The Weekly Probe

1st April 2016 **Volume 13 Issue 8**

Belated Congratulations – Congrats to Chris Y and her partner on the arrival of their baby girl severine River Matchett who was born on 19/3 Saturday weighing 3.35kg. Apparently mum and bubs (and dad) are all doing well

SESIAHS Tox service – A reminder that you can ring the South Eastern Toxicology Service (SEATS) based at POWH for phone advice on tox issues. You can reach them by one mobile phone call on 0423366022. This number is stuck on the glass window facing the paeds area or alternatively go through POWH switch. The toxicology service is staffed by consultants and fellows and will be keen to provide toxicology advice to the local health service.

Cleaning up after yourself- There has been ongoing increasing incidents of blood stained sheets being left on procedure room beds and then being covered up with the paper drawn out over it.
A reminder to all medical & nursing staff that sheets are to be removed post use if there is any fluid spill / soiling to sheets.

THIS WEEK

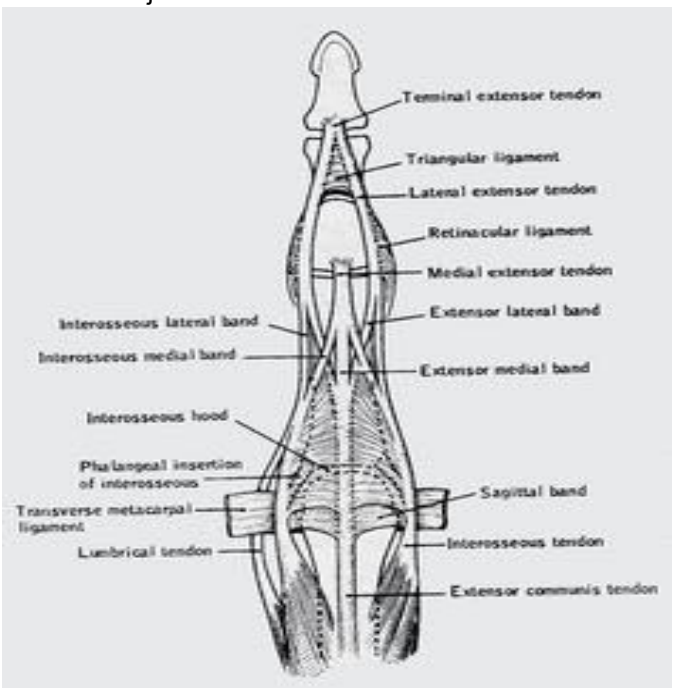
Extensor Tendon Injury (central slip) – Elson's test

A patient presents with a laceration of the dorsal PIP. They can extend the DIP and PIP. Could the tendon be divided ? If so which one and what test do we use to check this?

The finger may be extended by the extensor tendon or the weaker lumbricals (extend at DIP and PIP yet flex at MCP).

As shown on the diagram below, just proximal to the PIP the extensor tendon divides into.

- The central slip (labelled as the medial extensor tendon) which inserts onto the base of the middle phalanx. This allows extension of the PIP joint (without the need to extend the PIP).
- Two lateral slips – these pass down the side of the dorsum of the PIP and insert onto the base of the DIP. They extend the DIP but MOST IMPORTANTLY they can also extend the PIP joint.



The key with assessing any laceration is a thorough wound inspection. This includes a well lit, bloodless , anaesthetised wound with appropriate retraction and assessment in a range of positions (in particular the position the patient was in at the time of the injury).

However to assess for complete disruption of the central slip you can use **Elson's test** or a modified Elson's test
Elson's test – a picture (video) paints a thousand words so if you're reading this on your PC go to this you tube clip. If not then there are a couple of steps:

- Position the patient with the PIP flexed at right angles over the edge of a table
- Ask the patient to extend the PIP against resistance
- This extension will be comparatively weak, and most importantly , the DIP will extend due to reliance on the lateral slips (when the central slip is divided and the lateral slips are working)

Try this on yourself and you'll see that normally the DIP can stay flexed despite significant resistance to PIP extension.

