sonography of the recurrent laryngeal nerve: Anatomic and pathologic considerations. AJR Am J Roentgenol 1985; 145: pp. 989-993
2. Beekman R., and Visser L.H.: High-resolution sonography of the peripheral nervous system: A review of the literature. Eur J Neurol 2004; 11: pp. 305-314
3. Spratt J.D., Stanley A.J., Grainger A.J., et al: The role of diagnostic radiology in compressive and entrapment neuropathies. Eur Radiol 2002; 12: pp. 2352-2364
4. In Bianchi S., and Martinoli C. (eds): Ultrasound of the musculoskeletal system, ed 1. Bonn, Germany: Springer-Verlag, 2007.
5. Heckmatt JZ, Dubowitz V, Leeman S: Detection of pathological change in dystrophic muscle with B-scan ultrasound imaging, Lancet 1:1389–1390, 1980.
6. Heckmatt JZ, Leeman S, Dubowitz V: Ultrasound imaging in the diagnosis of muscle disease, J Pediatr 101:656–660, 1982.

Why should I write?

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Amrita Institute of Medical Sciences

This was the response I gave to my friend and senior neurosurgeon who was prompting me to publish. I'd just finished residency and was eager to finally be responsible for my own clinical outcomes. I didn't do a MD course to sit and type documents. Though I learned a lot about Birth related Brachial Plexus injuries from my thesis, doing the same again seemed boring. Regardless, I spent the time working with him to get our case report on 'Congenital subaxial cervical subluxation presenting as a bilateral Erb's palsy' (1) written up. Luckily as it had never been reported, nor the treatment we used known, there wasn't too much published work to sort through. After two years (between his busy schedule, my lack of motivation, and journal rejections) we finally got it into an international journal.

You might think the story becomes brighter from there. It doesn't. Not really knowing the value of what I had accomplished, I went back to daily clinical care and learning more skills to serve patients with. By 2015 administration was harrying us to publish for National Assessment and Accreditation Council (NAAC) accreditation. My thesis was the only manuscript I had in hand, so it got published. Vethalam (publications) flew back to its tree and patient care resumed.

January 2015 is when we started our Hyperbaric Medical service. Seeing people improve or stay the same got me wondering why this was the case. The literature reviews I'd done all showed that Hyperbaric Oxygen was not useful in stroke care, but this contradicted what I saw with my own eyes. The therapy worked so well Neurosurgery was keeping the chamber full and running all day. On looking deeper into those publications I saw they were designed poorly. The certification course I took in the US didn't provide answers either. When I asked my mentors at Palmetto they were clueless. That left me to find answers to my own questions. The errors in my hypotheses became evident as patients defied what I expected as outcomes. When this happens one is left with two options. Give up or persevere. I chose the latter, and with another Neurosurgery friend mapped out a prospective study protocol. One of the hardest things was getting enough patients who were comparable at baseline, and then from there getting it published. The four rejection letters I got from PMR journals lead me to publish the manuscript outside of Pubmed (2). By this point though I got what I really wanted. I knew enough to be able to counsel patient-parties about when they should use HBOT for their stroke patients and what to expect. I saw the publication as a fringe benefit.

I learned the value of publishing with my next HBOT paper. From an inpatient

service of 20-30 patients daily we were slowly being whittled down to 25, 20, and stabilized around 10-15. What had changed was all the Acquired Brain Injury patients were going home early. Why? With HBOT they were waking up faster and the family could care for them at home. So Neurosurgery gave us less transfers. Doing a job well has its ups and downs. We were left with spinal cord injury and Hypoxic Ischemic Encephalopathy patients. Interestingly the HIE patients showed improvements with HBOT. With guidance from the former head of Sri-Chitra Trivandrum this work got published in Neurology India (3). I learned a lot of new theory but the real benefit came later. Anyone can be a service provider and market, but getting published sets you apart as an authority. The suggestions in that paper have value as it cleared a peer review and is in Pubmed. Not everyone can do this. But you certainly must for the patients you serve.

FAQ

-Why should I write?

'The Eye sees what mind knows'. You are the primary beneficiary here. The more you learn on a topic the deeper your knowledge will go until you become an expert in your own right. Making a tight hypothesis and writing the discussion are often the hardest parts of a study because you'll have to review what others have done and explain your outcomes in relation to this. If you see yourself as a specialist of something, publishing on the same validate this opinion. If you don't, work for it.

