

# 24<sup>th</sup> May 2016

# Volume 13 Issue 16

**i-Telerad** A reminder that if there are issues with reporting of films there are radiologists available to discuss reports with the refers and other clinical issues. The numbers are on the bottom of the request forms and are 1800 009 945 or 03 85875300. Please Michele Harris, Glen Burt in Radiology or one the ED SSs know if there are any ongoing issues with this or anything to do with the service that you need them to escalate.

**Transfers to Delivery Suite** – There is a new policy for patients over 20 weeks who present with a pregnancy related issue who are transferred directly to Delivery Suite. They should be triaged and coded as Transfer Other Facility with a freetext comment that they are transferred to Delivery Suite. However there are some exclusions who should be treated in ED. These include:

- 1. Advanced Labour / foetal parts on view
- 2. Active bleeding, other than vaginal bleeding
- 3. Trauma presentations
- 4. Chest pain
- 5. Systolic BP >160 and/or Diastolic  $\ge$  110
- 6. Altered GCS
- 7. Respiratory / circulatory compromise
- 8. Superficial lacerations and minor trauma
- 9. Drug overdose
- 10. Psychiatric disorder

For a full view of the policy go to SUTED folder under Clinical – Specialities - O&G and also Clinical - Protocols

**Outpatient Tx of lower limb DVTs** – There is a also a new clinical policy for outpatient treatment of lower limb DVTs. To access the full document go to the SUTED folder (on desktop of PCs) – Clinical – Pathways – DVT.

The policy has inclusions / exclusion and the information sheets mentioned are at the end of this policy, so it needs to be viewed in entirety but Andrew has summarised the main points in a cheat sheet:

## RIVAROXABAN

- 1. Patient suitable for Rivaroxaban (NB contraindicated in pregnancy)
- 2. Discuss with Haematologist on call
- 3. Give patient 15mg Rivaroxaban
- 4. Issue RIvaroxaban take home pack (4 x 15mg tablets) located in Acute Drug Room (on top of shelves on right)
- 5. Patient to go to GP within 48 hours
- 6. GP to prescribe Rivaroxaban 15mg bd for 3 weeks, then 20mg daily
- 7. Make follow up appointment with Haematologist on call within next 2-4 weeks
  - a. Chong, Hugman, Lee 9113 2513
  - b. Badoux, Ho, Roncolato 9113 3943
  - c. Ramanathan 9540 8140
- 8. Patient given information sheet

#### WARFARIN

- 1. Patient suitable for Warfarin (NB contraindicated in pregnancy)
- 2. Discuss with Haematologist on call
- 3. Commence enoxaparin (1.5mg/kg daily, adjust for eGFR))
- 4. Continue for minimum 5 days AND until a therapeutic INR reached (2-3)
- 5. Issue prescription for intervening days (Enoxaparin easily obtained from Community Pharmacists)
- 6. Continue until GP or Southcare review (within 2-5 days)
- 7. HITS screen at day 5 or 6
- 8. HITS screen at day 12-14 if still on enoxaparin
- 9. Issue Warfarin take home pack
- 10. To take 5mg Warfarin daily over next 2 days
- 11. Ongoing warfarin dose to be determined my GP
- 12. Make follow up appointment with Haematologist on call within next 2-4 weeks
  - a. Chong, Hugman, Lee 9113 2513
  - b. Badoux, Ho, Roncolato 9113 3943
  - c. Ramanathan 9540 8140
- 13. Patient given information sheet

#### THIS WEEK

Epidura	al Abscess
Next W	/eek's case – Vertigo + Nystagmus
Joke / (	Quote of the Week
The We	eek Ahead

## EPIDURAL ABSCESS

#### Two cases

A 60yo man with a Hx of Ht'n, hypercholesterolaemia and PAF (on warfarin) presents with 2 weeks of non-traumatic gradual onset constant upper thoracic (~T1-T3) back pain with radiation across his scapular region & down his left arm to the elbow No diabetes, immunosuppression, travel, occupational or social risks.

afebrile – other obs normal - HS dual no murmurs including aortic regurg- no stigmata SBE –no pulse deficit. Neurologically N including his legs and perianal region. No skin changes. Bloods including WCC normal .What could be going on? What are you going to do?

