Renal Colic, Toxicology Update and Body Packers

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Renal Colic:

Classic triad of hematuria (absent in 10-15%, and not proportional to size of stone), flank pain and tenderness, yet there is a wide variation in clinical practice, with a propensity to over-image patients

Differential diagnoses of Hematuria: VINDICATE menmonic – Vascular (eg, renal vein thrombosis, AAA), Infectious (eg, UTI, pyelonephritis, renal tuberculosis), Neoplastic, Intoxicants (eg, sulpha drugs, mercury, blood transfusions), Congenital (eg, Polycystic Kidney Disease – PCKD, medullary sponge kidney), Autoimmune (eg, Goodpasture's syndrome, Wegener's granulomatosis, Lupus), Trauma, Endocrine/metabolic (eg, nephrolithiasis); also consider alternate diagnoses for renal colic: AAA, appendicitis, diverticulitis, ovarian torsion

Investigations:

Urine R&M: sensitivity = 85%; sterile pyuria (WBCs alone) is possible in nephrolithiasis, but should mandate further testing if signs of infections are present (eg, bacteruria, fever, general malaise)

Minimal workup (no blood work, no imaging) might be acceptable in young (<50y.o.) and previously healthy patients if the diagnosis is certain, especially if previously imaged (current study assessing whether this is a safe approach is ongoing)

More workup is needed in older patients, those with comorbidities, and potentially those with first-time presentations; remember that normal creatinine level does not completely rule out severe unilateral kidney damage if the contralateral kidney is fully functional

Non-contrast CT scan:

Very sensitive even for small stones (98%; and 95% for low-radiation protocols), but delivers significant radiation to patient (2 mSv for low-dose protocols, which is 200 times a CXR), especially given that many patients undergo repeated scans over their lifetime; a study quoted a 10% rate of other diagnoses found on CT, such as appendicitis, diverticulitis and ovarian torsion (but also incidental BPH and adenomas) – [Katz et al. Urology 2000;56(1):53-7]

Should be considered in elderly patients, those in which an alternative diagnosis is suspected, or those with important comorbidities: solitary kidney, renal transplant and septic patients

Can be considered in all first-time presenters, although a Scandinavian study [Lindqvist et al. Scand J Urol Nephrol 2006;40(2):119-24] showed that no adverse outcomes occurred when imaging was delayed by 2-3 weeks when the patients followed up with urology; some patients even passed their stone in this time interval, thereby avoiding imaging altogether

KUB x-ray:

Reasoning is that a KUB will identify the stones that may be amenable to lithotripsy and medical expulsive therapy (90% of stones have calcium in them and are therefore visualized)

However, large radiation doses are involved (1/3rd of a CT scan) and the urologist might repeat it anyways before lithotripsy to see whether the stone has passed and the procedure still indicated

Ultrasound:

Sensitivity is poor for detection of stones, especially in the middle 1/3rd of the ureter, but has good sensitivity to know whether interventions will be needed; consider in women if gynecological diagnoses are considered, and bedside ultrasound to rule out other diagnoses (eg, AAA)

Management:

Analgesia with NSAIDs (eg, rectal diclofenac or indomethacin, which cause a decrease in ureteric peristalsis and pelvic pressures by their prostaglandin-inhibition mediated decrease in GFR) plus opioids if necessary; patients who cannot be rendered pain-free, or who have abdominal pain or tenderness per se (as opposed to flank or groin pain) might need further investigation for other Dx

Medical expulsive therapy (alpha-adrenergic blocker like tamsulosin 0.4mg po qd, or calcium-channel blcokers, which block ureteral smooth muscle contraction) may help reduce the time to passage of stone by 40%, especially in moderate-sized stones

Average passage time for 4-5mm stone is 2wks; the size, location, shape and degree of obstruction of a stone determine whether it will pass (proximal stones >10mm rarely pass spontaneously)

Patients may be instructed to strain their urine and collect the stone(s), and follow up with their GP, who can analyze the stone and recommend dietary modifications

Referral to urologist should be considered when significant comorbidities are present, or for large-sized stones; admission should be considered for intractable pain and/or vomiting, if infection/ sepsis is present (urgent surgery may be required), or for solitary kidney or renal transplant

Toxicology Update:

Intravenous lipid emulsion therapy:

Antidote originally used for local anesthetic toxicity, and now for lipophilic drugs such as TCAs, bupropion, β-blockers, Ca^{2+}-ch. blockers, and drugs with lipid aqueous partition coefficient >2-3

Also called TPN (total parenteral nutrition); composed of soybean oil, egg yolk, phospholipid glycerin, aluminum and water; suggested mechanism is that it pulls away the lipophilic drugs from the tissue (thus decreasing damage) and increases the energy supply of free fatty acids to the myocardium

Indications: when other methods have failed (ACLS, usual antidotes) in setting of lipophilic drug OD

Practical kit to use in your ED (visit www.lipidrescue.org):

500cc bag of pre-packaged 20% TPN solution (stored at room T°), 50cc syringe and IV tubing

Use: IV bolus push of 100cc (or 1.5ml/kg, drawn out and pushed twice with 50cc syringe), then 400cc (the rest of the bag) over 15min (infusion of approximately 0.25ml/kg/min)

Safety concerns: no reported adverse events in the literature, but be careful in egg or soybean allergies and disorders of fat metabolism; theoretical concerns of worsening myocardial infarction and risk of fat embolus

Hydroxycobolamine for cyanide (CN) treatment:

Vitamin B12 precursor, used in fire victims with smoke-inhalation injury

Advantage over the "cyanide antidote kit":