An alternative test is the **modified Elson's test** . This is shown in the picture below (push the fingers together = extend the PIP jt)

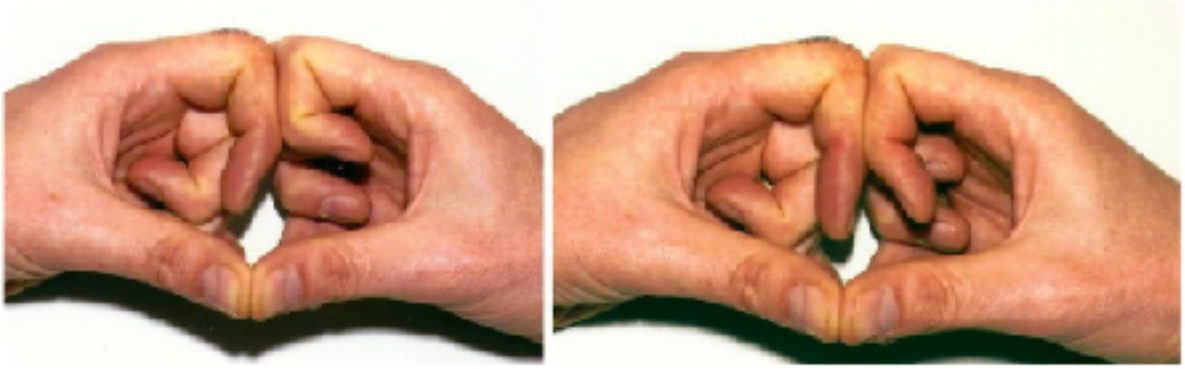


Fig. 2.
The modified Elson's test is performed by asking the patient to place the dorsal aspects of the middle phalanges of the injured finger and the same finger from the opposite side together with the proximal interphalangeal joints flexed to 90°. The patient is asked to push the fingers together whilst trying to extend the distal interphalangeal joint (DIPJ) of both fingers. The finger with the central slip injury will extend more at the DIPJ, the left index in this case.

Refs: Venus MR , The modified Elson's test in open central slip injury *Injury Extra* 41 (11) November 2010, 128–129 http://www.sciencedirect.com/science/article/pii/S1572346110003466

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)

Couple of weeks ago we discussed CADASIL syndrome. Another unusual neuro case from a couple of weeks ago.

62yo lady with a Hx of HT'n, DM and IHD presents with sudden onset of bilateral visual loss and headaches. BP 190/110 – obs otherwise normal Unable to see light / dark - Pupil reactive to light- Fundi normal. What is going on?

Progress and diagnosis:
CT head and venogram normal
On transfer she had 3 seizures described as "twitching of her eyes/ deviated to the left " lasting couple of minutes
MRI showed "Multiple foci of abnormal signal intensity in the cerebellum as well as within the parietal and occipital lobes bilaterally. The features are consistent with the diagnosis of PRES (Posterior reversible encephalopathy syndrome)"
She was admitted to ICU with BP control- improvement in vision 6/9 + 6/24- Discharged home 5 days later. What is PRES?

Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiologic neurotoxic entity characterized by headaches, altered mental status, seizures, and visual loss and is associated with white matter vasogenic oedema predominantly affecting the posterior occipital and parietal lobes of the brain.

Pathophysiology –Uncertain yet thought to be related to disordered cerebral autoregulation with two possible aetiologies. Endothelial dysfunction is a central component (chicken or the egg?) in either :

1)Cerebral vasospasm causing cytotoxic oedema
2) vasodilatation which results in vasogenic oedema.

The syndrome can be precipitated by various clinical settings including :

- severe hypertension- including eclampsia / preeclampsia – Note that Hypertension is not present or does not reach the upper limits to self-regulation (150-160 mmHg) in 25% of patients.
- haemolytic uraemic syndrome (HUS) / thrombocytopaenic thrombotic purpura (TTP)
- SLE
- drug toxicity- esp chemo agents / immune suppression such as cisplatin, interferon, EPO, tacrolimus, cyclosporin, azathioprine
- bone marrow or stem cell transplantation
- sepsis

- hyperammonaemia

The term PRES however can be a misnomer as the syndrome can:

- involve other regions / extend beyond the posterior cerebrum.
- Some patients may have residual neurological defects (yet most cases resolve with treatment of the precipitating cause)

However ES is a pretty crappy acronym so let's leave it as it is.

Location of Pathology- As the name suggests, typically the pathology involves regions supplied by the posterior cerebral artery with symptoms relating to changes in the occipital and parietal regions (~95% of cases). However other watershed areas may be involved including the frontal, inferior temporal, cerebellar and brainstem regions. Most commonly there is a bilateral, symmetrical distribution.

