South Warwickshire NHS Foundation Trust

Inside this issue...

Acute Medicine Educational Newsletter

October 2023 | Edition 6

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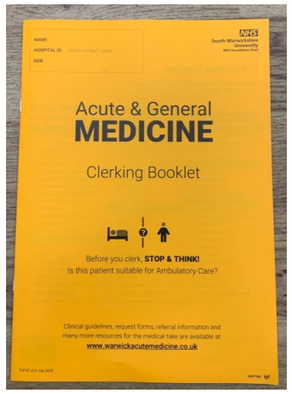
Editor
Angela Perrett | Acute Medicine ACP

Editors Update

Angela Perrett | ACP Acute Medicine

It has been a few months since the last edition of the newsletter and there have been lots of changes along the way. ADU has been renamed MSSU, EPMA has been introduced into Acute Medicine, there is a new clerking booklet, there are some new guidelines, doctors have been striking, members of staff have got married, some staff have gone off to have children, and some have moved onto pastures new.



This highlights how Acute Medicine is forever transforming and how important it is to work together, to look after each others wellbeing, and to teach each other along the way. I hope that you find the Acute Medicine Educational Newsletter can support you all in some way.

Summer is coming to an end, and even as I write this, we can see the Winter Pressures setting in. The ACPs have started a morning and post-work huddle, to discuss how we are feeling, and what has been good or bad about the day. This helps to relieve stresses from the work place. This might be something you would like to try with a colleague or two! Just remember, always be kind to everyone, you never know what kind of day they are having!

I am very grateful to the staff that have sent me articles for this edition, I have really enjoyed reading them all, and hope everyone can gain something. If you would be interested in writing for the next edition, please do get in touch.

01 Meet the Team: New roles

Lois Houlders | Advanced Clinical Practitioner

Having been supported by the Trust to complete a post graduate certificate in education last year I was successful in obtaining a secondment to lecture on the Advanced Practice ACP Master's programme at the University of Birmingham as an Associate Clinical Professor, for the school of Nursing, Midwifery and Institute of Clinical Sciences.

UNIVERSITY OF BIRMINGHAM

This new role allows me to teach trainee ACP's of the future and develop my own teaching, whilst still maintaining my clinical practice within acute medicine. In addition, I have been able to develop skills as an examiner through OSCE (objective structured clinical examination) examinations and marking written work at Master level. I currently support a small group of students as an academic personal tutor, meeting regularly with my students and their

clinical supervisors to support ongoing development. I have had the opportunity to become module lead for the Advanced Clinical Decision-Making module, developing the programme as part of my new leadership role. Keeping up to date with the changes in ACP education at a national level will benefit ACPs within our department and trust, as well as my experience of supporting ACP clinical supervision and providing opportunities for ACPS who would like to observe or participate in higher education teaching as part of their development.

02 Presenting complaint of back pain

Dr Vanisha Katamaneni | FY1

Your next patient to clerk...

72y/o male presented to ED with progressively worsening back pain for the last 7-14 days. Unable to get comfortable, pain worse on lying down. Not relieved by simple analgesia.

Differential diagnoses?

INTRINSIC	SYSTEMIC	REFERRED PAIN
Muscular lumbar strain/sprain	Malignancy	AAA
Disc disease- herniation/degeneration	Inflammatory spondyloarthropathy	GI: Pancreatitis, peptic ulcer disease
Cauda equina syndrome Spinal cord compression	Vertebral discitis, spinal abscess	Pyelonephritis, renal colic
Compression fracture		Pelvic: PID, endometriosis, prostatitis
Spinal stenosis		Herpes zoster

Red flags in back pain

- Saddle anaesthesia
- Bladder or bowel new onset incontinence, loss of tone - PR!!
- New onset profound / progressive neurological deficit
- Hx of malignancy
- Immunosuppression- prolonged corticosteroid use
- IV drug use
- Pain worse at night
- Thoracic back pain
- History of osteoporosis and fragility fractures
- Systemic sx- fever, fatigue, night sweats, weight loss

Taking a history

Things to explore in a history:

- SQUITARS/SOCRATES- duration, location, intensity, progression. Triggers? Does anything help the pain?
- Radiculopathic symptoms- tingling, burning, shooting
- Loss of sphincter symptoms - incontinence/constipation
- Hx of trauma
- Systemic symptoms?
- PMH: Previous/current malignancy, previous fragility fractures, T2DM, previous spinal injuries/issues
- Drug Hx: steroid use? Analgesia use? Allergies
- FHx: inflammatory spondyloarthropathies, cancer
- SHx: IVDU?

Examination

Monitor observations: temperature, haemodynamic stability. Full systems examination, including:

- Inspect the area of pain; any erythema, bruising, swelling? Palpate
- Full neurological examination to look for ?hyper-reflexia, loss of sensation, weakness, change in temperature/pressure sensation
- abdominal exam
- Renal angle tenderness
- PR! - examine anal tone, sensation, prostate
- IVDU- inspect groins, injection sites

Case 1

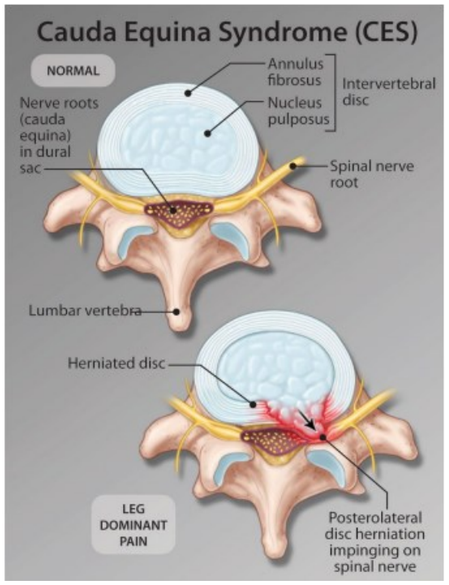
50y/o female. Lower back pain, numbness in medial thighs, bladder incontinence, strange sensation in anal area, poor anal tone on PR.

Cauda equina syndrome

Compression of lumbosacral nerve roots of the cauda equina L2-S5 Commonly due to lumbar disc herniation, trauma, spinal surgery, abscess

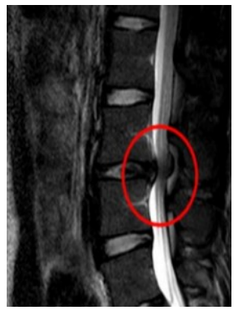
Features:

- Urinary incontinence- (can be urgency, poor stream, reduced awareness of urinating in earlier stages)
- Faecal incontinence, anal sphincter laxity
- Lower limb weakness
- Saddle anaesthesia “numbness” “pins and needles” (PR!)
- Bilateral sciatica in 50%



Investigation:

- Urgent MRI lumbar spine
- CT Lumbar spine if MRI unavailable/ contraindicated but less precise



Management:

Urgent decompression surgery within 48 hours of symptom onset- urgent discussion with neurosurgeons

Case 2

80y/o male. History of recently diagnosed metastatic prostate cancer. Reports altered sensation below a marked level, exam finds hyperreflexia of lower limbs.

Spinal cord compression

Compression of the spinal cord at any level, commonly due to tumours- primary/metastatic, infection, centrally prolapsed disc, haemorrhage, skeletal deformity.

Features:

- Unremitting back pain, keep awake at night
- Sensory loss under a certain level - test the ascending tracts
- Weakness/total loss of power
- Altered reflexes

Management:

- Urgent MRI imaging/CT
- High dose steroids with PPI cover
- Urgent radiotherapy if malignancy/ spinal cord decompression surgery

Case 3

65y/o male. Sudden onset back pain and abdominal pain, BP 70/50, HR 134. Pulsatile abdominal mass on exam.

Ruptured Abdominal Aortic Aneurysm

AAA definition: Diameter of 3cm or more/>1.5x expected AP diameter.

Triad of: 1) abdominal +/or back pain, 2) pulsatile abdominal mass, 3) hypotension = ruptured AAA

Immediate resuscitation:

- A-E assessment
- Hypotensive resuscitation (aggressive fluid replacement can exacerbate bleeding). Target BP 90-120 systolic and withhold fluids pre-op.

Risk factors:

Old age; CVD; Family History; Smoker; males.

Investigation:

Aortic ultrasound

Management for ruptured AAA:

Urgent surgical repair, (vascular surgeons), peri-op Abx, analgesia

Case 4

30y/o male presents with fevers, night sweats, localised back pain worse when lying down and weight- bearing. He tells you that he is a current IVDU user.