-'English isn't my first language'.

Don't worry, it's the only one I actually know. Apps like grammarly.com make it even easier to improve yourself. Between the two of these you'll have support, and many chances to get better.

-'How should I write?'

'Write for an idiot' (4). Explain from the basics up in simple language. Obviously your audience is physician peers, but doing this will make you aware of your own knowledge gaps in private. Filling those in gives you a solid foundation to act from later. When you start writing don't be surprised if you end up with many more questions than you started with.

'Who are you to tell me to write?'

I'm just another learner. If you look up my impact factor it's not that hot. This is my way of helping others and growing at the same time.

'I feel no motivation to write.'

Then sit back watch others surpass you, and write guidelines for you to follow. Those who reject guidelines blind themselves. Patients will eventually pick up on this. Utilizing the existing body of literature separates the professional from the practitioner. Look at the difference between a surgeon and a barber. 'Whereas the art of the barber has remained essentially unchanged over many millennia, the profession of the surgeon continues to evolve, stimulated by technical improvements and technological innovation. Wherein lies the difference between these two activities? Is it not in the building up and transmission of an evolving body of knowledge?'(5) If you don't even make an effort to improve, there is no one to blame but yourself when your work has no value.

References

1.Congenital subaxial cervical subluxation presenting as a bilateral Erb's palsy: surgical management, rehabilitation, and outcome, Childs Nerv Syst ,25 October 2015 DOI 10.1007/s00381-015-2946-5, Sankaran R, Shah R, Menon S, Pillai AB

2. Hyperbaric Oxygen therapy in Acute Ischemic Stroke, Sankaran R, NR Sreehari, Indian Journal of applied research Feb 2019

3. Hyperbaric oxygen therapy in patients with hypoxic ischemic encephalopathy. Sankaran R, Radhakrishnan K, Sundaram KR. Neurol India 2019;67:728-31.

4. Personal communication (U singh 4/ 3/ 20)

5. Booth, A. New Breed or Different Species: Is The 21st Century Health Information Professional Generic or Specific? Presentation at EAHIL Workshop, 12th–15th September 2007, Krakow, Poland

My Experiences With Spinal Cord Injuries

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Spinal cord injury rehab has been an area of interest for me since my PG days. It was 1991, I had just completed my internship, and was sitting in front of Professor Hariharan Sir, on day one of my PG course. After a brief introduction to PMR, Sir took me on a quick tour of the male ward, explaining about disability and what rehab can do to improve their lives. That had a profound effect on me, end result being that an aspiring surgeon got baptised into PMR. I would like to mention two cases of SCI which I distinctly remember among the ward cases, including their names, Mr.V , a 40 plus entrepreneur with T4 complete paraplegia and Mr. D, a 18 yr old C6 tetraplegia. Both these cases had an effect on my initial understanding of the extent of the problems, challenges and complexities of SCI rehab.

Mr.V was admitted with a huge infected sacral pressure sore, three weeks post fixation of spine simply because no nurse did something simple as position change in the orthopaedic wards(they were busy with too many patients) , nor did any doctor advise him about it or his attender to do it (they were also busy with lots of patients). He was malnourished, and severely depressed. After explaining about pressure sore, he had asked me a simple question- None of those doctors or nurses had five minutes to spare to explain about this to me and the care to be taken to avoid it ? Is it also not part of the treatment ? Along with my senior Dr.Sasikumar, we worked on his rehab. Gradually his pressure sore healed, and he was discharged in a wheel chair, partially dependant on transfers, wheelchair self propelled , went back to work in his advertising agency. He got a maruti car modified from the company to enable him to drive. Right from preventive rehab, the poor state of understanding of rehab by fellow specialties, pressure sore care, nutrition, and comprehensive rehab that was a case study for me.