Clinical presentation

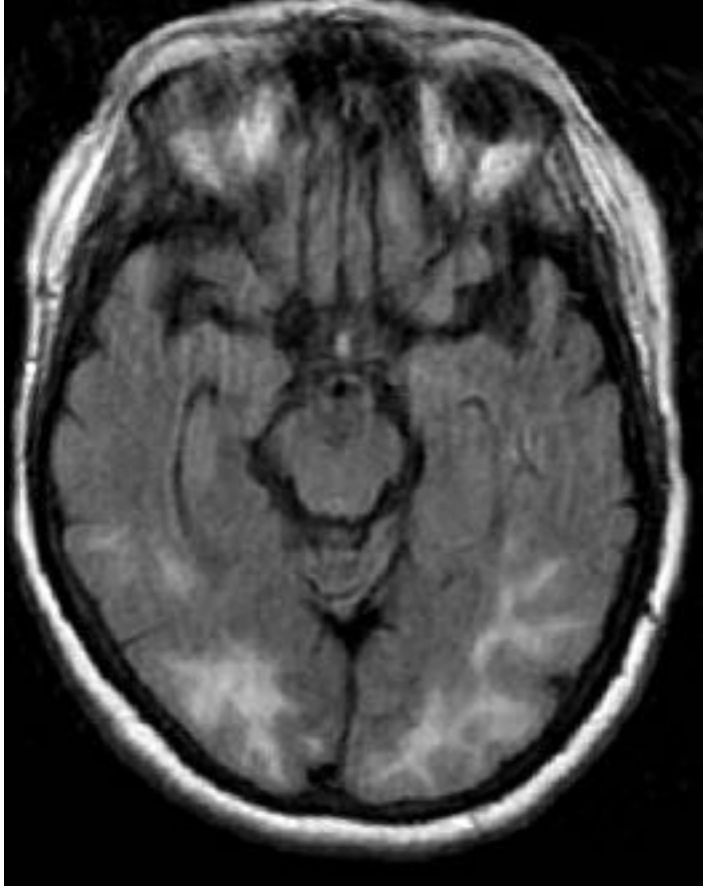
- Headaches- typically constant, persistent
- Altered consciousness- variable
- Visual disturbances- can range from hemianopia, visual neglect, auras, visual hallucinations, and cortical blindness (apparently this may be accompanied by denial of blindness). Fundi – may have chronic or acute hypertensive changes. There may be other focal neuro signs attributable to the region involved including upper cervical cord.
- Seizures- often the presenting problem- focal or generalised- may have preceding visual loss or visual hallucinations

Radiographic features

Most commonly there is vasogenic oedema within the occipital and parietal regions (~95% of cases), which are supplied by the posterior cerebral artery supply with the caveat as described above. Both cortical and subcortical locations are affected.

CT- affected region- if large, may see low attenuation regions c/w oedema / infarction

MRI – key Ix esp if CT normal or nonspecific. Demonstrates oedema involving the white matter of the posterior portions of both cerebral hemispheres, in a relatively symmetric pattern. Below are FLAIR MRI images demonstrating bilateral occipital lobe oedema. However as noted earlier the pathology can involve the brain stem, cerebellum, and frontal and temporal lobes. ~ 15% have (micro) haemorrhages.



DDs- Symptoms and signs may mimic other focal and generalised neurologic conditions, such as stroke, venous thrombosis, toxic or metabolic encephalopathy, demyelinating disorders, vasculitis, or encephalitis.

Treatment

- **Hypertension** – aggressive BP control aiming for DBP ~ 100-105 – use IV not oral – avoid excessive reductions particularly if long standing Ht'n as may drop below range of autoregulation - theoretical concern re vasodilalation yet most agents have this as mode of action
- **Seizure control-** benzos , phenytoin
- **Supportive Mn-**fluid, electrolytes, airway Mn **– ABCDEFGHILM**
- **Specific Tx**
    - Eclampsia – Mg – BP control- deliver baby
    - Cytotoxic – involvement of treating team – reduction or ceasing meds
    - TTP – plasmphoresis / Immunoglobulin

Refs –Up-to-date / Radiopaedia (http://radiopaedia.org/articles/posterior-reversible-encephalopathy-syndrome-1)

NEXT WEEK'S CASES

A 3 yo child presents with 30 minutes of ongoing generalised TC afebrile sezures despite 0.1mg/kg IVI midazolam. What is your next option?

Editor: Peter Wyllie

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.

Editor: Peter Wyllie