Infective

1. Epidural abscess

Spinal epidural abscess is inflammation with pus in the epidural space.

Features:

- Localised pain, not relieved by rest
- Fever
- Rigors.
- If neurology is present- not a good sign.

Risk factors:

- Intravenous Drug User
- Immunosuppressed
- Diabetes
- Endocarditis/bacteraemia,
- Recent spinal surgery/trauma,
- Contiguous local infection

Investigation:

- Bloods
- Blood cultures
- Direct aspiration for culture
- MRI whole spine with and without contrast.

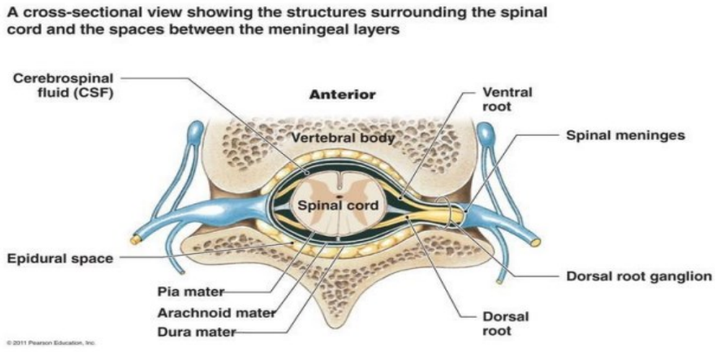


Image: Copyright Pearson Education 2006

Management:

- Intravenous Antibiotics
- if neurological deficit / not responding to Abx- surgery.

2. Discitis

Discitis is an infection of intervertebral discs

Features:

- Fever
- Weight loss
- Non-mechanical back pain,
- Localised tenderness on palpation, warmth, erythema.
- History of IVDU, immunosuppression, diabetes.
- Can get osteomyelitis associated as well.

Investigation:

- X-ray may show vertebral body destruction.
- MRI to further visualise area of infection.
- Raised inflammatory markers on bloods.

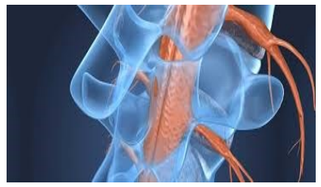
Management:

- Antibiotics
- Analgesia
- Assess neurology daily
- T&O involvement

Case 5

72y/o female. Previous breast cancer resected. Presents with "sciatica" progressively worsening for the last 1 month, no respite. Says she has noticed weight loss, and feels increasingly tired.

Malignancy



Features:

- Age >50
- Back pain at night and at rest,
- Systemic symptoms of fevers, weight loss, malaise.
- May have a history of malignancy.

Cancers that commonly metastasise to bone: think organs that are in "pairs" : Thyroid; Lung; Breast; Renal; and prostate

Investigations:

- Bloods for tumour markers.
- Imaging: X-ray, CT/MRI.

Management:

- Get relevant specialties involved for MDT discussion.
- Analgesia.
- If tumour destruction is extensive causing neurological compression then may need surgical input.

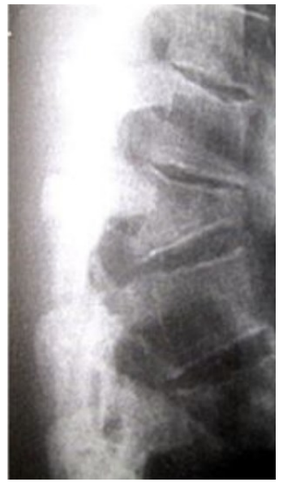
Case 6

32y/o male. Presents with acute severe back pain in the last 4 hours, came on when carrying heavy furniture. No neurology but is struggling to walk due to the pain. No PMH.

Musculoskeletal

1. Compression fractures

Features:



- Pain at rest and at night
- Previous history of fragility fractures,
- History of osteoporosis/ risk factors for osteoporosis

Management:

- Analgesia
- Early mobilisation +/- orthosis
- Osteoporosis prophylaxis
- If multiple columns involved: T&O involved, surgery, strict bed rest

2. Muscular strain/sprain:

Features:

- Sharp, spasm-like pain, may be an acute onset
- Usually no red flag signs on history

On exam:

- Restricted range of motion
- muscle tenderness
- trigger points
- Clinical diagnosis, after excluding other causes

Management:

- Consists of reassurance, encourage mobilising
- Analgesia: use pain ladder. NSAIDs, codeine, muscle relaxants e.g. cyclobenzaprine, diazepam

Back to our patient...

History:

No red flags for CES, but pain was unremitting and continued at night. No Hx of malignancy, not immunosuppressed. However, patient did have systemic symptoms of weight loss, fatigue on further questioning.

Past Medical History:

- Hypertension
- T2DM

Examination:

Patient systemically well. No neurological deficit, no CES signs. PR- anal tone and sensation in tact, prostate enlarged. No warmth/localised tenderness of spine.

Investigations:

- PSA raised at 22.
- CT done to r/o AAA- unfortunately found metastatic deposits at multiple vertebral levels.

Management:

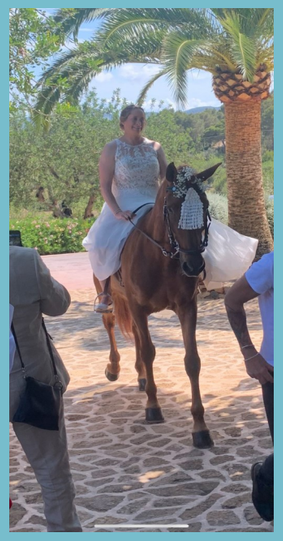
- Referred to Urology MDT
- Outpatient TURP and f/u with urology.
- Analgesia,
- Safety netting.

Key points

- Lots of differentials
- A good history and examination will help
- Always ask red flag signs and keep most worrying diagnoses In back of mind
- PR exam important
- If fevers, rigors, back pain - think abscess/discitis
- Always ask systemic symptoms
- In males with CVD Hx- think AAA

Resources used:

- BMJ Best practice
- Oxford Handbook of Acute Medicine 4th edition



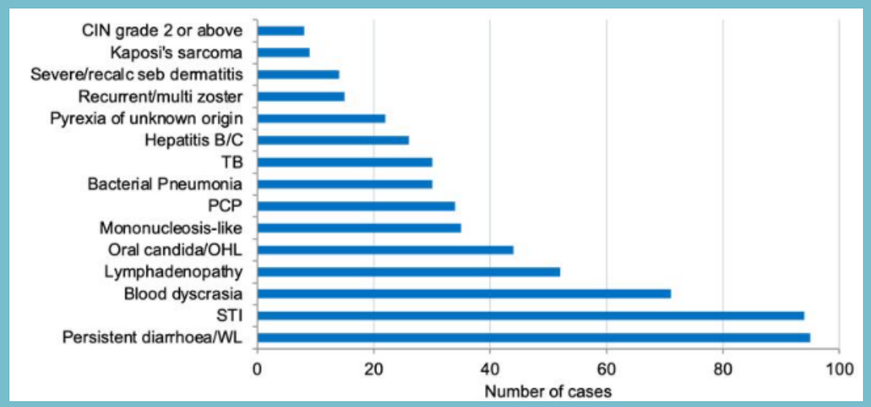
Congratulations to Dr Pollard and her husband Mr Barber on their marriage

#ibiza23

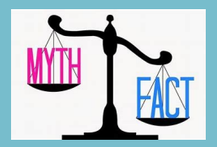
Think....does this patient need a HIV blood test?

Only 20% of patient's at SWFT who having conditions that indicate the need for HIV testing get tested ..... Can we make it 100%?

Most common conditions associated with HIV



- **Non-Hodgkin lymphoma**,
- Aseptic **meningitis**,
- Cryptococcal meningitis,
- Dementia ,
- Peripheral neuropathy,
- Severe/rectal psoriasis,
- Cryptosporidiosis,
- Parotid cysts,
- Toxoplasma,
- Unexplained retinopathy,
- CMV,
- Head/neck Ca ,
- **Hodgkin's lymphoma**,
- Multicentric Castleman's ,
- Shigella
- Progressive multifocal leukoencephalopathy,



Common Myths:

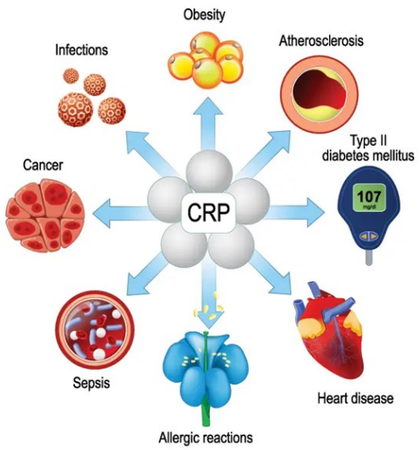
- **HIV is no longer a death sentence...early diagnosis and effective treatment means that people with HIV are able to live a normal life well into old age.**
- **Consent to perform a HIV test is no longer required ... we should however inform patients that a HIV test will be performed alongside other blood testing.**

03 Not another blood test: C-Reactive Protein (CRP)

Sadie Davis | ACP Acute Medicine

CRP is a protein which is produced by the liver and elevated levels can indicate the presence of inflammation, infection or tissue damage in the body. Other causes of a raised CRP are pictured below.....

