W26: NeuroUrology- A case studies based approach to investigations and management

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21 October 2014 09:00 - 12:00

Start	End	Topic	Speakers
09:00	09:05	Introduction	• Rizwan Hamid
09:05	09:20	Pathophysiology of neuropathic bladder dysfunction	• Rizwan Hamid
09:20	09:45	Spinal cord injury - upper motor neuron type lesion, case based discussion	• Giulio Del Popolo
09:45	10:10	Spinal cord injury - lower motor neuron type lesion,case based discussion	• Julian Shah
10:10	10:30	Multiple Sclerosis -case based discussion	• Regina Alvarenga
10:30	11:00	Break	None
11:00	11:20	Spina bifida - case based discussion	• Karl Sievert
11:20	11:45	Management of complications - case based discussion	All
11:45	12:00	Questions	All

Aims of course/workshop

The aim of this workshop is to provide a disease specific, case based approach in the diagnosis, management and treating potential complications of the most common neurourologic conditions including spinal cord injury, multiple sclerosis and spina bifida. This will be undertaken by individual case based studies of these diseases in a step wise manner involving the audience in understanding the best management of neuropathic bladder in context of different pathologies. Special emphasis will be on the long term management and treatment of complications. An effort will be made to highlight the important differences in the management of a neuropathic bladder secondary to spinal cord injury vs multiple sclerosis.

PATHOPHYSIOLOGY of Neuropathic Bladder Dysfunction

The coordinated control of bladder and urethral function is a complex process dependent on neural pathways in the brain and spinal cord. This control ensures storage of urine at a low bladder pressure with continence and complete bladder emptying when socially acceptable.

A host of different disease processes can lead to damage to the various different regions that have a role in controlling bladder filling and emptying. Conditions include head injury, stroke, multiple sclerosis, dementia and Parkinson's disease. In general, the severity of the urinary tract dysfunction is reflected in the extent of the general disease process; it is unusual to see a patient with severe urological problems and little evidence of other neurological effects of their brain disorder.

Suprapontine Lesions

Suprapontine lesions can lead to problems with either neurogenic detrusor overactivity (NDO) or urinary retention although the latter is less commonly seen as the presenting problem.

It is important to appreciate that NDO seen with a suprapontine lesion is not associated with detrusor sphincter dyssynergia (DSD). The brain lesion is leading to an alteration in the "set point" at which the micturition reflex is activated but the reflex itself remains coordinated as the spinal cord and brainstem centres are all still intact and able to deliver sphincter relaxation as the bladder contraction occurs. This results in low pressure bladder emptying and allows voiding to completion to occur so that suprapontine lesions are very rarely associated with renal damage as a result of high pressure urine storage and voiding.

Suprapontine lesions may present with a variety of different

symptoms. These include urinary retention, a lack of awareness of bladder filling and emptying, impaired appreciation of the social context of micturition, frequency with urgency and incontinence.

Supraconal Spinal Cord Injury

Injury to the spinal cord can occur through different disease processes. These include trauma, cervical spondylosis, prolapsed intervertebral discs, multiple sclerosis, transverse myelitis, tumours, vascular disease and abscesses. The majority of lesions will be incomplete, that is there will be some preservation of sensation and voluntary movement in the dermatomes and myotomes that are distal to the site of the cord damage. However, the pathophysiological effects of cord injury are best appreciated by considering the patient with a complete spinal cord injury.

We will consider a patient with a cord transaction at the T4 level. The immediate effect of such an injury is a loss of reflex activity in the cord below T4; this is termed spinal shock. They will have a flaccid paralysis and loss of sensation below the injury level. Loss of sympathetic nervous system activity will result in reduced blood pressure due to vasodilatation which will also lead to penile engorgement. Parasympathetic paralysis leads to urinary retention (with eventual overflow incontinence) and an ileus with constipation. Spinal shock resolves over days and weeks as reflex activity returns.

