Emergency medicine

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Avoiding litigation in emergency medicine

The conference opened with a discussion on how to avoid being sued in emergency medicine. Unsupervised, inexperienced and tired juniors are particularly at risk of being sued, and common areas for mishap include failure/delay in diagnosis and medication errors. A significant minority of errors in one series resulted from procedural complications.

Specialties can be ranked in terms of their risk of accident and resultant litigation, with obstetrics and plastic surgery at highest risk, and surgical subspecialties and radiology next. Medicine ranks alongside pathology, psychiatry and anaesthesia while geriatrics is seen as no more risky than public health medicine.

Common claims made to the Clinical Negligence Scheme for Trusts (CNST) in the area of emergency medicine concern failure to diagnose, wrong diagnosis and errors resulting from investigation oversights and treatment mistakes.

The Harvard Medical Practice Study[1,2] found that iatrogenic and hospital-acquired adverse events occurred in 4% of admissions in the USA. Of the 40,000 patients in the study, 1,600 were harmed, with death and permanent disability occurring in 14% and 7%, respectively. The authors attributed these events to medical error in 50% of cases of harm, and to negligence in 33%.

In medical admissions unit work, the following advice was given:

- patients should be dealt with one at a time and pressures not to admit resisted where necessary
- interactions with relatives should take place after due consideration and not in haste
- in cases where there were 'do not resuscitate' orders, reasons should be carefully documented
- all paperwork, including ambulance notes and letter to the GP, must be reviewed.

Further advice was to listen to nurses and be wary of mental exhaustion.

A 'dirty dozen' of common errors in diagnosis in emergency medicine are:

- cardiovascular system: myocardial infarction, infective endocarditis
- central nervous system: subarachnoid haemorrhage, meningitis, subdural haematoma
- endocrine: diabetic ketoacidosis, hyponatraemia
- gastroenterology: occult neoplasm
- respiratory: asthma, pneumonia, pulmonary embolism (PE)
- pharmacology: medication errors.

If complaints are received, the notes should be studied, held overnight and then re-reviewed before committing anything to paper. All relevant documentation should be considered in the response, including nursing notes and prescriptions. Notes should never be altered. When commenting on a complaint, opinion should be separated from fact, the term 'negligence' should be avoided, and the reply should be addressed to the individual who requested the report, usually a member of the complaints department.

Only about 10% of complaints result in successful negligence claims. In order for negligence to be proved, it must be established that there was a duty of care and this was breached, 'liability'. Further, it must be proved that harm resulted from the breach of duty of care, ie 'causation'.

Acute coronary syndrome

For an acute coronary event to fulfil the definition of a myocardial infarct (MI), there must be both an enzyme rise and a history of chest pain or the event occurring during coronary intervention. Electrocardiogram (ECG) changes, or events occurring

Conference programme

How to avoid getting sued in emergency medicine
Dr Chris Evans, Royal College of Physicians and Medical Defence Union

Emergency ECG interpretation
Mr Francis Morris, Northern General Hospital, Sheffield

Acute coronary syndrome
Dr Stephen Saltissi, Royal Liverpool University Hospital

Management of community acquired pneumonia
Dr Derek Bell, Royal Infirmary of Edinburgh

Acute airflow limitation
Dr Simon Watkin, Norfolk and Norwich University Hospital

Upper GI haemorrhage
Dr Ian London, Leighton Hospital, Crewe

Acute pharmacological emergencies
Dr Robin Ferner, City Hospital, Birmingham

Acute confusion
Dr Solomon Almond, Royal Liverpool University Hospital

during revascularisation, are necessary to fulfil new diagnostic criteria set out by the European Society of Cardiology[3] and American College of Cardiology.[4]

History of chest pain can be typically cardiac, atypically cardiac or non-cardiac. When assessing the history of chest pain, the overall risk profile of the patient should be considered. Examination can establish signs of pre-existing cardiac disease or results of acute coronary syndrome (ACS) but is frequently normal. Haemodynamic compromise is important in risk stratification as is ECG and troponin. Initial ECG is normal in 35% of ACS cases. A number of risk stratification tools are in use.