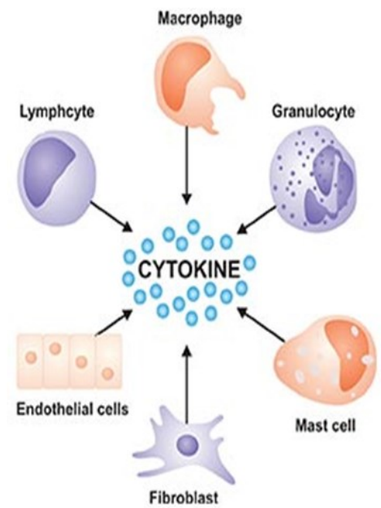
Increased levels of C-reactive protein



CRP is an acute-phase reactant protein that is primarily synthesised in the liver in response to cytokine signals, such as interleukin-6, which are released by macrophages and other immune cells during the early stages of inflammation.

CRP can also be produced by adipose tissue and other peripheral tissues, and its production is regulated by a complex network of transcription factors that respond to various stimuli, such as lipopolysaccharides and oxidative stress.

The production of CRP is a critical component of the innate immune response, as it plays a key role in the recognition and clearance of pathogens and damaged cells. In addition, CRP has been implicated in numerous other biological processes, including complement activation and regulation, cell adhesion and migration, and tissue repair.



The CRP blood test is a highly sensitive test that can detect even small changes in CRP levels in the blood. This makes it a valuable tool for monitoring the progress of an inflammatory disease or infection over time. The test is also useful for identifying patients who are at high risk for developing cardiovascular disease, as elevated levels of CRP have been linked to an increased risk of myocardial infarction and stroke.

Less than 4 mg/dL is a negative result. Very high levels of CRP, greater than 50 mg/dL, are associated with bacterial infections about 90% of the time. In multiple studies, CRP has been used as a prognostic factor in acute and chronic infections, including hepatitis C, dengue and malaria. Clinical correlation is required when interpreting the results of CRP.

Certain medications, such as non-steroidal anti-inflammatory drugs (NSAIDs), will falsely decrease CRP levels. Statins, as well, have been known to reduce CRP levels. Magnesium supplementation also can decrease CRP levels.

The frequency with which the CRP test is recommended depends upon the reason for testing and the underlying medical condition being monitored. For example, in individuals with an autoimmune disease, such as rheumatoid arthritis, monitoring of CRP levels may be done every few months to assess disease activity and response to treatment.

Costing is approximately £2.00 for the NHS, noted privately online companies offering testing for up to £154.00.

References

Blann, A. (2013) *Routine Blood Results Explained.* 3rd edn. London: M&K Publishing

Kumar, P. and Clark, M. (eds.) (2009) *Clinical Medicine.* 7th edn. London: WB Saunders

National Institute for Health and Care Excellence (2017) *Sepsis: recognition, diagnosis and early management* [online] available from <https://www.nice.org.uk/guidance/NG51>

Sara M. Nehring; Amandeep Goyal; Bhupendra C. Patel (2022) CRP Testing (online)

Thank You AMU

Dear Colleagues,

I received an email from a member of staff whose father and husband each attended ED and then her father transferred to Oken and Malins Ward and her husband to Guy Ward with treatment from the OPAT team.

I would like to thank you very much for the care that you and your staff provided which was noted by the member of staff as caring, brilliant, compassionate and amazing.

We very much value the appreciation of patients and it is tremendous to hear about your exceptional work.

Yours sincerely,

Adam Carson
Managing Director

Thanks to AMU for welcoming non-executive member, Sue, for being an amazing team and for supporting updated admission assessments.

Falls Prevention Month

04 Cellulitis Part 1

Julie Statham | OPAT ACP

Cellulitis is a spreading bacterial infection of the skin and the soft tissues immediately beneath the skin. It is characterised by redness, heat, swelling and pain which often spreads rapidly. Often there is associated oedema of the site. **Limb cellulitis is usually unilateral.**

Cellulitis may be also accompanied with systemic symptoms of infection including fever, rigors, nausea and tachycardia.

Cellulitis is typically caused by a break in the skin which is one of the body's key defence mechanisms against infection. The break in the skin may not always be at the site of cellulitis.

Risk factors

Whilst cellulitis can affect anyone of any age there are some factors that increase the risk:

Risk factors for cellulitis	
Previous cellulitis	Athlete's foot
Venous insufficiency	Inflammatory skin conditions
Elderly patients	Insect Bites
Alcohol dependency/ IV drug use	Diabetes
Lymphoedema	Obesity
Immunosuppression	Pregnancy

Main sites and bacteria associated

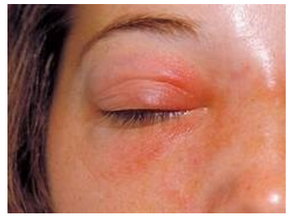
Cellulitis can affect any body site, however it is most common in the lower leg. Other typically affected sites include hand /arm and face and antibiotic choice will differ depending on the affected site and the likely causative bacteria.

Leg cellulitis: in the absence of ulcers/significant wounds most lower leg cellulitis is caused by bacteria that

make up normal skin flora including Staphylococci and Streptococci.

Hand/arm cellulitis: Staphylococci and Streptococci are still the most likely pathogens but cellulitis following hand injuries e.g. whilst gardening may require anaerobic cover in addition. Also cellulitis following an animal bite will require broader antibiotic cover so that animal oral bacteria are also covered (please see animal bite guidelines).

Facial cellulitis: Facial, peri-orbital and orbital cellulitis can be caused by Staphylococci and Streptococci but also by anaerobic bacteria therefore the addition of anaerobic cover is necessary.



Patients who are immunocompromised are more likely to have cellulitis caused by a less typical organism and therefore often require a broader spectrum of antibiotic cover.

Patients who have previously been colonised with MRSA may need alternative antibiotic treatment e.g. with vancomycin or teicoplanin rather than a beta-lactam antibiotic.

Investigations

All patients with suspected cellulitis should have an FBC, CRP, U&Es and LFTs taken.

A set of observations should be taken to determine if any signs of systemic illness.

Blood cultures should be taken if a patient is pyrexial, ideally prior to any antibiotic therapy or change in antibiotic therapy if already on antibiotics.

History

A comprehensive history including underlying medical conditions, any risk factors and potential exposure to more specific pathogens should be taken.

Recent foreign travel, occupation (farmers, butchers, those working with aquatic life) and patients with underlying immunosuppression may change the antibiotic choice.

Patients with underlying lymphoedema are more likely to need extended courses of antibiotics and more likely to need intravenous antibiotics to ensure good penetration to the site of infection

Examination

A physical examination of the area, including marking of the borders of the cellulitis and to rule out other differential diagnoses (e.g. DVT) should be performed.

- For limb cellulitis check for any tracking of the cellulitis (this may not be continuous from the site of cellulitis so check further up the leg or arm as well).

- Check under any bandages for signs of wounds or ulcers which can be swabbed and may need addressing alongside the cellulitis treatment. If an ulcer is present check for previous swabs of the ulcer to see if any significant bacteria has previously grown.

- Check for signs of athletes' foot/ fungal nail infection. Send clippings for MC&S in potential fungal nail infection. Treat any underlying fungal infection.

- Check to see if any underlying metalwork over site of cellulitis (prosthetic joint, metal plate or screw) as may indicate a deeper infection or require a longer course of antibiotics. Low threshold for T&O review.

- If history of animal (i.e. cat/dog) or human bites will need different antibiotics.

In cases of severe cellulitis tests for HIV, hepatitis B and C and diabetes should be sent to rule out any undiagnosed underlying disease.

Patients who present with suspected orbital cellulitis should have an ophthalmology examination and a CT sinus/orbits to rule out eye involvement and abscess formation. These patients require admission due to risk of complications. Pain on eye movement and or loss of colour vision are signs of orbital rather than periorbital disease. All cases of orbital or peri-orbital cellulitis should be referred to OPAT at presentation as they often need alternative antibiotics to the standard cellulitis guideline and are likely to be discharged under OPAT.