Mr.D a student who got cervical spine injury when he just slipped and fell with a load of grass on his head, ended up with a complete C6 tetraplegia. He slowly accepted the prognosis as days went by, and expressed a desire to write again with a pen, but couldn't grasp and I made a POP splint for him with provision to fix a pen with a rubber band, and he wrote his name with a huge smile on his face , and then wrote "thank you". He was discharged dependant on transfers and self care, wheelchair bound, with an indwelling catheter. His home and surrounding terrain was not wheelchair accessible, rooms had no space for wheel chair mobility. One person

couldn't be staying with him as carer as they were labourers and barely could make ends meet. He came for review three times, with repeated Urinary tract infections, the father progressively feeling defeated as he sold his property and took loans to sustain his treatment. Later I came to know that he succumbed to yet another UTI. It was a revelation on how economic status affects rehab possibilities and how most of our rehab efforts fall apart at the community level and how a large population of spinal injured patients were underserved.

Today, we could search and access any journal or article sitting in the comforts of a room and searching google. It was the nineties, computers were coming into the scheme of things, dot-matrix printers were in use, dial up internet connections were not yet widely available, and literature search meant hours spent in the library. That is what I did along with Dr.Abdul Rasheed, as part of an ICMR project on SCI by Prof (late) Suseelavarma, one of the pioneers in spinal injury rehab in India, who was trained by none other than Prof. Sir. Ludwig Guttman. It was exciting to hear the history of how SCI care evolved over the years and also helped broaden my interest in SCI rehab.

During 1997, on an eight week clinical attachment as part of D.Med Rehab exam, I was at the Spinal Injury center at Sheffield , UK. Funded by NHS, all spinal injury patients were admitted directly to the unit, with the Neurosurgeon called in only for those requiring surgical intervention. The number of patients were significantly lower than in India, care was very organised. There were regular team meetings where every case was discussed, and progress reviewed. Case managers were nurses, and every member contributed, all of them - doctors, allied rehab professionals took their job seriously. Social workers, Psychologists, all worked to ensure smooth continuation of care after discharge in the periphery. Proper case documentation, use of common terminologies, standard scales, Goal setting…It was for me highly organised quality care with a lot of professionalism. Needless to say, it was a very different experience for me.

Working for the Ministry of health in Kuwait for a good part of my professional career till date, most of my experience with SCI rehab is from that part of the world. There were two groups of patients , the Kuwaiti nationals and expatriates. Kuwaiti SCI patients were of younger age group, and mostly as a result of high velocity traffic accident .These cases completed their rehabilitation, had access to the best wheel chairs and could do the necessary home modifications, all funded by Government. They were provided monthly allowance to compensate their loss. These cases were followed up regularly in PMR departments in regional hospitals. Most of the spinal injuries in expatriates were due to fall from height in the construction industry or hit and run accidents. They were provided highly subsidised institutional rehab and almost all of them returned to their countries after treatment, and hence were lost to follow up.

As part of a Canadian accreditation processimplemented across MOH hospitals, quality of rehab care in the outpatient and inpatient setting was analysed. I was part of a large team which undertook this process which stretched through many stages and many years, progressively making changes to the existing system of care. Clinical practice guideline was formulated for spinal cord injury rehab, enabling uniformity in approach to SCI. Essentials of documentation was enforced, including use of standard terminology, and accepted SCI scales. Minimum essential data to be included in both outpatient and inpatient settings were defined. Period of hospital stay was defined for Paraplegia and tetraplegia. Criteria for referral to Urodynamics was also made. Team meeting was introduced, along with the whole rehab team, with goal setting and detailed rehab plan one week after admission . This was followed by monthly review

and pre discharge meeting, and arrangements for follow up . A detailed discharge summary was given to all patients. Incident reporting was insisted for any medical events including DVT, Pressure sore, Urinary infection etc. Regular feedback was obtained from patients undergoing rehabilitation about patient perception of care. This whole process made every rehab professional appreciate the huge difference in quality of care when standardisation was brought into rehabilitation care.

A few Kuwaiti patients had opted to try Stem cell treatment for Spinal cord injury. They had travelled to other countries – like Portugal, China, India for Stem cell treatment. Upon retrospective analysis of these cases, many got minimal change in their sensory status, but no appreciable functional gains. The methodology and protocol of treatment was unclear in most of these cases, and documentation by the treating centerswere poor.