Two days later he returns as the pain has increased and now runs down his right arm to, but not including, the hand. He has recently seen his GP for these "myalgias" and was noted to have a low grade temp – commenced on tamiflu.

On exam T 38C- exam as above – normal WCC 9 – what are you going to do now? DDs? Ct thoracic spine and chest for ? epidural haematoma both normal – admitted for observation. However next day he developed weakness in his right arm and leg. Ct head N – MRI of spine showed spinal epidural abscess from C2-T2

Urgent decompression and drainage with significant return of neurological function. Swabs and blood cultures grew Staph Aureus. In retrospect he reported problems over the years of recurrent folliculitis / small pustules.

What do we need to know about spinal epidural abscesses?

When a patient presents with back pain with or without neurological symptoms we should consider all the different potential causes and pathology. Remember from your pathology- inflammatory, infective,

tumour, trauma etc – and we should be looking at the age, background of the patient, history and exam to sort out the likely aetiology.

**Remember "Red flags ".** The NSW Emergency Care Institute (ECI) site which is accessible via "Links" in CIAP has a list of these red flags which should heighten youor concerns when a patient presents with **acute low back pain** - indicating need to senior review, further investigations and earlier consults. They include:

- History of cancer (including multiple cancer risk factors with no current diagnosis)
- History of significant trauma
- Intravenous drug abuse (remember to be sceptical in patients who deny recent IVDU in context of previous use)
- Recent bacterial infection or fever
- Immune suppression (HIV, transplant, corticosteroids)
- Unexplained weight loss
- Saddle anaesthesia
- Bladder dysfunction (urinary retention or incontinence)
- Bowel dysfunction (faecal incontinence)
- Neurological deficit in either or both lower limbs (especially if progressive)
- Severe pain when supine and/or at night
- Persistent symptoms for >4 weeks (recurrent presentations, worsening pain)
- Pregnancy.

You'd also add on a couple of other flags:

- Recent surgical, or dental procedures which may have resulted in bacteraemia
- Anticoagulants / antiplatelet agents concern re epidural or retroperitoneal haematoma
- Back pain with chest, abdo or pelvic pain concern re AAA, dissection, etc
- New non-lumbar pain esp thoracic
- Age < 16
- Representations

One cause of back pain that occasionally presents to the ED is the **epidural abscess**, which if missed can lead to devastating consequences. It has a 45% morbidity rate and 15% mortality, partly related to it being associated with multiple ED visits with diagnostic delays (in 68% of patients).

Predisposing factors include spinal surgery, recent trauma, immunosuppression, a distal site of infection and intravenous drug use; however, **20% of patients will have no clear predisposing factor**.

Predisposing factors	Frequency, %
Infections	44
Skin abscess	
Vertebral osteomyelitis/discitis	
Pulmonary/mediastinal infections	*
Sepsis	*
Immunodeficiency	34
Diabetes mellitus	
Intravenous drug use	
Alcoholism	
Chronic renal failure	*
AIDS	
Spinal procedure or surgery	22
Trauma	10
No predisposing factors	20

Most common between the ages of 30 and 60 (in the past most commonly seen in  $6^{th}-7^{th}$  decades), it is seen more commonly in males at a ratio of almost 2:1

Related to t	these pred	isposing facto	ors are the	most common	organisms isolated:

Organism	Frequency, %
Staphylococcus aureus	63
MRSA	15
Gram-negative bacilli	16
Streptococci	9
Coagulase-negative staphylococci	3
Anaerobes	2
Others	1
Unknown	6

The organism also ties in the primary source of the infection which has lead to bacteraemia or local invasion.

Primary sources of infection in spinal epidural abscess

Source of infection	Median (%)	Range (%)
Skin and soft tissue	18	7-45
Urinary Iracl	10	2-36
Previous sepsis of unknown origin	8	5–11
Respiratory tract	5	3–16
Abdomen	4	2–11
Endocardilis	3	1–8
Infected vascular access	2	1–8
Dental abscess	2	1-11
Ear, nose and throat	2	= 1 <b>-</b> 11

The epidural space is a continuous vertical casing located outside the dura mater and within the wall of the bony vertebral canal. This space is normally occupied by fat, areolar tissue and a network of veins. Anterior to the spinal cord, the dura adheres to the vertebral bodies, leaving only a potential space. As a result, 80% of spinal epidural abscesses lie posteriorly.