To be continued ....

05 Drug of the Issue: Migraine Treatment

Harriet Meakin | Acute Medicine ACP

Sumatriptan

Acute treatment of migraine

Indications:

Treatment of acute migraine and cluster headaches. Can be given orally, subcutaneously and intranasally.

Oral Dose:

50-100mg when headache occurs, can be repeated after two hours if necessary max 300mg 24 hours

Cautions:

With the elderly, history of seizures, mild controlled hypertension, conditions which may increase risk of coronary artery disease, risk of seizures .

Side effects:

Dizziness; drowsiness; feeling abnormal; flushing; nausea; myalgia; pain; skin reactions; temperature changes; hypersensitivity reactions; vomiting; diarrhoea; seizures; hypotension; MI.

Very common with intranasal- epistaxis and altered taste sensation

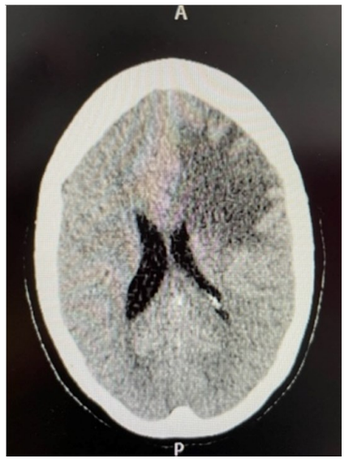
Case Study

56 year old female presents to ED with 2-week history of frontal headache with some mild photophobia and visual disturbances

PMH migraines related to menstruation stopped 30 years ago now post-menopausal.

Decision to trial sumatriptan as previously responded to triptans as paracetamol not helping

CT head was done later that day which shows multiple metastatic deposits in the frontal lobe with midline shift, significant oedema and uncal herniation. MRI confirms high grade glioma and she was accepted under the care of neurosurgeons.



Keppra was started as per neurosurgeons as no harm to the patient- but it is important to highlight caution with triptans as they should be avoided in patients who have increased risk of seizures

Amitriptyline

Long term treatment of migraine (prophylaxis)

Indications:

migraine prophylaxis and chronic tension-type headache prophylaxis- prevention of acute migraine attacks.

Oral Dose:

10-25mg daily initially if tolerated can be increased in increments of 10-25mg every 3-7 days max dose 75mg

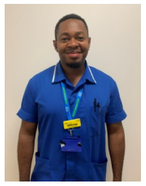
Cautions:

Cardiovascular disease; chronic constipation; diabetes; epilepsy; history of bipolar disorder; history of psychosis; hyperthyroidism may increased risk of arrythmias; caution in the elderly; increased suicide risk.

Side effects:

Drowsiness; prolonged QT interval; anxiety; breast enlargement; arrythmias and cardiac disorders; hypertension; hypernatremia; weight changes; withdrawal symptoms; suicidal behaviours; concentration impairment; peripheral neuropathy.

06 New Starters in Acute Medicine



Offiong Ekpe

Band 5

Jenny Lowe

Band 5

Jessica Kelly

Band 5

Wadzanai Wenjere

Band 5

Kiera Locke

Band 5

Mency Sampath-kumar

Band 5

Helen Miller

Band 5

Chantelle Fox

Band 4 (NAR)

Lydia Fairley

Band 4

Gwenola Dugue

Band 5

Akinsoji Akintola

CSW

Alexandra Pearson

CSW

Daniel Dalling

CSW

Caroline Wells

CSW

Welcome to the Acute Medicine team

07 AKI and Fluid Balance: The basics

Anna Tillotson | Acute Medicine ACP

So What?

Around 15% of patients admitted to hospital with an acute illness will have / develop a reduction in kidney function referred to as an AKI (acute kidney injury) there is an overall 23.8% mortality rate for those who develop an AKI whilst in hospital.

The kidneys are two organs in the body that lie either side of the backbone, they receive blood through the renal artery. The main functions of the kidneys are to:

- Maintain salt and water balance
- Filter/remove waste products from the blood via urine
- Control blood pressure by releasing hormones (renin-angiotensin-aldosterone hormonal system) if the blood pressure falls a healthy kidney can increase renin production to improve blood pressure, an unhealthy kidney can have an overproduction of renin which can cause hypertension.
- Helps to produce new red blood cells as well as maintaining healthy bones

What is AKI (Acute Kidney Injury)?

A rapid (days to weeks) decline in the kidneys' ability to filter wastes from the body and excess water. This causes a build-up of waste products in the blood which makes it hard for the kidneys to keep the right balance of fluid in the body. These waste products in the blood are called creatinine and urea and they will be raised when the kidneys are not functioning correctly. AKI's can also effect other organs in the body such as the heart, brain and lungs. More common in older adults.

AKI can be categorised in severity of AKI stage 1, AKI stage 2, AKI stage 3, this is based upon the individual's baseline kidney function.

	CREATININE AND/OR	URINE OUTPUT
STAGE 1	Rise 1.5-2 fold from baseline	≤0.5 ml/kg/hr more than 6 hours
STAGE 2	Rise of 2-3 fold	≤0.5 ml/kg/hr more than 12 hours
STAGE 3	Rise >3 fold	≤0.3ml/kg/hr more than 24 hours

Symptoms

Signs and symptoms of acute kidney injury differ depending on the cause and may include:

- Reduced urine output
- Swelling in legs, ankles, and around the eyes (oedema)
- Fatigue or tiredness
- Shortness of breath
- Confusion
- Nausea
- Seizures or coma / reduced GCS in severe cases
- Chest pain or pressure

In some cases, AKI causes no symptoms to the patient and is only found through blood tests.

Management

Nursing staff administering medications —if you know your patient has AKI, consider the nephrotoxic drugs below that should be withheld whilst that patient has an AKI. Examples of some commonly prescribed drugs which are considered nephrotoxic are: **ACE inhibitors** e.g. lisinopril; **ARB** e.g. losartan; **NSAIDs** e.g. naproxen; **diuretics** e.g. furosemide, spironolactone. Nursing staff should check with the medical team prior to administration if these drugs are still prescribed for a patient with an AKI.

REMEMBER OUR AKI BUNDLE***

CAUSES: THINK STOP

S epsis
T oxicity
O bstruction
P arenchymal disease

ASSESS:

– Fluid balance
  BP
  Urine output
– Examine chest and bladder

INVESTIGATIONS

- Routine blood test, ABG/VBG
- Urine dip
- Arrange US if suspected obstruction or not responding to fluids

MANAGEMENT

- IV Fluids: Aim BP>100 systolic and urine output>0.5ml/kg/min
- Consider sepsis and treat accordingly
- Stop antihypertensives if hypotensive
- Stop nephrotoxins (e.g. ACEIs, ARBs, NSAIDs), diuretics and metformin
- Avoid contrast if possible
- Discuss with Renal Team if stage 3 (any stage if urine dip +ve)

IV fluid treatment and fluid balance are paramount in treating AKI.

Maintaining accurate fluid balance charts is vital to ensure that the medical team can accurately assess and treat the patient correctly. Balancing input/output and assessing how much urine the kidney is making is essential in treatment. The kidneys job is the production of urine and thus a key indicator of improvement/ deterioration.

In general improved fluid balance monitoring can lead to reduction in the prevalence of AKI's .