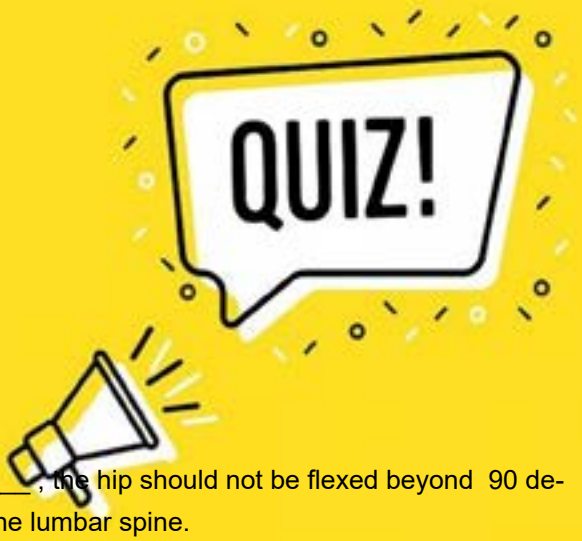
After returning to Kerala, in 2017 and restarting my practice after a nine month break, one of the first SCI cases that I had seen, really disappointed me as a physiatrist. An young man in his late twenties, with T8 ASIA A paraplegia, spinal fixation done in a reputed center a year ago, ended up in my OP with severe spasticity and contractures, bed ridden with multiple infected pressure sores, UTI and was totally dependant on all activities. This happened in Trivandrum city where at least 30 physiatrists practice at present. He was not referred for any rehab, but for "home physio" which was being done (Rs.500 per day) for the past one year! This is not an isolated case and I have seen many patients , some with good potential -bedridden and lost to rehab. Apparently some of the fellow specialists have not caught the bus and is still stuck at the bus stop. While this is an undeniable fact, It is also prudent for us to do a bit of introspection and critical analysis of our work and to how to improve our output.

What can be done to further improve SCI rehab care in the state..? No easy answers, but a few suggestions could be worth consideration. Over the years, a good chunk of the population has been opting for private sector and unfortunately moving away from medical colleges. With this reality in mind, we need to enhance our presence in the private sector. It is heartening to note that many PMR departments have come up in the private sector across the state, and are doing good work too. Also many hospitals and specialists are requesting for PMR department. Although still early, this is a welcome change. PMR is still largely focused on institutionalised care, and we need to devise plans to have an outreach to the community. Is it possible..? We need to ponder and come out with practical ways to address this. A good chunk of patients still are lost in the periphery with no rehab care. Here I respectfully remember the far sightedness of visionaries like Hariharan sir who was pushing for Community based Rehabilitation even in our PG days.

Some points to address

1. Bring out a Practice Guideline for standardisation of care in SCI.
2. Proper documentation of cases, to show improvement objectively and also for other specialists to see and appreciate.
3. Use of common terminology, Functional and outcome scales and measures. (eg. ASIA, FIM/ SCIM, MAS)
4. Project our work and show our output to fellow specialists through multiple avenues- conferences, publications, and social media.
5. Engage with other specialists like Neurology and Neurosurgery
6. Create a database for SCI patients across multiple centers.
7. Create a database of specialised centers for SCI available across the state so that referrals could be made among physiatrists.
8. Plan programs to be in touch with the SCI patient population, and voluntary organisations working in this area to make our presence felt.

A journey of thousand miles starts with the first few steps.



Quiz 2

1.) When examining a patient with an acute injury below ____, the hip should not be flexed beyond 90 degrees as this may place too great a kyphotic stress on the lumbar spine.
(a) T5 (c)T7 (c) T6 (d)T8

2.) The epiconus consists of the spinal cord segments..?
(a) L3-L5 (b) L2-L5 (c) L4-S1 (d) L1- L4

3.) Spasticity in epiconus lesions is seen in all muscle groups except? (a)Knee extensors (b) Hip flexors (c) Toe flexors (d) Ankle plantar flexors (e)Hamstring muscles (f) a & b

4.) Ambulation, in incomplete tetraplegia, can be predicted by having ____ sensation sparing in ____ % or more of the dermatomes in the Lower Extremity?
(a) Pin prick, 20 (b) Light touch, 70 (c) Pin prick, 40 (d) Pin prick, 50

5.) Lesions in patients with incomplete SCI (AIS B through E) were found to have a mean length of </= 20 mm, whereas those in patients with neurologically complete injuries had a mean length of____mm?
(a) 60 (b) 55 (c) 50 (d) 40