The T4 lesion will, of course, preserve normal spinal cord function above T4 but below the injury level the spinal cord will be viable and functional but disconnected from the higher centres in the brain. The segment below the injury level is called the distal autonomous cord (DAC). Somatic reflex activity in the DAC will lead to increased striated muscle tone and reflexes with associated spasm as a result of stretch reflexes which lack descending inhibitory influences. There will be complete sensory loss from the injury level down.

Reflex activity in the sympathetic system is also uninhibited and this leads to the important syndrome of autonomic dysreflexia. The phenomenon is seen when a noxious stimulus such as bladder distension, constipation or trauma sends a sensory input into the DAC. The response is a mass sympathetic outflow which will result in vasoconstriction in particular. This will decrease the intravascular space and increase venous return to the heart and lead to an increase in cardiac output (Starling's law). The brain and spinal cord above the injury level will respond by increased vagal activity (bradycardia and negative inotropic effects) and vasodilatation above the injury level. For patients with an injury level below T6, the compensatory mechanisms are sufficient to prevent significant blood pressure changes. However, injuries above T6 have a large part of the body under the influence of the DAC and a rather limited area supplied by the normal brain/spinal cord. The distal vasoconstriction cannot be compensated for and there is a rise in blood pressure, which can be severe. The classical picture is of a patient with a severe headache, facial flushing, a cool trunk and lower limbs and a bradycardia. Immediate treatment is required; removal of the noxious stimulus will lead to a rapid resolution of the crisis. If the stimulus cannot be removed then antihypertensive medication must be administered in a way which produces an immediate effect.

Urinary tract reflex activity will also return as spinal shock resolves. Initially bladder filling occurs with normal bladder compliance but then the bladder will contract spontaneously in response to filling – neurogenic detrusor overactivity. This reflex is probably mediated (at least in part) by afferent C-fibres which are not generally involved in normal voluntary detrusor contractions. The response of the distal striated sphincter to the bladder contraction is active contraction rather than relaxation – detrusor sphincter dyssynergia (DSD). This results in a prolonged bladder contraction with obstructed voiding; in men, the bladder neck and prostatic urethra is seen to be widely opened on X-ray screening, with hold up at the distal sphincter.

When the sphincter eventually relaxes some voiding will occur but bladder emptying is often incomplete as the detrusor contraction fades prematurely. Increased intravesical pressures are associated with vesico-ureteric reflux in a minority of patients. It should be noted that the degree of DSD that is seen varies from patient to patient; some patients will have complete reflex bladder emptying with little evidence of any distal sphincter hold up while others will not void at all during episodes of neurogenic detrusor overactivity.

Bowel pathophysiology follows a similar pattern with reflex bowel evacuation occurring but with obstructed defaecation in some patients due to sphincter dyssynergia. Penile erection will occur in response to direct genital stimulation (and psychological stimuli in some cases with injuries below the T11-L2 sympathetic outflow). Reflex ejaculation can be preserved if the injury is above T11 but is usually inhibited so that relatively strong genital stimulation is needed to fire the reflex.

Conus and Cauda Equina Injury

Once again it is apparent that most distal spinal cord and peripheral injuries are incomplete. However, it is helpful to consider the effects of a complete injury in order to understand the pathophysiological effects of damage at this level of the neuroaxis.

There will be a complete sensory loss in the affected dermatomes;; the “saddle area” is supplied by S2 downwards. Somatic motor nerves will have been destroyed (cell bodies are in the anterior horn of the spinal cord) and this will result in a flaccid paralysis. Anal tone will be reduced as a result.

The detrusor will be areflexic but compliance will be reduced in some patients so that the bladder is not necessarily “safe” as resting pressures may be high with a risk of upper tract dilatation in some cases. The distal striated sphincter will be flaccid but the smooth muscle of the distal sphincter will retain tone; in some cases, tone will be low and stress incontinence will be prominent

while in other cases, tone will be relatively high with the potential to allow a poorly compliant bladder to retain urine at high pressures. If smooth muscle tone is high, the condition is described as “non-relaxing urethral sphincter obstruction”.