CAUSES OF ACUTE KIDNEY INJURY

Prerenal

Sudden and severe reduction in blood pressure (shock) of interruption of blood flow to the kindeys from severe injury or illness

- Blood loss
- Dehydration
- Heart failure
- Sepsis
- Vascular occlusion

Intrinsic Renal

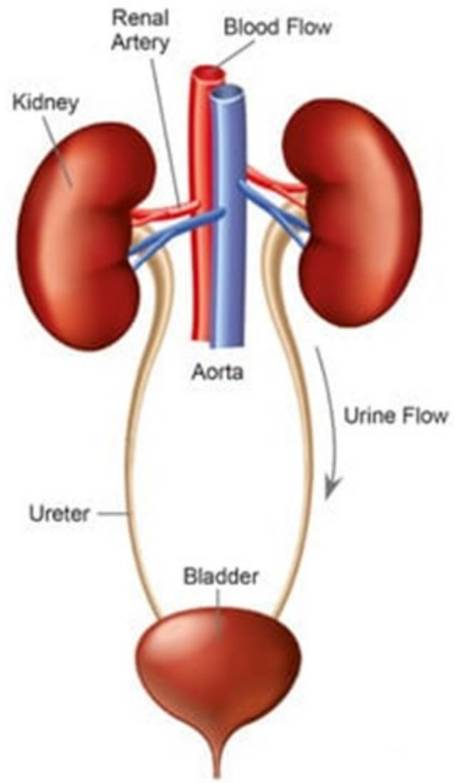
Direct injury to the kidneys by inflammation, drugs, toxins, infection, or reduced blood supply

- Acute tubular necrosis
  - Drugs
  - Toxins
  - Prolonged hypotension
- Glomerulonephritis
- Acute tubular necrosis
  - Drugs
  - Toxins
  - Autoimmune disease
  - Infection
- Small-vessel vasculitis

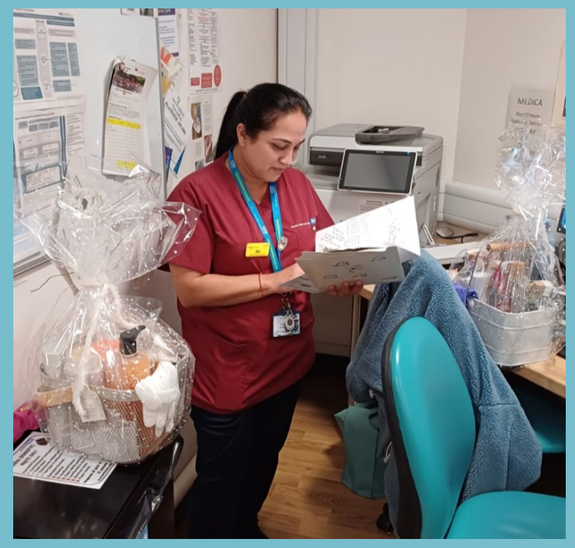
Postrenal

Sudden obstruction of urine flow due to enlarged prostate, kidney stones, bladder injury or tumor

- Benign prostatic hyperplasia
- Cervical cancer
- Meatal stenosis/phimosis
- Retroperitoneal fibrosis
- Prostate cancer
- Urinary calculi



Goodbye Iki ....
You will be missed in Acute Medicine



Good Luck from us all xx

08 Headache Standard Operating Procedure Audit

Charlotte Potter | Acute Medicine ACP

The Headache SOP

The Headache Standard Operating Procedure (SOP) was introduced into practice in November 2020 following a clinical incident. The purpose of this SOP is to provide guidance to clinicians regarding the assessment and subsequent management of adult patients (age >17 years) presenting to hospital with an acute severe headache.

The SOP features specific guidance on the diagnostic work up for Thunderclap headache/ suspected Subarachnoid Haemorrhage, and other red flags present in the history / examination that would mandate further investigation.

Where can it be found?

The Headache SOP is located on the Trusts Acute Medicine intranet site.

The Aim of the Audit

To evaluate the impact and efficacy of a clinical headache SOP in the management of patients presenting with an acute atraumatic headache.

Specifically examining the pathways baseline performance with respect to selecting the appropriate neuroimaging for this patient population.

Background

- Headaches can be defined as a diffuse pain that is not confined to the area of nerve distribution.

Typically classified into two main types **Primary** and **Secondary.**

- **Primary headaches** are those which have no underlying organic disease as their cause.
- **Secondary headaches** include any head pain of infectious, neoplastic, vascular or drug induced origin.
- Headaches are one of the most prevalent neurological conditions and is estimated to be the third highest cause of disability worldwide.
- Headaches are one of the most common reasons for emergency department attendances and admissions.
- The direct cost is estimated to be around **one Billion pounds per year within the NHS.**
- Headaches are complex and difficult to manage.
- A key challenge is how to differentiate which headache patients require further investigation.
- Despite improving guidelines, neuroimaging of this population of patients continues to increase.
- Approximately 90% of headaches seen in practice are of the primary variety, meaning most headache patients could be managed in primary care.
- General Practitioners are often uncertain about when to refer patients to secondary care for diagnostic tests
- In the United Kingdom (UK) the annual primary care consultation rate for headache is 4.4% per 100 4 % of these are referred to secondary care

The Audit

Secondary data held by the trust was collected in the form of small snapshot audit.

Inclusion criteria was all adult patients discharged from the AEC unit with the diagnosis of headache.

Data was collected 6 months prior to the SOP being introduced into practice and 6 months post SOP introduction.

Sample size was 20 patients for each group.

Notes were retrospectively reviewed focussing on:

- Patient's age
- Ottawa score (Calculated retrospectively)
- CT scan performed (YES/NO)
- Lumbar Puncture performed (YES/NO)
- MRI performed (YES/NO)
- Maximal intensity of headache documented (YES/NO)
- Red flags present (YES/NO)
- SAH as suspected diagnosis (YES/NO)
- Working Diagnosis

The Results

Audit found that post SOP introduction we saw an improvement in documentation regarding the maximal intensity, the presence of red flags and OTTAWA scores (see figure 1 on the following page).

There was an increase in all means of investigation post sop, however this was also reflective of the clinical presentations – so results on this are difficult to interpret.

For both groups the working diagnosis for the majority of patients was that of primary headache (see charts in figure 2 and 3).

Figure 1 – Pre and post sop documentation

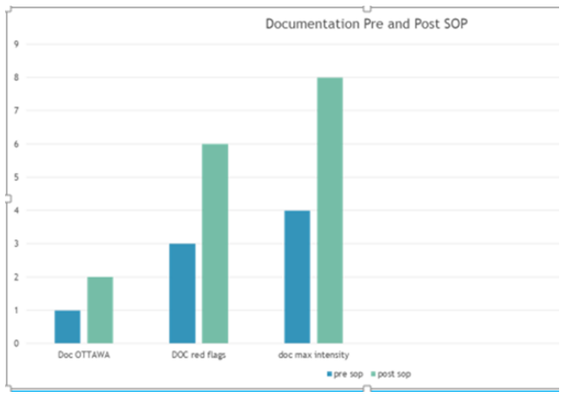


Figure 2 Pre SOP working diagnosis.

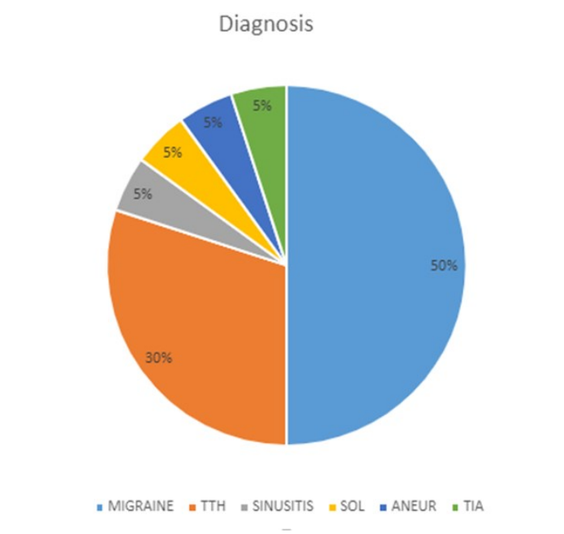
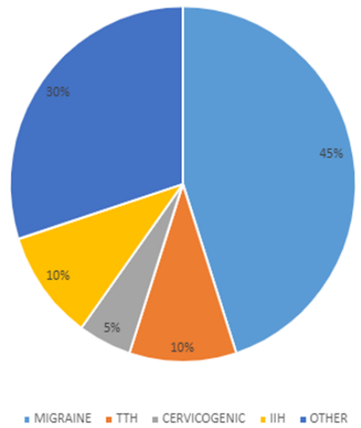


Figure 3 Post SOP working diagnosis.



The main finding from this audit was that the SOP improved clinician's history taking and documentation. Headaches are complex to diagnose and manage, the use of clinical decision rules like the OTTAWA SAH tool and identifying the presence of red flags can help clinicians decide and justify the need for further investigation.

The takeaway message for clinicians reading this is to familiarise yourself with the SOP and apply the Ottawa SAH tool where appropriate and enquire about red flags when taking your patient history, and document your findings in the patients' medical notes. The Ottawa SAH tool and red flags are briefly discussed below, references will be provided at the end of the article for further reading.

Subarachnoid Haemorrhage

SAH is a devastating condition with a high fatality rate, thus the primary goal of emergency physicians is to efficiently and accurately identify or rule out SAH (Godwin et al 2019).