6.) (i)Driving a specially modified van is possible for C5 level lesion.
(ii) A lift for access allowing the patient to be fully independent in this activity, is needed.
Which statement(s) is/are true?
(a) I (b)ii (c) both (d) neither

7.) The “3-2-1” bowel program is used for what kind of injuries?
(a) UMN (b) LMN (b) Conus Cauda lesion (d) None of them

8.) Which kind of fractures are most common in SCI patients?
(a) Supracondylar femur fractures (b) Pelvic fractures (c) Distal tibia fractures (d) Proximal tibia fractures

9.) The most sensitive imaging study for diagnosing early Heterotopic Ossification is?
(a) Color Doppler USG (b) MRI (c) Contrast CT (d) Triple phase bone scan

10.) The _____ tract is the main tract for voluntary muscle activity.
(a) Medial corticospinal tract (b) Lateral spinothalamic tract (c) Medial spinothalamic tract (d) Lateral corticospinal tract

Memoirs Of My PG Life

Dr. Nitha J, Consultant Physiatrist, Trivandrum

It has been a cold rainy day, with a hot cup of Nescafe in my hands, my thoughts slowly wandered to my PG life. Those three years, which have left an indelible mark and molded me, right from the inception.

As I stood gaping at the enormous grandfather clock adorning the brick lined walls of the pre independence era building of CMC Vellore, clutching my 1-month old son, a deep sense of excitement laced with fear overcame me. I had been admitted to CMC for their prestigious PM&R Postgraduate program. The three-year roller-coaster ride would go on to widen my horizon and shape my outlook towards life.

My Ego

Days went by thick and fast. The rigors of PG life combined with maternal obligations for a suckling son, was driving me crazy. Amidst those demanding times, the belief that "I am doing a good job; I am treating patients, CURING them, if not fully at least partially: was the silver lining. Or rather it was my Ego, my sense of self-esteem. This false sense of self-importance came crashing down when my professor bluntly said," whatever you do, you cannot improve the neurological level in a complete spinal cord injury. If an ASIA A SCI becomes ASIA C, it's not the greatness of your treatment, it's the natural process". With the realization soon dawning upon me "I cannot Cure", I was left pondering "Why am I treating? what am I doing? Is this what I want?"

As I contemplated on these wise saying of Churchill, "Cure is not the end, but the beginning." I couldn't help but realize that medicine is not just curing the disease, its treating someone's distress, it's improving somebody's quality of life, and it can be even guiding someone to a better life. From that day, my perception changed - patients became "persons," disease became "impairment, activity limitation and participation restriction" and treatment became "optimization and well-being." From a doctor a molding process to rehab physician had started.

Lesson 1: Practicing medicine is not just prescribing drugs and performing surgeries; it's about providing/imparting health (the ultimate sense of well-being).

The Wait

It was raining heavily, and the grandfather clock chimed 10 times. Done with my routine work, the only pending task at hand was Mr. X's sexual rehabilitation. He was a paraplegic with erectile dysfunction. As oral

medications had failed, it was decided to administer papaverine injection. I explained the procedure and the consequences and gave him a shot of intracavernosal papaverine. That night he was given a private room and advised to report to the duty doctor/ nursing station in case of any difficulties. Having informed the duty doctor and in charge nurse, I retired to my quarters. As I walked in, the next morning, the campus was at its full glory, tiny specks of water droplets on the leaves, glistening in morning sun. Seeing me, Mr. X came rushing, in his wheelchair. Looking pale & anxious, he revealed his predicament. He had "priapism." (Priapism is one of the uncommon complications of intracavernosal injections). Stunned, I rebuked, 'Why didn't you inform anybody?'. He whimpered 'I was waiting for you'. This person waited for me with a medical emergency for almost 8 hours! We treated the priapism. Doing the corporal aspiration, my heart was heavy, and a thousand thoughts hit my mind 'why the wait?'.

What went wrong? I had done everything according to the protocol, but my patient was left in distress the entire night. I had expected that he would seek help, and not wait, in case of complications. From that day my approach changed, in every intervention, I tried to cross check for complications personally rather than relying on patient reporting, whenever possible.

Lesson 2: The patients may not report their problems, they may be waiting for their doctor even in distress. It's our responsibility to make sure that they are not waiting.