The bedrocks of management of non-ST elevation ACS are drug therapy, intervention, subsequent investigation and secondary prevention. Drug therapy consists of anti-platelet therapy (aspirin, clopidogrel, GPIIb/IIIa inhibitors), low molecular weight heparin and 'anti-ischaemia' agents (β-blockers, nitrates, calcium channel blockers, nicorandil). Statins and angiotensin-converting enzyme inhibitors play important roles. GpIIb/IIIa studies suggest that the patients who benefit most are those given the drug early, particularly those undergoing revascularisation and with a postive troponin and ongoing symptoms/evolving ECG changes.

Percutaneous revascularisation and bypass grafting play an important role in the management of ST elevation MI, as does thrombolytic therapy. Initiatives including expansion of revascularisation and pre-hospital thrombolysis are improving treatment for MI patients. The advantages of managing ACS patients in a dedicated, cardiologist-led unit were set out.

Community-acquired pneumonia

Community-acquired pneumonia (CAP) predominantly affects those at the extremes of age. About a third of those with CAP are hospitalised, amounting to 1–4 per 1,000 population. Mortality in the community is less than 1% but climbs to 10% for those admitted to hospital and 40% for patients in intensive therapy units (ITUs).

While a proportion of CAP is caused by atypical pathogens, the term 'atypical pneumonia' can be misleading as the presentation frequently differs little from that of CAP caused by typical pathogens. *Streptococcus pneumonia* and *Haemophilus influenzae* remain the commonest overall pathogens, while *Legionella pneumophilus* and *Staphylococcus aureus* account for a disproportionate amount of those admitted to ITU, 17.8% and 8.7% respectively; they cause a minority of infections in those admitted to general wards (3.7%, 2.1%). *Mycoplasma pneumoniae, Chlamydia psittaci, Coxiella burnetti* and viral pathogens are important but rare causes of CAP.

In addition to considering the aetiology of CAP, initial management includes oxygen and fluids. Attention must be paid to illness severity and co-morbidity. Poor prognostic markers include:

- age: >65
- co-morbidity: renal failure, chronic obstructive pulmonary disease, congestive cardiac failure etc
- clinical: respiratory rate >30/min; blood pressure <90/60; temp ≥38.3; abbreviated mental test <8/10
- investigations: white cell count <4,000 or >20,000; PaO_2 <8 kpa or SaO_2 <92%; Ur >7; blood culture positive
- chest X-ray: multilobar.

Initial investigations include culture of blood, sputum and pleural fluid as well as urine and serology for atypical pathogens.

Mainstays of initial treatment for CAP, whether managed in hospital or in the community, include penicillins, macrolides and quinolones with enhanced pneumococcal activity (such as levofloxacin or moxifloxacin). Severely ill patients require intravenous therapy, and co-amoxyclav or third generation cephalosporins with a macrolide are appropriate initial agents. Subsequent antibiotic management must be guided by culture results and microbiological advice.

In the case of a non-responding patient, the following questions should be asked: whether the condition is infective, whether the lung is the site of infection, what the pathogen is, and whether the patient is immunocompetent. All sites should be re-cultured and computed tomography imaging should be considered.

Non-infectious conditions that can mimic CAP include PE, pulmonary oedema, Wegener's granulomatosis and malignancy.

Management of acute upper gastrointestinal haemorrhage

Upper gastrointestinal (GI) bleeding is the most common GI emergency, occurring in 1 in 1,000 UK adults per year, accounting for 25,000 emergency admissions. The mortality is 8–10%, with the elderly being disproportionately at risk of poor outcome. Peptic ulceration accounts for upper GI bleeding in 30–35% of cases and varices in 5–10%; no cause is identified in at least 20% of cases.

Important clues as to the aetiology of a bleed are available from the history: retching suggests Mallory-Weiss tear; weight loss may indicate malignancy; and alcohol excess suggests the possibility of varices.

Strictly speaking, the term 'haematemesis' implies the vomiting of fresh blood, which indicates a site of bleeding proximal to the jejunum. Melaena suggests at least 60 ml of blood loss into the upper GI tract and may also occur in more distal bleeds, including those affecting the right colon. Haemochezia (passage of fresh blood per rectum) occurring in the context of upper GI bleeding suggests extensive blood loss.

Non-steroidal anti-inflammatory drug usage, low dose aspirin and *Helicobacter pylori* positivity confer significant risk of upper GI bleeding.