The OTTAWA SAH Rule

The Ottawa SAH rule was developed in 2010 on the basis of data derived from a multicentre cohort study in Canada (Perry et al 2010; Perry et al 2013). It takes into account clinical features that were deemed a high risk for SAH including:

TABLE 1. The Ottawa subarachnoid haemorrhage rule

Age >40 years
Neck pain or stiffness
Loss of consciousness
Onset during exertion
Thunderclap headache (instantly peaking pain)
Limited neck flexion on examination

It is a 'rule-out' tool for use in ED. A recent review of the literature yielded four studies across four countries externally validating the rule, involving a total of 3,317 patients.——Sensitivity across all studies was 100% and specificity ranged from 7.6–13.6%. Therefore it's important to note the use of the Ottawa rule is limited by its specificity (Foley et al 2021) and it therefore cannot be used to diagnose SAH, even in patients whom all criteria are positive.

Only apply the rule in patients aged ≥15 years with:

- New severe headache that reaches maximal intensity within 1 hour of symptom onset
- Presents within 14 days of headache onset
- GCS of 15/15
- No history of fall or head injury within the past 7 days.

DO NOT use the rule in patients with

- New focal neurological deficit / papilloedema
- Previous diagnosis of SAH, brain tumour, hydrocephalus or ventricular shunt
- Previous evaluation with CT and LP for the same headache (or LP within the last 72h)
- Established chronic headache syndrome (≥3 similar headaches over a period of >6 months)
- Confirmed diagnosis of SAH prior to arrival

If any one or more of these clinical features are present, the patient must undergo computed tomography (CT).

Red Flags in Headache

- new-onset daily headache in a patient aged > 50 or with a history of cancer / immunosuppression
- new-onset neurological dysfunction, seizures, or impaired conscious level
- new-onset cognitive dysfunction or change in personality / behaviour
- worsening headache with fever +/- meningism / photophobia / rash (meningitis)
- history of recent (within last 3 months) head trauma
- significant change in character of usual headaches or progressive headache worsening over days to weeks
- acute visual loss (pituitary tumour / apoplexy, raised ICP, idiopathic intracranial hypertension, GCA)
- headache precipitated by coughing, straining, physical exertion, or sexual intercourse with orthostatic / postural aggravation (low CSF pressure / intracranial hypotension) or recurrent early morning headaches with nausea +/- papilloedema (high CSF pressure / intracranial hypertension)
- age > 50 with scalp tenderness, jaw claudication, visual disturbance, systemic malaise (GCA / temporal arteritis → check PV / CRP)
- migraine with atypical aura (duration >1 hour or including significant / prolonged motor weakness)
- unilateral, painful red eye / fixed semi-dilated pupil, hazy cornea, misted vision, haloes, nausea and vomiting (acute angle closure glaucoma)
- headache associated with nausea, lethargy, impaired cognition / concentration +/- multiple affected members of the same household (carbon monoxide poisoning)
- pregnancy or post-partum (pre-eclampsia, CVST, stroke, IIH, RCVS, TTP)

A 2010 prospective study aimed to determine whether the use of red flags accurately identified whether patients (with non-acute headache) should receive neuroimaging. The study was conducted over a five-year period, over that time a total of 530 patients underwent imaging, abnormalities were detected in only 11% of those patients, these results suggest that the utilisation of red flags may lead to an increase in unnecessary imaging (Clarke et al 2010; Ramanayake and Basnayake 2018).

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Godwin, S. A., Cherkas, D.S., Panogos, P.D., Shih. R.D., Byyny, R., Wolf, S.J., (2019) 'Clinical policy: Critical issues in the evaluation and management of adult patients presenting to the emergency department with acute headache: Approved by the ACEP Board of Directors'. *Ann Emergency Medicine* 4, 41-74.

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National Institute for Health and Care Excellence (NICE) (2012) 'Headaches in the over 12s: diagnosis and management (CG150). London [online] available from https://www.nice.org.uk/guidance/cg/cg150 (accessed on 01/06/2020)

Perry, J.J., Stiel, I.G., Sivilotti, M.L., Bullard, M.J., Lee, J.S., Eisenhauer, M., Symington, C., Moretensen, M., Sutherland, J., Lesiuk, H., Wells, G.A., (2010) 'High risk clinical characteristics for subarachnoid haemorrhage in patients with acute headache: prospective cohort study' *BMJ* 28, 341.

Perry, J. J., Stiel, I.G., Sivilotti, M.L., Bullard, M.J., Lee, J.S., Eisenhauer, M., Symington, C., Moretensen, M., Sutherland, J., Lesiuk, H., Wells, G.A., 'Clinical Decision rules to rule out subarachnoid haemorrhage for acute headache' *JAMA* 25 ;310 (12), 1248-55.

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09 Condition of the issue:

Supraventricular Tachycardia

Emma Byrne | Acute Medicine ACP

SVT

(narrow complex tachycardia)

Is a condition of the heart, whereby the electrical impulses are malfunctioning. The features of SVT are a rapid heart rate, normal heart rate being between 60-100 bpm. A patient in SVT will have a fast (tachycardia), sometimes irregular, heartbeat. This condition although rarely life threatening can, if left untreated, develop into heart failure over a period of time. The heart rate can be anywhere between 100 – 200 bpm (or even higher). SVT can occur at rest or whilst exercising/exerting. Supraventricular - meaning above the ventricles - occurs when the pathway from the SA node (the heart's natural pacemaker) is interrupted, or there is an extra pathway or the signal is faulty.

Signs and symptoms

- Tachycardia
- Sweating / dizziness / light-headedness
- Shortness of breath
- Fatigue / lethargy
- Chest pain / palpitations

Causes

Medical conditions such as: Wolff-Parkinson-White; heart failure; chronic lung disease; pregnancy; structural heart disease; Family history of sudden cardiac death; diabetes, also contribute to SVT. Unavoidable risk factors such as, age, race, gender may also play a role.

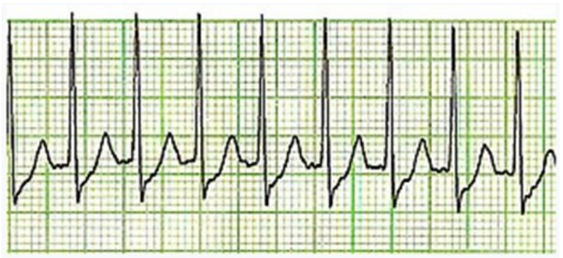
Like with other tachycardia's, SVT may be caused by external factors such as:

- Smoking
- Alcohol
- Stress
- Drugs (prescribed or illicit)

Investigations

As always a good history taking from the patient will give a focus to the clinical exam. A simple ECG will confirm an arrhythmia, of which there are many types. SVT is characterised by a narrow QRS complex and fast rhythm >100 BPM along with a history of associated symptoms and rapid onset.

Bloods tests to investigate underlying causes will also be valuable – routine bloods such as: FBC, U&E, LFTs, TSH and HbA1c.



Lead II electrocardiogram strip showing PSVT with a heart rate of about 180.

Treatment

Whilst SVTs may be uncomfortable and unpleasant for the patient they are rarely life-threatening and can often self-terminate. In cases which require admission and hospital treatment, our first intervention should be a non-invasive, Valsalva Maneuver. This is done by asking the patient to sit or lie down, hold their breath and bear down – as if on the toilet – often we use a syringe and ask the patient to blow into the tip 'trying to move the flange along the shaft'. Straining for 10-15 seconds in order to rest the heart's electrical impulses and thus, return the heart to its baseline. Another non-chemical solution, may be to massage the carotid's (one at a time). This simple act of massaging one side of the neck, also known as Vagus nerve massage, for 5-10 seconds, whilst monitoring the heart may be enough to revert SVT. Asking the patient to cough is another intervention which comes in under the umbrella term of 'Valsalva Maneuver'.

A chemical cardioversion may be used, such as the administration of Adenosine, this is carried out under close cardiac monitoring and by a trained, competent practitioner. This drug is safe in both pregnancy and asthma (with the exclusion of brittle asthma). It is not only used as treatment but is also a useful diagnostic tool.

Given into a large vein using a wide bore cannula > 20g, it is a rapid 6 mg bolus followed by a rapid 20 ml sodium chloride flush. Elevating the arm immediately post administration increases the drug's efficacy, a second and third dose may be required at 12 mg each. Time should be taken prior to administration, to discuss the effects of the drug with the patient – ensure they are aware of the potency and physical symptoms which may be experienced on administration, such as a 'sense of impending doom', sweating, chest discomfort, shortness of breath and headaches – provide reassurance that the symptoms are transient and safe.

Monitoring

Close monitoring through-out, by way if a defib machine, with printability or a 12 lead ECG able to print a rhythm strip, is the gold standard, (if AV block is observed on the rhythm strip this will confirm SVT and not a ventricular issue). If successful the patient will revert into sinus rhythm, see BNF for full administration, doses, cautions and contraindications.