Within the epidural space, longitudinal extension is common; abscesses often involve 3 to 5 spinal cord segments and can affect the whole length of the spinal cord.

Spinal epidural abscess (SEA) most often starts in the thoracic (50%), followed by the lumbar (34%) and cervical regions (15%).

The effects of SEA progress locally, from reversible neurological deficit and destruction within the spinal cord, to permanent central nervous system dysfunction and death. The underlying pathophysiologic mechanisms are poorly understood, because neurologic dysfunction is often disproportionate to the degree of spinal cord compression. There is a hypothesis that oedema and inflammation involving the cord and epidural space eventually involve the epidural venous plexus, compromising the circulation and causing cord ischemia. Most likely, the synergistic effects of compression and ischemia are responsible for the neurological deterioration seen with SEA.

#### **Clinical diagnosis**

Spinal epidural abscess is characterized by the classic triad of localized back pain, neurological deficits and abnormal inflammatory parameters (fever, leukocytosis and elevated ESR.

However, a recent study demonstrated that the "classic triad" was present in only 8 (13%) of 63 SEA patients.

Subsequently most of these patients suffered diagnostic delays as a result and two-thirds required multiple ED visits (from 1 to 8 visits – mean 2 visits) before a diagnosis was made.

During these delays, 57% experienced further neurological deterioration, and residual motor weakness was 3 times more likely (45% v. 13%) in patients with diagnostic delays. One study that only 39% of 188 SEA patients made a full recovery if treatment was delayed more than 36 hours after the onset of motor weakness.

In more detail symptoms include the following:

- Fever, present in **only one third** of patients in one study
- Localized back pain in the majority, usually the first symptom
- Radiculopathy with radiating or lancinating pain, including truncal girdle pain (This, at times, may simulate myocardial infarction or other causes of chest or abdominal pain.)
- Spinal cord syndrome, typically involving paraparesis with prospective progression to paraplegia: Epidural abscesses at the level of the cauda equina cause symptoms consistent with cauda equina syndrome rather than a spinal cord syndrome.
- Sphincter dysfunction

• Headache and neck pain: These may be present, especially with cervical epidural abscesses. (Of course, these symptoms also suggest meningitis.)

Note the progression of the symptoms is generally more rapid in those cases where there is haematogenous seeding (primary SEA) in comparison to where the abscess is secondary to local infections such as osteomyelitis.

Physical findings vary with the degree of spinal cord compression or dysfunction.

- In the most advanced cases, a transverse cord syndrome is seen with motor, reflex, and sensory levels found upon neurologic examination.
- Localized tenderness to percussion or palpation at the site of the abscess may be noted. Paraspinal muscle spasm may be present.
- Signs of spinal cord dysfunction may be observed.
- Complete transverse spinal cord syndrome with paraplegia and sphincter dysfunction
- Incomplete spinal cord syndromes
- Reflexes may vary from absent to hyperreflexia, with clonus and extensor plantar (Babinski) responses. Areflexia may indicate spinal shock with transient inhibition of spinal reflexes.
- Nuchal rigidity may be present, particularly with cervical epidural abscesses.

Because of this inability to rely on the triad, some suggest to use risk factors to screen ED patients for SEA, since they are more sensitive (98.4% v. 7.9%). These authors also note that the ESR is more sensitive and specific than the total white blood cell count (abN in 60% only). What about CRP? – One study 87% of patients with SEA had elevated CRP levels as compared with 50% with spine pain not due to an SEA.

Blood culture +ve in ~ 60%.

As in many diseases the errors arise from not looking rather than from not knowing and thus the most important step in diagnosing SEA is consideration of the entity.

Imaging - Radiological modalities are the basis for SEA diagnosis.

MRI (especially with gadolinium enhancement which allows the differentiation of pus from CSF ) with 91% sensitivity, is now considered the diagnostic gold standard. Although CT allows better bony visualisation, the MRI is superior to CT because it better delineates SEA from contiguous structures and differentiates SEA from spinal tumours, hematomas, transverse myelitis, spinal cord infarction and intervertebral disc prolapse. Note the sensitivity of CT without myelography is poor for SEA and you **cannot rely** on CT to rule this out.