The CN-kit contains nitrates as well as sodium thiosulfate, the former of which is dangerous in smoke-inhalation toxicity due to concomitant carbon monoxide (CO) poisoning: nitrates form

methemoglobinemia (which will adequately treat CN toxicity), but also carboxyhemoglobin (because of the CO poisoning), causing high levels of abnormal hemoglobins and leading to severe hypoxia that can be life-threatening (on top of the vasodilation and hypotension caused by the nitrates)

Hydroxycobolamine, however, does not form methemoglobinemia, and therefore is not dangerous in concomitant CN and CO poisoning

When to suspect CN on top of CO poisoning:

Fire victims covered in soot with triad of (1) hypotension, (2) metabolic acidosis (with anion-gap) and (3) elevated lactate level (>8-10) -> such patients should be treated empirically for CO *and* CN toxicity: hydroxycobolamine is the agent of choice, and nitrates should be avoided (the sodium thiosulfate agent also present in the CN-kit may be used as well)

Other situations: fumigators, photographers, jewelers, nail polish use, apricot pits ingestion or iatrogenic nitroprusside overdose

Use: Hydroxycobolamine 5g IV (needs to be diluted) over 15min (both 2.5g vials of the "Hydroxycobolamine kit" need to be given), repeated up to 15g total if necessary
Pediatric dose is 70mg/kg per infusion

Side-effects: chromoturia (red discoloration of urine, and possibly of blood drawn), possible hypertension (due to NO scavenging properties – do not treat the hypertension with nitrates)

High-dose insulin therapy in Calcium-channel blocker (CCB) overdose

Old therapy, but people remain reluctant to use in recommended doses:

Regular (short-acting) insulin at 1u/kg bolus (ie. 70 units!), followed by 0.5u/kg/hr infusion

Glucose (1 vial of D50W, or 25g of dextrose) may be given concomitantly, and glucose (and potassium) should be carefully monitored given that a falling glucose level means that the CCB overdose is adequately resolving

Mechanism: CCBs block the pancreatic insulin-releasing cells (that are calcium dependent), causing hypoinsulinemia and preventing the myocardium from using circulating glucose that it would normally use after the free fatty acids have been depleted in a state of shock

Indications: "Slow and low" – bradycardic and hypotensive patient (due to CCB, beta-blocker, digoxin or clonidine overdose) with HR of 30-40 and sBP of 70-80; use in conjunction to standard ACLS algorithm (including atropine) and other antidotes (calcium, glucagon, fluids)

Body Packers:

Body packers (or internal carriers, drug mules) pre-meditatively insert packets in their rectum, vagina or by swallowing them in order to smuggle drugs across borders, then excrete the drugs and exchange it for money; body stuffers, in contrast, swallow small amounts in an unplanned way to avoid getting caught by police

Cocaine (as well as hashish, heroin, methamphetamines) is usually transported, in doses up to 100 packets of 10g (1g being the lethal dose), in well-sealed packet surrounded by layers of hard wax

Investivations:

Urine tox screen: not generally recommended to guide management, as sensitivity is only 37%, and active metabolites will be detected up to 4hrs only in acute ingestions (thereby creating false negative), and up to 4-5d in chronic users (false positive); may be useful if initial urine tox screen is negative and subsequent screen becomes positive, indicating a leaking or ruptured packet

Abdominal x-ray: 85-90% sensitive, and useful to detect bowel obstruction

Bedside ultrasound: quick and safe, but very operator and patient dependent; consider it's use in patients presenting in severe, unstable cocaine toxicity with no obvious history of packing

CT abdomen or barium contrast x-ray: best sensitivity, although not 100%, so may miss some packets; may be used to confirm excretion of all packets

Clinical features of cocaine intoxication:

Cocaine's mechanisms include re-uptake blockade of norepinephrine, dopamine and serotonin (causing hyperthermia, hypertension and tachycardia, and CNS agitation, seizures or coma), sodium-channel blockade (causing widening of QT interval and potential arrhythmias like VT), as well as endothelin production and nitrous oxide scavenging (causing vasoconstriction and vasospasm, with further hypertension and potential STEMI and aortic dissection)

Management: Supportive care with benzodiazepines (to calm the patient, reverse the hyperthermia and hypertension, and control seizures) and active cooling of the patient; the hypertension can be treated with nitrates or phentolamine, but β-blockers (even non-selectives like labetolol) should be avoided for fear of unopposed alpha-adrenergic activity; widened QRS (>120ms) should be treated with $NaHCO_3$ (the Na^+ treats the Na^+-blocking properties); VT should be treated with $NaHCO_3$

Management of cocaine body packers:

Asymptomatic patients (90% of patients):

After patient consent obtained, use whole-body irrigation (WBI) through NG tube, at 250cc of PEGlyte q10min, up to 4L over around 2.5-3hrs

Multi-dose activated charcoal (1g/kg) may also be considered given that it will adsorb cocaine from possibly leaking packets in the GI tract

Patients are usually considered to have passed all packets when they have passed 3 packet-free stools (confirmatory CT scan or barium x-ray may be undergone, although not 100% sensitive)

Patients may be discharged in the hands of RCMP officers (with clear instructions on what symptoms to look for and when to come back) before all packets have passed, after a period of 24hrs

Symptomatic patients:

Symptomatic patients need aggressive decontamination treatment as above, as well as cocaine-specific detoxification methods (benzodiazepines, cooling, nitrates/phentolamine, $NaHCO_3$)

Immediate Surgical consult mandated, as symptomatic patients need emergent laparotomy

Indications for surgical consult:

Symptomatic patient, bowel obstruction, failure to pass all packets (possibly due to impaction) either after the 24-hr observation period, or after a 6-day trial while in RCMP's facilities