If we haven't already done so, during this stage or after we would usually inform our cardiology colleagues.

For reference

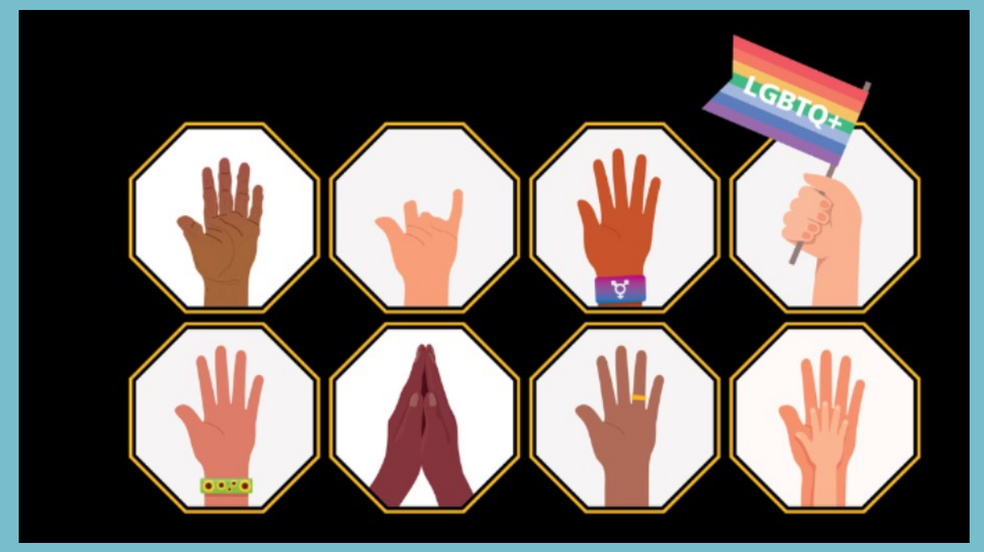
https://cks.nice.org.uk/topics/palpitations/diagnosis/assessment/

https://bnf.nice.org.uk/drugs/adenosine/

https://www.bhf.org.uk/informationsupport/conditions/arrhythmias

https://www.webmd.com/heart-disease/atrial-fibrillation/valsalva-maneuver#1-3

https://geekymedics.com/narrow-complex-tachycardia/



10 Paracetamol Overdose: Update, Audit and Snap

Dr Tom Heaps | Acute Medicine Consultant

Paracetamol Overdose in context

38,775

Admissions 2020-2021 (HES)

25.1%

Consultant referrals 2020-2021 (NPIS)

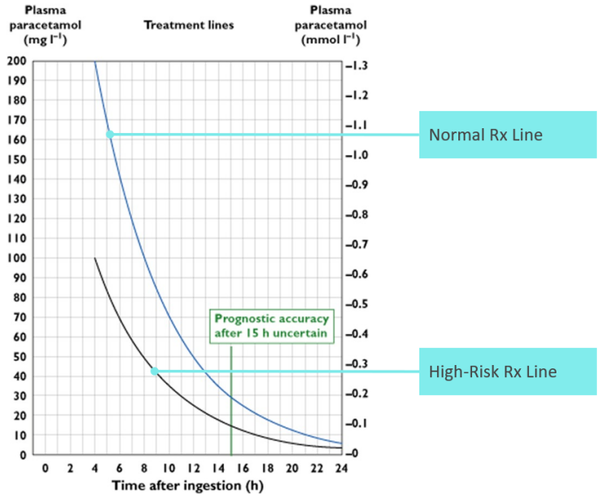
235

Deaths in 2020 (ONS)

Previous treatment regime

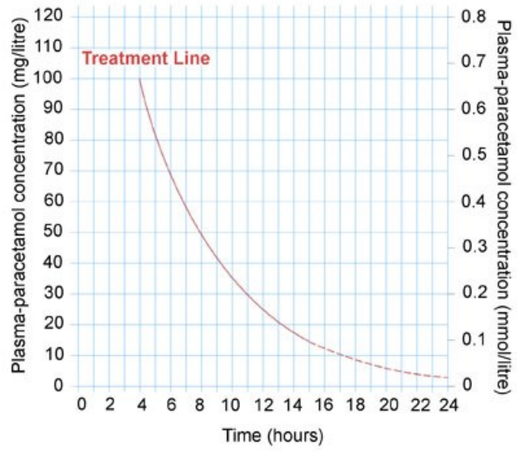
In the UK, paracetamol overdoses are treated using a highly effective antidote, N-acetylcysteine (NAC), which has been administered intravenously using the same complex regimen since the 1970s. This has involved three weight-related doses of NAC given in dextrose over three different time frames. Treatment was recommended for patients who had a timed plasma paracetamol concentration above the '200 mg/L line on a nomogram after a single acute ingestion or a dose of 150 mg kg^{-1} or more within 24 h of a staggered ingestion or unknown timings. Patients with risk factors for hepatotoxicity (poor nutrition, chronic alcohol excess, enzyme inducing drugs) were given NAC if their timed blood paracetamol concentration was above the '100' line. The NAC regimen is associated with a high incidence of adverse drug reaction (ADR).

What are the changes?



In 2012 the UK's Commission on Human Medicines' (CHM) review identified 9 deaths of patients with acute paracetamol OD who did not receive treatment of NAC due to the timed paracetamol concentrations being too low. They recommended the following changes in the management of paracetamol poisoning:

1. No assessment of risk factors for hepatotoxicity
2. Use of single 100mg/L nomogram Rx line
3. NAC for ALL staggered OD
4. Increased duration of initial NAC 15—60 min



Impact (Bateman et al, 2014)

11% increase in proportion of patients admitted

36% increase in the number of patients receiving NAC

0% reduction in number of patients having ADR

£7.8m cost to NHS

ADR's to NAC

Occurs in up to 30% of patients

CLINICAL

Nausea & vomiting
Flushing
Tachycardia
Urticaria / angioedema
Bronchospasm
Hypotension / collapse

RISK FACTORS

Dose dependent (1st bag)
Low paracetamol levels
Females
Asthma or atopy
Family history of allergies

IMPACT

Additional treatment
Extended hospitalisation
Anxiety / treatment refusal
Delays in NAC therapy

IMMUNOLOGICAL

NOT IgE-mediated
'Anaphylactoid'
Previous exposure not necessary
Direct release of histamine

Management of NAC ADR

ACUTE

- **Stop NAC temporarily**
- IV chlorphenamine 10mg
- +/- Nebulised salbutamol
- No role for steroids
- Restart infusion
- Consider halving rate

SUBSEQUENT INFUSIONS

- Previous reactions are NOT a contraindication to future NAC
- Consider slowing 1st bag
- Pre-treatment with IV chlorphenamine +/- ranitidine
- Nebulised salbutamol if previous bronchospasm

Other effects of NAC...

ANALYSER INTERFERENCE

- Underestimation of paracetamol levels by up to 40%
- Only affects some (older) analysers not an issue at SWFT

COAGULATION PROFILE

NAC may prolong INR up to 1.5

- Inhibits activity of vit K-dependent clotting factors
- ALT will remain NORMAL

Scottish and **N**ewcastle **A**nti-emetic **P**re-treatment **(SNAP)** for paracetamol poisoning study (Thanacoody et al, 2013)

Aimed to determine if ADRs from NAC could be reduced by...

- A shorter (12h), simpler (2 bag) NAC regimen or;
- Anti-emetic (ondansetron) pre-treatment or;
- Both of the above combined

Method:

Double-blinded RCT, 222 patients, 3 UK hospitals

Two regimes:

21h NAC Regimen

1	150 mg/kg	1h
2	50 mg/kg	4h
3	100 mg/kg	16h

Bloods taken at 20h

12h SNAP Regimen

1	100 mg/kg	2h
2	200 mg/kg	10h

Bloods taken at 10h and 3rd bag @ 200 mg/kg given over 10h if:

- INR > 1.3
- ALT > 2x or > 100 U/L
- Paracetamol level > 20 mg/L

Results:

N = 222	ANAPHYLACTOID REACTIONS	ADDITIONAL NAC REQUIRED	ALT INCREASE 2x BASELINE
21h STANDARD NAC REGIMEN	31 (27.7%)	11 (9.8%)	11 (9.8%)
12h SNAP REGIMEN	5 (4.5%)	6 (5.5%)	8 (7.3%)

Conclusion:

SNAP regime had 23.2% less anaphylactoid reactions, less patients needed additional NAC and there was a reduction in ALT levels.