Reflex rectal activity is absent so that constipation is present. Anal sphincter weakness will lead to faecal incontinence. Impotence is usually present although some residual psychogenic erectile activity may be preserved. There is no reflex ejaculation.

CASE STUDY 1:

SPINAL CORD INJURY – Upper motor neuron type

A 31 years old man sustained a T2 spinal cord injury (SCI). He is wheelchair bound and has been emptying his bladder spontaneously. He complains of recurrent UTI’s and urinary leakage. He is not on any medications.

Q: What is the likely type of injury he has sustained?

A: He has sustained an upper motor neuron (UMN) type injury, with reflex bladder voiding, detrusor sphincter dyssynergia and poorly sustained bladder contractions.

Q: What are the main investigations in this patient that will help you in management?

A: Video-urodynamic studies and ultrasound scan of kidneys.

Q: What are the findings on this VCMG trace? (will be shown during presentation)

A: There appears to be neurogenic detrusor activity. It appears

to be a classical saw tooth pattern. The detrusor pressures are very high and sustained.

Q: What is shown on this picture?

A: This demonstrates bilateral reflux and a trabeculated bladder.

Q: What is the abnormality on the voiding study?

A: This demonstrates a blown up prostatic fossa and a contracted sphincter suggestive of detrusor sphincter dyssynergia.

Q: What is detrusor sphincter dyssynergia?

A: It is defined as involuntary contraction of the urethral and/or periurethral striated muscle simultaneously with a detrusor contraction. This is specific to a supraconal neurological disorder.

Q: How will you treat this patient?

A: The aim is to achieve low pressure storage and complete bladder emptying without incontinence.

One will be guided by the urodynamics result but assuming that this patient with T2 SCI has neurogenic detrusor overactivity and DSD, he will be started on anticholinergic medications and institute a program of clean self intermittent catheterization (CSIC). He will be closely monitored and will undergo ultrasound scan of the kidneys and repeat urodynamics to ensure the bladder pressures have come down. Assuming that there are no problems i.e. UTI;s or problems with CSIC he will be reviewed yearly.

Q: He comes back and although he was bothered by UTI’s he doesn’t like CSIC and wants to know other options?

A: Apart from CSIC the other options include:

- Behavioural & timed void – not suitable for him

- Indwelling catheter – Suprapubic or urethral – not acceptable to him as he is very young and mobile
- Urethral stents / sphincterotomy with sheath - again not acceptable to him
- Augmentation cystoplasty with or without Mitrofanoff but will need to perform CSIC (almost 100% certain)
- Sacral anterior root stimulator (SARS) – not suitable for him as he has an incomplete SCI. This is an option for wheelchair bound patients who have complete injury as it requires a posterior rhizotomy to abolish NDO. This will also lead to a loss of reflex erections.

Q; What are the pros and cons of SARS?

A: The benefits include:

- Abolition of reflex bladder
- Increased bladder capacity
- Abolition of autonomic dysreflexia
- Bowel management

However the downsides are:

- Stress incontinence
- Loss of reflex erections
- Loss of reflex ejaculation
- Loss of reflex defecation

Q: What are the problems with indwelling catheters?

A: They include

- rUTI's
- Blockages
- Need for regular changes
- Stones
- Risk of cancer

Q: What are the complications of stents in his case?

A: He will be incontinent and will have to wear sheath with risk of detachment. The stents can dislodge, block and there is a risk of encrustation. External sphincterotomy is the standard alternative to the use of a stent for patients with problematic DSD who wish to use penile sheath collection, but is irreversible.

Q: He can perform CSIC if urinary leakage can be controlled but feels anticholinergics are not helpful in the absence of infections?

A; I think he has uncontrolled NDO and I will suggest intravesical botulinum toxin.