The End

As the years sped by, I slowly started mastering Spinal cord injury rehabilitation. In all lower thoracic paraplegics, we could attain community ambulation using orthosis and aids with good cardio-pulmonary endurance. They could be completely independent in i-ADLs (instrumented Activities of Daily Living). Bladder management was thorough, included routine ultrasound examinations, cystometrograms and cystoscopies. Most patients were discharged with proper vocational rehabilitation.

One day itstruk me "Are you doing enough?". What is the end point of rehabilitation or when is it a real success? Palliative care ends with death or hospice. What about rehab? The continuum of care; would it be life-long? When can we say that we have optimized all the abilities of a SCI patient? I didn't have an answer. I discussed my mental conflict with my HOD and he left me with another question to ponder, "When can you say, your life is a success?". I struggled with this question for a while, and to me the answer was "if I can bring happiness to other's life, one way or the other, my life is a success."I believe the same is true with rehabilitation. From that day onwards, my definition of optimization of functional capacities included motivating each one of them, to set an example, and equip them to help others who are in same condition.

Lesson 3: The rehabilitation ends when the rehabilitated person himself can be an inspiration to other fellow persons. We should aim for that.

The rumbling of the thunder has broken my reverie. Years passed, since I left the fabled institution after mypostgraduation. The experience from my post-graduate days, have made a mark in my life and how I deal with my patients.

Things Patients Taught Me

Bineesh, Consultant Physiatrist, Trissur

I was more than 30 yrs old, when I got an admission to pursue my post graduate degree in Physical Medicine & Rehabilitation(to be exact, I was 30 years, 3 moths & 3 weeks old). Government Medical College, Kozhikode, had one of the oldest P.M.R departments in the state, &was the ideal place for someone like me to study. I did ask around quite a bit, about the scope of this field, before deciding to settle for it. To this day, I'm eternallygratefful to God & everyone who helped me take this decision.

I had promised myself to take it easy, at least for the first few months of the course. It was fun, strolling around the lush green campus, making new friends, & whiling away the rest of the time reading & listening to music. At the department, the new residents were divided into two units, & were instructed on how they will be expected to learn & eventually help out with OPD & ward work. Besides this, as expected we were expected to attend scientific sessions & conferences.

The course was more fun than I thought it would be. My colleagues were easy going & helpful. My seniors were also extremely supportive, as were the teaching faculty. The department became my home away from home.

One fine day, I heard two of my seniors discussing about an 'orthosis'. My first thought was that it must be the name of a resident who has a brother in the Orthopedics department. The very next day, during rounds, one of the professors, used that word again. I looked around to see if anyone was willing to answer the question, or if the rest of the residents were giggling. Maybe 'Orthosis' is on leave...Maybe orthosis wasn't a nickname…Maybe asking around will seem lame…

My addled brain found relief only when I came upon the definition of an orthosis or an orthotic device- "An external mechanical device applied to a body part to keep it in it's functional or anatomical position ". I was quite fascinated by these devices & did some research, which thanks to Google, lasted less than 10 minutes!

Apparently the oldest orthotic devices were shoes. Nearly 2000 years ago, humans decided to insert layers of wool into sandals to relieve fatigue & foot strain, as they spent most of the day on their feet. The first evidence of the use of spinal orthosis was supposedly by Galen, as far back as 131- 201

AD. In those days orthoses were made from leather, whale bone & tree bark.

The father of prosthetics & orthotics is the French surgeon Ambroise Pare. He had devised everything from metal corsets for treating scoliosis, to orthoses for correcting CTEV. He even designed a cosmetic prosthesis for amputation patients.

Another important name in this field is that of the Welsh

surgeon, Hugh Owen Thomas, who in the nineteenth century devised the Thomas splint, Thomas collar, lower limb & hip orthoses. He is credited with developing the method of examination of deformities in the lying position. Many claim that he was an eccentric with a very short temper, who adopted extreme means to study fracture union, & how it can be modified.

In the 1950s because of the spike in polio cases, lower limb orthoses were in high demand. Metal & leather were used to make orthoses to meet the surging demands. Eventually though, with technological advances, the focus was diverted to make lightweight, durable, patient friendly orthoses.