Safety and efficacy:

SNAP was adopted into clinical practice in Royal Infirmary Edinburgh, 2015, St Thomas' Hospital, London 2016 and Royal Victoria Infirmary Newcastle, 2016. Pettie et al, 2019, collected patient data prospectively (N = 3340, 21h N = 1488 SNAP N = 1852), to determine the safety and efficacy of the SNAP regime.

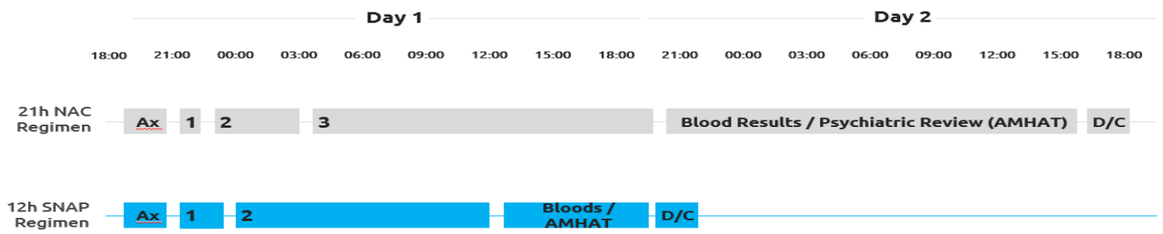
Findings:

N = 3340	ANTIHISTAMINE TREATMENT	EXTENDED NAC TREATMENT	PEAK ALT > 1000 U/L
21h STANDARD NAC REGIMEN	163 (11.0%)	160 (10.7%)	64 (4.3%)
12h SNAP REGIMEN	**37 (2.0%)**	171 (9.2%)	67 (3.6%)

SNAP Benefits

SAFETY	EFFICACY	SIMPLICITY
NNT 10 to prevent one anaphylactoid reaction	No difference in efficacy compared with 21h regime	Simpler regimen with less potential for medication errors and delays
INDIVIDUALIZED	**REDUCED LOS**	**COST SAVING**
Massive OD receives 480mg/kg NAC in 21h with SNAP vs 300mh/kg with 21h regime	Potential to reduce Length of stay by up to 24 h	Reduction In LOS and treatment for ADRs

Representative timeline



SWFT Paracetamol OD Audit

- Retrospective 4-month period (June -Sept 2021)
- 109 adult cases identified via coding a laboratory testing
- 47% 0f patients required treatment

Treatment duration and delay

- Expected time of treatment 21 hrs
- Mean duration 23 hrs 32 mins
- Mean delay bag 1-2 1 hr 24 mins
- Mean delay bag 2-3 1 hr 7 mins

Using SNAP actual treatment duration would be reduced by 10hrs 7 mins

Potential annual benefits from SNAP

114 **Bed days saved** Extrapolating from 38 bed days saved over 4m audit period

£43,092 **Cost Saving** Based on 2019-2020 NCC estimate of £378 per bed-day

15 **ADRs to NAC prevented** Based on 51 x 3 patients treated and NNT of 10 to prevent 1 ADR

Limitations of Audit

- Incomplete data
- Seasonal variation
- Some patients on SNAP may require additional NAC
- Does not account for AMHAT delays

What is the downside of SNAP?

Currently not endorse by the MHRA so use is 'off-licence', however, the Royal College of Emergency Medicine issued a statement recommending the use of SNAP regime should be standard practice in paracetamol overdoses, and it can be found on both Toxbase® and the National Poisons information service websites.

Like many other trusts, SWFT have adopted this regime within the Paracetamol Overdose : Assessment and Management Guideline, which can be found on the Acute Medicine intranet page:

https://extranet.swft.nhs.uk/download_file/view_inline/356/182/SWH_01737_Paracetamol_Overdose_-_Assessment_and_Mgt_v2.0_May_2023.pdf

References:

Thanacoody HK, Gray A, Dear JW, et al. **BMC Pharmacol Toxicol.** 2013; Apr 4:14-20

Pettie JM, Caparrotta TM, Hunter RW, et al. **The Lancet.** 2019; May 11:11-17.

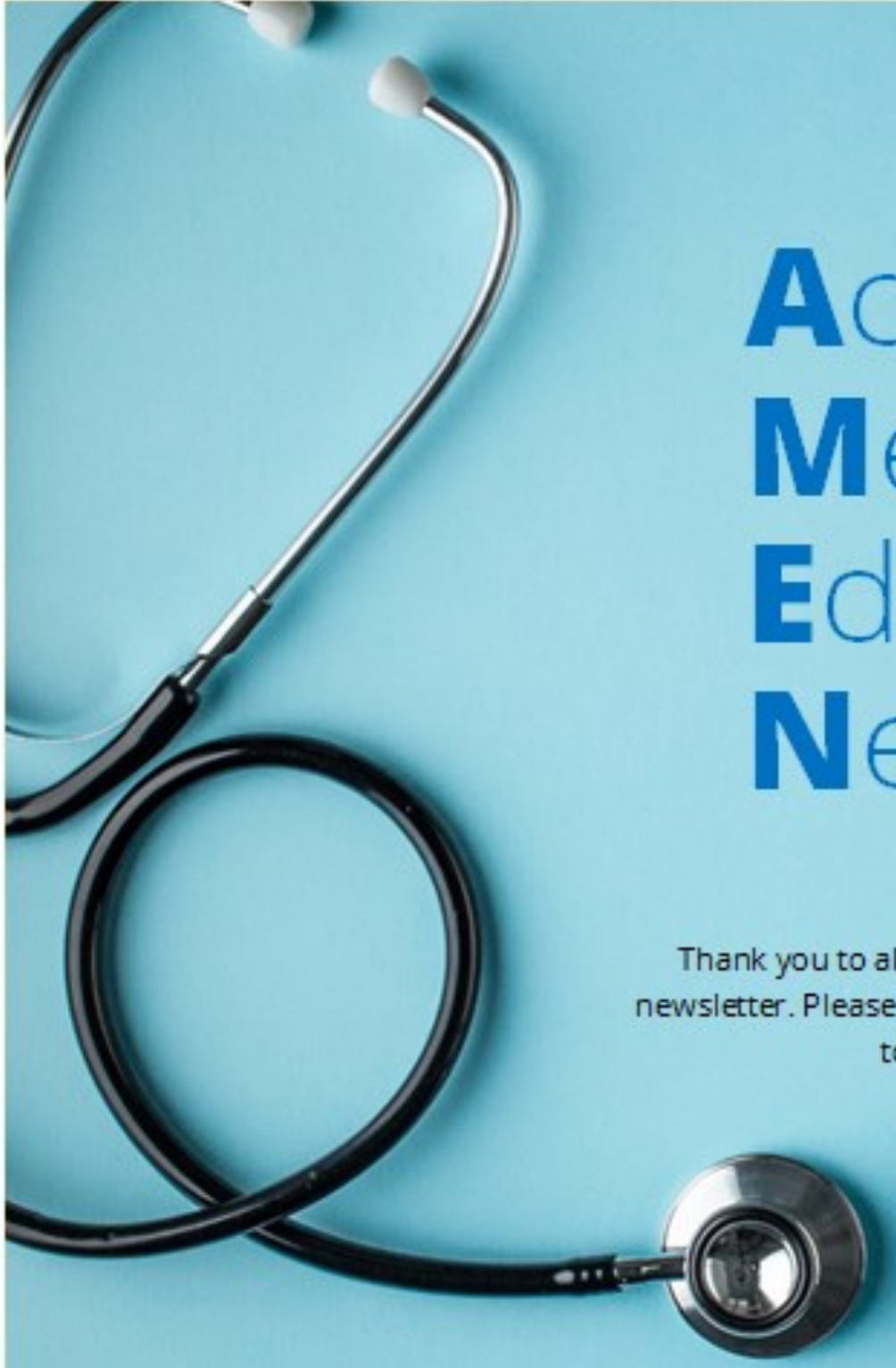
Welcome to your new staff portal!

https://extranet.swft.nhs.uk/

The Staff Portal Project Team have worked hard over the last six months to ensure the transition to the new platform is as seamless as possible. This platform offers you an upgraded experience that will streamline your access to important resources and information.

Your input is invaluable to us! If you encounter any issues or need any changes or updates to your department pages, please email Communications@swft.nhs.uk.

There are many quick tabs to find what you need:



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Acute Medicine Educational Newsletter

Thank you to all the staff that have provided articles for this newsletter. Please contact me via teams or email if you would like to be a part of the next edition.

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