Q: What type of botulinum toxin is normally used?

A: Botulinum toxin A is used in urology.

Q: What's the efficacy and side effects of botulinum toxin?

A; The efficacy ranges from 36-89% (mean 70%). The effects last from 4-10 months (mean 6 months). The side effects include pain, UTI (<5%), bleeding (<5%), no benefit, need for further injections, need for self-catheterization (very variable 4-45%). In NDO it is more than 50%. The general effects include flu like symptoms, dry mouth and malaise. It is an unlicensed indication.

Q: How does it work and how you give the injections?

A: It is thought to work by blocking the presynaptic release of acetylcholine at the neuromuscular junction. It achieves this by blocking the SNAP 25 protein. This is the basis of decrease in detrusor pressures and phasic contractions in both idiopathic and neuropathic bladders. However, the patients also report a significant decrease in urgency and hence, it is suggested that botulinum toxin also modulates the sensory pathways. This is thought to work by acting on P2X receptors.

It can be administered under general or local anaesthesia. In NDO 300IU (now 200IU) mixed in 30 mls of normal saline are injected at 30 sites sparing trigone. The injections can be submucosal or intra-detrusor.

Q: Are you aware of any long term effects of repeated injections or Botox loses its efficacy after repeated injections?

A: No significant bladder fibrosis has been reported on histological examination after repeated injections. Also up to 9 repeat injections have not demonstrated any decrease in efficacy of botulinum toxin.

Q: is there a role of sacral neuro-modulation (SNS) for treatment of NDO with incontinence in this patient.

A: Recently, it has been shown that SNS might be beneficial in controlling NDO in SCI patients but this is still in investigational stage.

Q: What is the basis of SNS

A: The basis is continuous use of mild electrical activity to stimulate the sacral nerves to the bladder and pelvic floor which in turn activate or inhibit neural reflexes.

Q: How is the SNS delivered?

A: Generally a 2 stage technique is used as it has been shown that the 2 stage method improves the rate of positive tests from 50% to 80%. This is a minimally invasive procedure and can be performed under general or local anaesthesia. Initially a test implant is inserted into the S3 foramina. The patient keeps a symptoms diary for 2 weeks and this is compared with the pre-operative evaluation. A greater than 50% benefit in symptoms entitles the patient to have the second stage (permanent implant) fitted.

Q: What is the efficacy / side effects and complications of SNS?

A: It is thought to be effective in 60-70% of cases. The effects last for 3-5 years. Occasionally, the patient complains of pain at the site of neurotransmitter implant. They can also develop leg pain. The explantation rate is 10%. This is mainly due to infection or lack of sustained efficacy.

Q: What is a clam cystoplasty (see picture) and how it works?

A: The principle is to bivalve the bladder like a clam and patch the defect with a piece of bowel, generally ileum. This increases the capacity of bladder and lowers the intra detrusor pressures. Additionally, it decreases the amplitude of contractions by preventing sustained detrusor contractions.

Q; What are the contra-indications to a clam?

A: Severe inflammatory bowel disease i.e. Crohn’s disease, post surgery or radiotherapy critically short or abnormal bowel. Patients’ unwillingness or inability to perform self-catheterization.

Also, significant renal or hepatic impairment as it may result in an inability to cope with metabolic consequences of bowel in urinary tract.

Q: What are the potential complications of clam cystoplasty?

A: This includes:

- Mucus: the average daily production from the incorporated bowel segment is 35-40 grams. This does not decrease over time. This can lead to infections, stone formation and blockages. Bladder washouts might be required with acetylcysteine to dissolve excess mucus.

- Bacteriuria & UTI: Almost 100% of patients will have asymptomatic bacteriuria. The incidence of clinically significant UTI is around 4-43%.