As a part of my case presentation, around 2 months into my course, I was supposed to read up about orthoses in Spinal cord injury. To be honest, I enjoyed boning up about these devices. Spinal orthoses prescribed after spinal trauma serve one or more of these functions- stabilizing the spine after fracture, limiting spinal motion in cases of sprain or painful conditions, preventing deformity after paralysis & supporting posture, & also for post surgical stabilization.

Besides the spinal orthoses, upper limb & lower limb orthoses are pivotal in improving the status of patients with spinal cord injury. Depending on the level of injury, the type of injury,& stage of recovery, patients with cervical& high thoracic injuries, may need one or more of these upper limb orthoses viz, Balanced Forearm orthoses, Universal cuff, Reachers, Long opponens splint, & Short opponens splint. Lower limb orthoses also play a big role in rehabilitating spinal injury patients, with KAFOs, AFOs doing their job, where appropriate(usually for Thoracic lesions &Lumbosacral lesions). The 'three point control' concept was the basis of all upper and lower limb orthotic designs. In simple terms, a strong force is applied at a joint, & a counter force applied proximal & distal to that joint. The tolerance of tissues to both compressive & shear forces must be understood if the orthosis is to be designed & fabricated safely. There are more than 30 pressure sensitive bony prominences in the wrist, hand &fingers alone!! Higher pressures of 100-300 mm of Hg were tolerated for 2-4 hrs continuously, while lower pressures of 20-50 mm of Hg are tolerated up to 12 hrs a day continuously.

I slowly realized that rehabilitating a patient with spinal cord injury was like putting a jigsaw puzzle together. Treatment using medications (including injections) & prescribing exercise regimes helped in putting two thirds of the puzzle together, but without the proper use of orthoses the puzzle remains incomplete. Again for the puzzle to fall in place, the pieces of the puzzle, in this case orthoses, had to be prescribed with appropriate donning & doffing instructions.

Besides being used in spinal cord injury, orthoses have too many indications in the management of many disease conditions, like stroke, cerebral palsy, rheumatological conditions, sports injuries, & peripheral nerve injuries. Appropriate & timely use of orthoses will help hasten recovery & also improve the functional status of the patient.

While studying for my post graduate degree, I made great friends, had the privilege to be taught by highly qualified & compassionate professors, experienced what it feels like to be loved unconditionally, & learned lessons, that I hope I'llnever forget. One lesson I hope I'll remember, is to stop making assumptions. Another important one was, to keep asking questions & then searching for their answers…

Stone Soup and the KJPMR

Dr Ravi Sankaran, Associate Professor,

You might know this old village tale from Europe.

Some travelers come to a village, carrying nothing more than an empty cooking pot. Upon their arrival, the villagers are unwilling to share any of their food stores with the hungry travelers. The travelers go to a stream and fill the pot with water, drop a large stone in it, and place it over a fire. One of the villagers becomes curious and asks what they are doing. The travelers answer that they are making “stone soup”, which tastes wonderful and which they would be delighted to share with the villager, although it still needs a little bit of garnish, which they are missing, to improve the flavor.

The villager, who anticipates enjoying a share of the soup, does not mind parting with a few carrots, so these are added to the soup. Another villager walks by, enquires about the pot, and the travelers again mention their stone soup which has not yet reached its full potential. The villager hands them a little bit of seasoning. More and more villagers walk by, each adding another ingredient. Finally, the stone (being inedible) is removed from the pot, and a delicious and nourishing pot of soup is enjoyed by travelers and villagers alike. Although the travelers have thus tricked the villagers into sharing their food with them, they have successfully transformed it into a tasty meal which they share with the donors.(1)

The message here should be obvious. With the current times, writing much easier than the 1960’s. As our journal is available at the website, whatever you write is being archived. About 50% of our group has shown willingness to make this the ‘best stone soup ever’. Join your colleagues and contribute to your future, that of PMR in Kerala and India. For those of you knowledgeable of Star Trek I leave you with the words of the Borg.

(Ashliman, D. L. (November 15, 2008). “Stone Soup: folktales of Aarne-Thompson-Uther type 1548”.)

Quiz - KEY

1. (d) When examining a patient with an acute injury below T8, the hip should not be flexed passively or actively beyond 90 degrees as this may place too great a kyphotic stress on the lumbar spine. Therefore, it may only be possible to test hip flexor muscle strength isometrically. While asking the patient to lift the leg straight off the bed, the patient's movement is resisted & the examiner's judgement is required to grade the muscle force as 2 through 5.