In the assessment of patients with recognised or suspected upper GI haemorrhage, it is necessary to identify those at highest risk immediately, particularly those presenting with hypovolaemic shock. An early sign of cerebral hypoperfusion is agitation; this progresses to obtundation at later stages. With respect to haemodynamics, pulse is a much more sensitive indicator than blood pressure. Tachycardia occurs after loss of 12–15% blood volume whereas hypotension typically does not

occur until there has been 30% loss. Postural hypotension can occur in the context of more modest blood loss of 10–15% or 1,000 ml. Urine output and respiratory rate are also important parameters to be considered in assessing likely blood loss. The Glasgow Admission Score takes account of initial haemoglobin, blood urea, haemodynamics and mode of presentation and aims to identify those requiring early intervention. Rockall scoring also takes account of endoscopy findings and co-morbidity and can be used as a predictor of re-bleeding and poor outcome.

Initial management of upper GI bleeding requires adequate venous access, centrally for significant bleeds. A urinary catheter and nasogastric tube (to clear blood from the stomach) should be considered. It is important to check liver function tests, clotting and to cross-match the blood for patients with large bleeds. Clinical shock requires resuscitation with blood. The initial aim should be to maintain the haemoglobin in the region of 10 g/dl.

Upper GI endoscopy (OGD) remains pivotal in the investigation and treatment of upper GI bleeding and identifies the site of bleeding in 80% of cases. Findings at OGD are strong predictors of outcome; in the case of an actively bleeding ulcer, the risk of re-bleeding is up to 90%, but if the ulcer has a clean base this is reduced to 2%.

Endoscopic therapy for upper GI bleeds has been shown to reduce the risk of re-bleeding/referral for surgery by 75% and to offer a 40% reduction in mortality. Sclerotherapy with adrenaline injected around the ulcer and thermal therapy with a heater probe are common modalities of endoscopic therapy; thrombin or glue applications are also used occasionally. There is a modest role for mechanical devices deployed endoscopically, such as clips over the bleeding point.

An important study by Lau and co-workers[5] showed a significant reduction in re-bleeding, surgery or death with the use of full acid suppression with intravenous omeprazole (80 mg initial dose followed by 8 mg/hour for 72 hours). It should be noted that Lau looked at a Hong Kong population who tend to be younger, have less co-morbidities and less use of non-steroidal anti-inflammatory drugs than those with GI bleeding problems in the UK. Intravenous H2 receptor antagonists offer no benefit in the setting of a bleeding duodenal ulcer and modest benefits for gastric ulcers.

Embolisation of bleeding points delivered non-invasively by interventional radiologists offers results comparable to surgery in terms of re-bleeding rates and death, and is often the only option in very high-risk patients.

Bleeding from oesophageal varices still causes massive mortality – up to 50% following the first episode. Early OGD is crucial and the mainstays of endoscopic therapy are band ligation and/or sclerotherapy by injection of ethanolamine. Use of glue in this setting requires further evaluation.

Pharmacological emergencies

Three main areas of interest amongst medication-related emergencies were highlighted: adverse drug reactions, medication errors, and deliberate self-harm. As the number and range of drugs in use has widened, drug effects have taken over from conditions like syphilis as the great imitators of other diseases, and almost any presentation can be due to drugs.

Three general principles underpin the treatment of poisoning:

- separate – poison from patient
- support – vital and higher functions
- specific – antidote maybe available.

The importance of toxbase and the National Poisons Information Service as information repositories were highlighted. Induced emesis and gastric lavage are not now recommended in poisoning, whereas early deployment of activated charcoal is useful for some cases. Opiates, tricyclics, paracetamol and stimulants are significant culprits in serious poisoning, and carbon monoxide poisoning remains an important problem.

Methadone was specifically mentioned, as it is slow acting and, given its presentation as a brightly coloured liquid, a risk for childhood poisoning.

While cases of poisoning due to tricyclic antidepressants have decreased over the last 10 years, they still cause significant morbidity and mortality; the cardinal signs are dilated pupils, fits, coma and tachyarrythmias.

Ecstasy poisoning is manifested as suggested agitation, hallucinations and abnormal jaw movements; dantrolene has some advocates, management being largely supportive. Paracetamol overdose should be approached by administration of N-acetyl cysteine with particular attention paid to high-risk patients (such as those with pre-existing liver disease).

The issue of 'body packing' (swallowing sealed packages of drugs) was highlighted. Ruptured packages can present as emergency poisoning, particularly with opiates and cocaine. If any doubt exists, radiology input should be sought and surgeons may have to be involved to remove the drugs.

References

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