- Biochemical abnormalities: The presence of absorptive bowel in the urinary tract leads to reabsorption of ammonium chloride and excretion of bicarbonate resulting in acid base imbalance. This is clinically important in very few cases. These patients can develop hyper-chloraemic acidosis. The treatment is administration of bicarbonate in about 15% of cases.

- Intermittent catheterisation rate: The rate is around 50% in idiopathic patients but near to 100% in the neurogenic patient.

- Stones: The reported rates are highly variable between 0-53%. It is generally thought to be around 15%. They are more common with an associated Mitrofanoff procedure.

- Perforation: Spontaneous perforation is a rare complication (<1%) but carries a mortality of 25% mostly due to delay in diagnosis.

- Cancer: There is an increased incidence of cancer in augmented bladders. However there is a long latent period (>10 years). This is associated with chronic inflammation, urinary stasis and recurrent UTI’s. The tumors are generally adenocarcinomas and in the region of

anastomosis. The mechanism seems to be related to bacteriuria. This leads to reduction of urinary nitrates to nitrites by colonic bacteria. This reacts with urinary amines to form N-nitrosamine, implicated in carcinogenesis.

- Bowel changes: This usually results in diarrhea. The symptom is troublesome in up to 30% of cases. Also there can be a decrease in absorption of B12 and folic acid leading to neurological complications.

Q: How do you follow up a patient with clam cystoplasty?

A: Once stable the patients are seen on a yearly basis with ultrasound scan of kidney and an x-ray KUB. The biochemical analysis includes evaluation of kidney and liver function and estimation of serum chloride, bicarbonate, B12 and folic acid. They are advised to contact the department urgently if they develop recurrent UTI's, haematuria significant weight loss or severe lethargy. They will undergo yearly surveillance cystoscopies 10 years after operation. However it seems that the rate of pick up by this method is questionable.

Q: What are the main complications with UMN type SCI?

A: This is NDO with DSD leading to rUTI's, low bladder capacity with urinary incontinence, reflux with scarring and deterioration of upper tract function. However, the most dangerous problem at present is autonomic dysreflexia (AD).

Q: What is AD?

A: It is sudden disordered sympathetic activity due to a specific stimulus below the level of SCI in patients with an

injury level above T6.

Q: What are the causes of AD?

A: It can be nociceptive stimulation in the region of the distal autonomous cord. In the majority of cases the trigger is an overfilled bladder, constipation or distal skin infection.

Q: What is the mechanism of AD?

A: There is sympathetic over activity of the distal autonomous cord leading to vasoconstriction in the territory supplied by the sympathetic nerves originating from the distal autonomous cord with compensatory vasodilatation of the normally innervated sympathetic territory

Q: What happen below the level of SCI A: These include

- Increasing vasoconstriction
- Decreasing compensatory vasodilatation
- Splanchnic venoconstriction
- Increased venous return to heart
- Increased cardiac output with increased systemic resistance
- Severe hypertension
- Compensatory bradycardia

Q: What does patient experience?

A: The patient complains of the following symptoms

- Headache (usually)
- Hypertension
- Bradycardia
- Sweating and flushing

Q: What is the treatment?

A: this is a life threatening condition. The management includes:

- Remove cause (drain bladder, evacuate bowels, examine toes)
- Sit up the patient
- GTN spray
- S/L Captopril or IV Labetolol, Phentolamine

CASE STUDY 2:

SPINAL CORD INJURY – Lower motor neuron type

A 38 years old man sustains a L3 SCI.

Q: What is the likely clinical picture of this type of injury?

A: Assuming that there has been damage to the conus, this will be a lower motor neuron (LMN) type SCI. The features include:

- Flaccid paralysis and sensory loss
- Absent conus reflexes.
- Detrusor areflexia or reduced compliance.
- Sphincter weakness or non-relaxing urethral sphincter obstruction.