2. (c) The segment above the conus medullaris is termed the epiconus consisting of spinal cord segments L4-S1. Lesions of the epiconus will affect the lower lumbar roots supplying muscles of the lower part of the leg & foot, with sparing of reflex function of sacral segments. The BCR & micturition reflexes are preserved, representing an UMN or suprasacral lesion. Spasticity will most likely develop in sacral innervated segments (e.g., toe flexors, ankle plantar flexors & hamstring muscles). Recovery is similar to other UMN spinal cord injuries.

3. (f)

4. (d)Persons with incomplete tetraplegia have a better prognosis for recovery. Upper extremity motor recovery is approximately twice as great in incomplete tetraplegia as in complete tetraplegia, with the potential for varying degrees of lower extremity motor recovery & functional ambulation. Ambulation can be predicted by having pin prick sensation sparing in 50% or more of the dermatomes in the lower extremity(L2-S1).

5. (d)
   [OTHER PREDICTORS OF NEUROLOGICAL RECOVERY]
   An intramedullary hemorrhage correlates with a more severe initial neurologic deficit, & carries a poorer prognosis. Hemorrhage location corresponds anatomically to the level of the neurologic injury. The greater the extent of cord signal abnormality on MRI the greater the chance of having a complete injury. Lesions in patients with incomplete SCI(AIS B through E) were found to have a mean length of </= 20mm, whereas those in patients with neurologically complete injuries had a mean length of 40mm. The presence of cord edema greater than 1 vertebral body segment is also a poor prognostic finding. Smaller degrees of cord edema & especially the absence of abnormal signal in-

tensity on MRI in the spinal cord are considered positive predictors for neurologic recovery. In the chronic stage after SCI, persons with persistent cord signal changes on MRI demonstrate little improvement in AIS grades relative to the improvements of patients with resolution of signal abnormalities.

6. (c)

7. (a) The “3-2-1” program consisting of a stool softener(i.e.,docusate sodium) three times per day, two senakot tablets given 8 hours before the suppository, & one suppository started once bowel sounds are present, is used for managing UMN bowel dysfunction.

8. (a) Supracondylar femur fractures are the most common fractures, followed by the distal tibia, & proximal tibia. Many individuals with SCI have impaired sensation, & thus may not seek medical treatment. Symptoms of acute fracture include fever, acute pain, swelling, or increased spasticity. A plain X-ray will usually give a definitive diagnosis.

9. (d)The incidence of HO, in traumatic SCI, ranges between 13% to 57%, & is usually found in the first 6 months (peak at 2 months) after injury. HO may occur beyond 1 year & is usually associated with a newly developed pressure ulcer, DVT, or fracture. Risk factors for HO include older age, neurological complete lesions, male gender, spasticity, DVT, & pressure sores. These risk factors maybe cumulative. Only joints below the NLI will develop heterotopic bone, with the most common location being the hips (anteromedial aspect), followed by the knees, then shoulders. The pathogenesis of HO is not fully understood, but involves changes of soft tissue mesenchymal cells to osteogenic precursor cells by humeral, neural, & local factors. Bone morphogenic protein is thought to play a key role. A triple phase bone scan is the most sensitive imaging study in diagnosing early HO.

10. (d) The lateral corticospinal tract is the main tract for voluntary muscle activity. Its origin is the precentral gyrus of the frontal lobe of the brain. Their axons descend through the internal capsule to the medulla oblongata. Approximately 80-90% of the axons cross at the pyramidal decussation to the contralateral side of the medulla & descend in the lateral white columns of the spinal cord, in the lateral corticospinal tract.
    At each level of the spinal cord, the axons from the lateral tract peel off & enter the gray matter of the ventral horn & synapse with secondary neurons. The 10-20% of uncrossed axons continue down on the same side of the cord travel in the ventral corticospinal tract. The axons of the ventral tract then cross over at the corresponding level of muscles that it innervates. Both tracts travel from the precentral gyrus to the ventral horn as a single uninterrupted neuron & are termed UMN, while the secondary neurons that they synapse on, are termed lower motor neurons.

Quiz Answers