**Image the entire spinal column** even when patients have focal signs or symptoms in one region because multiple skip lesions are common and patients may not have pain or tenderness in all of the affected areas

Plain radiographs of the spine may reveal changes of osteomyelitis or discitis but are rarely diagnostic of SEA.

Lumbar puncture is now regarded as an unnecessary procedure likely to increase the risk of spreading the infection to the subarachnoid space.

**Therapy** - Rapid treatment improves outcomes. When SEA is clinically suspected, even prior to diagnostic imaging, immediate ED administration of dexamethasone is recommended to minimize the progression of compressive oedema, cord ischemia and neurological damage.

Parenteral antibiotics effective against staphylococci, streptococci and Gram-negative bacilli should be initiated as early as possible. Appropriate initial empiric regimens include flucloxacillin with metronidazole and either cefotaxime or ceftazidime. Vancomycin can be substituted for flucloxacillin in cases of penicillin allergy or if methicillin-resistant S. aureus is considered likely. The duration of antibiotic administration is usually 4-6 weeks then 2 to 3 months of oral antibiotic therapy . However this should also be coupled with surgical treatment which consists of decompressive laminectomy and surgical debridement (particularly for those cases related to haematogenous spread).

**Paediatric perspectives -** Spinal epidural abscess is rare in children. Unlike adults, only 35%-38% of children have a predisposing medical condition, most often sickle cell anaemia or immunosuppression from chemotherapy or malignancy. Clinical presentation, bacteriology, diagnostic approach and treatment strategies are otherwise similar to adults. Fortunately, 75%-85% of children with SEA have a favourable outcome.

Three more illustrative cases:

41yo with a Hx of Hep C on methadone presented with chest pain and ? abnormal gait on a background of 8 weeks of thoracic back pain. Paucity of notes but unsteady gait ? drug related ? weakness but no neuro exam documented– walked out of ED. Represented 4 days later with progressive leg weakness – WCC 12.2 – CXR normal – blood cultures –ve – MRI epidural mass c/w abscess – Staph aureus on surgical sample – subsequent partial paraplegia.

34yo presents with 2 weeks of low back pain with L4 radiculopathy on background of "previous" IVDU and type 1 diabetes. O/E temp 37.6C – neuro NAD – Xray NAD – CRP 89 WCC 10.1 – home – recalled after blood cultures +ve for Gm +ve cocci (Staph aureus grew)- MRI showed L4 (L3) osteomyelitis and epidural abscess at L3/4 space. No sequelae

55yo lady presents with lumbar pain – afebrile – normal exam – home with endone and advice to organise MRI. Represents with increasing back pain – noted to have temp 38C – exam unchanged – reassurance – home. Represents with delirium – found to have Gm+ve cocci in blood cultures – MRI showed extensive epidural abscess.

### CLINICAL BOTTOM LINE - Remember the Red Flags

Back pain + IVDU ("ex"-IVDU) +/- any sign of inflammation/ fevers = MRI

Back pain + immunosuppression + any sign of inflammation/ fevers = MRI

Or following from the first case

Back pain + any sign of inflammation/ fevers = consider epidural abscess & MRI

**Refs** - CJEM by Louis, Med 2005;7(5):351-4, Infectious Diseases (Cohen & Powerly) and Textbook of Clinical Neurology (Goetz.), Sendi P et al, Spinal epidural abscess in clinical practice DOI: <u>http://dx.doi.org/10.1093/qimed/hcm100</u>

### NEXT WEEK'S CASE

A 52yo man with a Hx of hypertension presents with vertigo. The RMO reports horizontal nystagmus. What is your approach to this case?

### JOKE / QUOTE OF THE WEEK



Please forward any funny and litigious quotes you may hear on the floor (happy to publish names if you want)

THE WEEK AHEAD

Tuesdays - 12:00 – 13:45 Intern teaching -Thomas & Rachel Moore

Wednesday 0800-0900 Critical Care Journal Club. ICU Conf Room / 12.00-1.15 Resident MO in Thomas & Rachel Moore

Thursday 0730-0800 Trauma Audit. Education Centre / 0800-0830 MET Review Education centre / 1300-1400 Medical Grand Rounds. Auditorium.