Q: What do you know about detrusor leak point pressure (DLPP) & abdominal leak point pressure (ALPP)

A: DLPP is the lowest detrusor pressure at which urine leakage occurs in the absence of either a detrusor contraction or increased abdominal pressure. McGuire observed in spina bifida patients that if this is greater than 40 cmH2O then there is a higher risk of damage to upper tracts.

The ALPP is the intravesical pressure at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction. If this is greater than 150 cm H2O then the urethra is unlikely to be the cause of urinary incontinence.

Q: How will you then manage this patient?

A: If compliance is normal he will have a generally safe bladder and the options include:

- Behavioural & timed voiding in incomplete lesions

- Emptying by straining if sphincter weakness allows the bladder to empty effectively in this way
- CSIC if incomplete voiding or has UTI's If he complains of urinary incontinence then the treatment options include:
    - Sheaths
    - Bulking agents
    - Slings
    - Artificial urinary sphincter (AUS)

Q: How many components are there in an AUS?

A: Three. Cuff, reservoir and control pump

Q: What is the success rate of AUS?

A: It is around 80% at 10 years. However it is less in SCI patients especially if they are wheelchair bound.

Q: What are the complications of AUS?

A: They include

- Infection & Erosion
- Persistent leakage
- Mechanical failure

CASE STUDY 3:

MULTIPLE SCLEROSIS

A 48 years old lady has been diagnosed with Multiple Sclerosis about 3 years ago. She presents with worsening lower urinary tact symptoms. Her main complaints are frequency, urgency, nocturia with occasional urinary incontinence. She also suffers from urinary tract infections from time to time.

Introduction

MS affects women three times as often as men. The clinical picture depends on the distribution of demyelination in the nervous system with spinal cord disease being particularly associated with urinary tract symptoms. Progression is very variable and follows different patterns (e.g. relapsing/remitting and primary or secondary progressive disease). Acute deterioration can occur in response to urinary tract infections.

The commonest pattern of lower urinary tract dysfunction is a combination of neurogenic detrusor overactivity and impaired bladder emptying due to detrusor sphincter dyssynergia and poorly sustained detrusor contraction. The severity of the urological symptoms is broadly correlated with the overall level of disability.

Clinical Assessment and Management

This must include a general assessment (cognitive ability, hand function, mobility etc.) and enquiry into urinary, bowel and sexual

symptoms. If the patient is not catheterised, a urine sample should be tested in order to look for evidence of infection.

The ambulant patient most commonly presents with urgency, frequency and incontinence. Problems with bladder emptying are also commonly reported. Specific investigation can be limited to a measurement of residual urine volume. A classic algorithm is in use which suggests that intermittent self catheterisation (ISC) be introduced if the residual is over 100ml. Anticholinergic drugs are used if there is urgency present either in the patient with good bladder emptying or despite bladder emptying issues being addressed by ISC. The success of this basic approach is dependent on the expert assistance of specialist nurses.

In patients with persistent symptoms, it is advisable to undertake urodynamic studies in order clearly to define the underlying pathophysiology.

Intravesical injections of botulinum toxin have established a clear role in managing persistent detrusor overactivity, although virtually all MS patients will need to self-catheterise after treatment.

Desmopressin has been shown to be useful in reducing nocturia although the usual precautions need to be taken in order to detect hyponatraemia which will affect a minority of patients who are on long-term therapy.

Surgical treatment has a very limited role in treating persistent detrusor overactivity in patients with MS. Neuromodulation (sacral nerve stimulation) has been reported to be effective in some patients but the evidence base is extremely weak. Cystoplasty is rarely indicated as future neurological deterioration may interfere with the ability of the patient to self-catheterise.

Containment of incontinence is an important aspect of care in the patient with more advanced disease. Pads in women and the use of penile sheath collection systems should be introduced

with the support of continence nurse specialists.

Suprapubic catheterisation is needed in many patients either because bladder emptying is not adequately addressed with ISC or because incontinence cannot be contained. It is important that patients and carers do not fall into the trap of expending excessive amounts of time and energy in trying to avoid catheterisation, as effective and convenient bladder drainage can frequently be achieved by insertion of a suprapubic catheter. A catheter valve should be offered as many patients prefer to use a valve rather than a leg-bag during the day; urine storage problems can be addressed by using anticholinergics, botulinum toxin or urethral closure.

For the catheter-intolerant patient with repeated blockages, frequent infections or pain which does not respond to standard measures, an ileal conduit diversion may be needed. In such patients, a decision has to be made as to whether to undertake a simple cystectomy at the same time as the diversion by way of prophylaxis against the development of pyocystis.

Bowel management is also important. Patients with MS usually have intact sacral reflexes; this allows a regime that involves stimulating a reflex bowel evacuation at a convenient time (typically alternate days). Firstly stool consistency and bowel motility are addressed with oral laxatives and, secondly, bowel evacuation is triggered by rectal stimulation, suppositories or enemas. An alternative is to use rectal irrigation.

Sexual dysfunction should also be addressed. Management of erectile dysfunction follows conventional lines.

CASE STUDY 4:

CAUDA EQUINA SYNDROME

A 33 years old fit builder develops sudden onset of lower back ache after a period of heavy lifting. He starts to develop numbness in lower limbs spreading to perineal area. He takes pain killers but the symptoms do not settle and over the next few days he starts to develop urinary and bowel dysfunction. An emergency MRI reveals slipped disc at L5/S1 level. He undergoes emergency decompression. The recovery is ok for limb numbness but he cannot empty the bladder and is dependent on self catheterization.

A classical cauda equina syndrome (CES) arises from compression of the nerve roots that are passing along the spinal canal in the lumbar region. Nerves that will be affected include:

- Nerves to skeletal muscle - Compression of these nerves will result in muscle weakness, typically affecting the lower legs, toes and anal canal regions. Absent reflexes may also be detected.

- Somatic sensory nerves - These nerves provide sensations over the skin of the saddle area in particular. This includes the perianal region, the perineum, the back of the legs and the feet.

- The innervation of the lower urinary tract - This will classically result in retention of urine with damage to the nerve supply

to the external urethral sphincter resulting in stress incontinence. There will also be a loss of sensation of bladder filling and voiding.

- The innervation to the lower gastrointestinal tract - Damage to these nerves will result in constipation. Weakness of the anal sphincter may result in faecal incontinence. There will be an absence of bowel related sensation.

- Damage to the innervation of the genital tract - This will result in an absence of penile erection along with absent ejaculation and a loss of sense of orgasm and other sexual sensation.

The commonest presentation of CES is an acute central disc prolapse in the lumbar region. Severe pain will be accompanied by neurological symptoms and signs and by urinary retention (and/or incontinence) with constipation. This is a neurosurgical emergency that mandates investigation with an immediate MRI scan of the spine and surgical decompression.

Diagnostic difficulty can arise if the onset of symptoms is insidious and the syndrome incomplete.

Clinical Assessment and Management

Patients with CES are often discharged from spinal surgery units soon after undergoing spinal decompression. Too often, there is an underestimation of the degree of disturbance to pelvic organ function and the patient will be left with life-changing symptoms that have not been adequately addressed. It often falls to the urologist to pick up on these issues. Admission to the regional spinal injuries unit is worth considering as specialist nursing and medical input is important.

The patient will usually either be in a degree of chronic retention

or be emptying by straining. Stress incontinence is very likely to be present. ISC should be discussed and trialled. Urodynamic studies should be performed. If the patient remains wet due to stress incontinence while on ISC, surgical treatment should be offered with an artificial urinary sphincter in men and either an artificial sphincter or pubo-vaginal sling in women. It is important to appreciate that a patient who is reliably dry will inevitably need to self- catheterise; if bladder emptying is achieved by straining, some degree of stress incontinence will always be present and the trade off that the patient faces is that treatment of incontinence will necessitate a switch to ISC.

Urine storage at low pressure cannot be guaranteed in patients with CES as poor compliance is a feature in a minority of cases and patients with incomplete lesions may have neurogenic detrusor overactivity. Poor compliance is most reliably dealt with by augmentation cystoplasty; there is limited data available on the effect of botulinum toxin injections on bladder compliance and long-term, reliable restoration of safe urine storage probably cannot be relied on. An alternative to cystoplasty in the patient with a poorly compliant bladder is suprapubic catheterisation, with the catheter maintained on free drainage in order to keep bladder pressures low.

Patients who have problems with urethral ISC due to physical or psychological difficulties with the technique should be offered a continent, catheterisable abdominal (Mitrofanoff) conduit.

If bladder compliance is normal but a patient elects to use a long-term suprapubic catheter to empty the bladder, a catheter valve should be offered. Stress incontinence may need to be treated in order for the patient to be dry while the valve is in use.

Bowel problems are typically of constipation and stress faecal incontinence (embarrassing loss of flatus and anal seepage are particularly problematic). Patients may find an anal plug helps to contain seepage. Manual evacuation of faeces by the patient is the mainstay of management in this group of patients; dietary

adjustments and the use of oral agents may be helpful as well. Rectal irrigation is finding an increasing role. A minority of patients with CES will find their bowel problems so difficult to manage that a colostomy is requested; a transverse colostomy may be preferred to a sigmoid stoma as the left colon will be denervated and a sigmoid colostomy may be constipated as a result.

Impotence and lack of orgasm are additional problems that need to be considered. Once again, management of erectile dysfunction follows traditional lines.

PRINCIPLES OF MANAGEMENT OF A NEUROPATHIC BLADDER

The main principle of management of a neuropathic bladder is to assure that the detrusor pressure remains within safe limits during both the filling phase & the voiding phase with the aim of:

1. Protecting of the upper urinary tract
2. Improving of urinary continence
3. Improving the patient's quality of life
4. Restoring normal (parts of) lower urinary tract (LUT) function

When treating patients with neurogenic lower urinary tract dysfunction (NLUTD) it is important to consider any patient's disability, cost-effectiveness of the treatment, technical intricacy

of administrating the treatment and any possible complications.

The treatment of NLUTD falls into the following main categories:

- Behavioral approaches
- Lifestyle interventions
- Pads, portable urinals
- LUT rehabilitation
- External appliances:

  clean intermittent catheterization

  condom or Foley catheterization for patients with incomplete bladder emptying (e.g. elevated PVR)
- Pharmacotherapy Antimuscarinic anticholinergic agents are the standard therapy
- Surgery

  Intradetrusor Botulinum toxin injections

  External Sphincterotomy

  Bladder augmentation

  Bladder reconstruction

  Urinary Diversion

The choice of treatment depends on which LUTS is being treated i.e. storage, voiding and/or post-micturition symptoms.

SUMMARY AND TAKE HOME MASSAGES

The management of a neuropathic bladder secondary to various neurological conditions has to be individualized not only according to the disease process but also keeping in mind the patient's desires and limitations of the healthcare system. The overall principles of managing a neuropathic bladder are fairly standardized as per EAU & ICI guidelines.

Recommended Reading & References

1 - A UK consensus on the management of the bladder in multiple sclerosis. CJ Fowler, JN Panicker, M Drake, C Harris, SCW Harrison, M Kirby, M Lucas, N Macleod, J Mangnall, A North, B Porter, S Reid, N Russell, K Watkiss and M Wells. Journal of Neurology, Neurosurgery and Psychiatry 2009, 80, 470-7.

2 - British Association of Urological Surgeon's suprapubic catheter practice guidelines. SCW Harrison, WT Lawrence, R Morley, I Pearce and J Taylor. BJU International 2011, 107, 77-85.

3 - International Consultation on Incontinence – 2012

4 - European Association of Urology Guidelines -2013