Aetiology of cerebral oedema in diabetic ketoacidosis

The excellent evidence based review of the emergency management of diabetic ketoacidosis (DKA) in adults by Hardern and Quinn perpetuates the premise that “unnecessarily large volumes of intravenous fluids should be avoided because of the high case fatality rate of cerebral oedema”. This presupposes that the rate of fluid delivery is causally related to the development of cerebral oedema, which has not been proved. The large 15 year paediatric study in the USA that analysed 6977 hospitalisations for DKA found among the 61 cases of cerebral oedema (0.9%) that after multiple logistic-regression analysis with random and matched controls, the only variables statistically associated with cerebral oedema were higher initial serum urea nitrogen concentrations and lower partial pressures of carbon dioxide at presentation. In addition, smaller increases in serum sodium concentration during treatment and the use of bicarbonate were also implicated. Importantly, the rate of fluid, sodium, and insulin administration were not associated with the development of cerebral oedema, nor was the initial serum glucose or its rate of change.

Clearly these findings relate to patients aged 18 years or less but most occurrences of cerebral oedema in DKA are in children and adolescents, with only rare cases in adults. However, the underlying aetiology should be no different. One unifying hypothesis is that the cerebral oedema is related to cerebral vasconstriction, brain ischaemia, and hypoxia, as hypopcapnoea causing cerebral vasconstriction and extreme dehydration would both decrease cerebral perfusion. In addition, as children’s brains have higher oxygen requirements than adults this may explain their unique susceptibility.

Perhaps clinicians should focus more on recognising the warning signs of cerebral oedema such as headache, lethargy, and deterioration in conscious level, prior to seizures, incontinence, pupillary changes, bradycardia, and respiratory arrest as brain stem herniation occurs. Early hyperosmolar treatment and presumably supplemental oxygen with exemplary supportive care are then essential. Finally, accepting that cerebral oedema may well be idiopathic rather than iatrogenic could have important medicolegal connotations too.

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Contact lenses can compromise the corneal epithelium and act as pathogenic vectors, facilitating the development of bacterial keratitis. Most corneal abrasions heal quickly when treated with topical antibiotics, which act as lubricants and antimicrobial agents. However, in contact lens wearers there may be rapid progression to corneal scarring or even perforation.

Two patients with contact lens related corneal abrasions, who were initially treated with topical fusidic acid or chloramphenicol, have presented with corneal stromal abscesses. The abscesses developed 12 hours and three days respectively after diagnosis of simple corneal abrasion. Visual acuity was perception of light and hand movements. Both required admission for intensive topical fortified gentamicin and guttae cephalosporin.

Pseudomonas aeruginosa and Proteus were grown, which were resistant to chloramphenicol and fusidic acid. Best corrected visual acuity were 2/60 at presentation. Resolution of the infections; one patient has proceeded to corneal grafting.

A 15 year study of resistance in bacterial isolates from corneal scrapings found that 20.4% of isolates were resistant to chloramphenicol1 (54% of Gram negative organisms), with a significant increase in resistance during this period. Once microbial keratitis is established, a combination of topical fortified aminoglycoside and cephalosporin, or fluoroquinolone is indicated2; no trend for increasing resistance to these antibiotics was observed in the aforementioned study.

Contact lenses are the most important risk factor for the development of bacterial keratitis. In the emergency department, a history of contact lens wear should be sought, with urgent review of worsening abrasions. We advise that all contact lens related red eyes should be referred to the ophthalmology department, as clinical signs may initially be subtle and corneal scraping may be warranted. Timely commencement of guttae oloxacon with the first sign of infection, may greatly reduce the chance of poor outcome.

Contributors
Shane Quin treated the second patient, reviewed the literature and wrote the paper. Jeffrey Kwartz treated both patients and contributed to the discussion of core ideas. He was the supervisor and is the guarantor.

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References
Sudden death and the myth of CPR


Most emergency physicians will sometimes recognise a feeling of futility during cardio-pulmonary resuscitation (CPR)—the algorithm is followed despite the fact that most of those present know the attempt is doomed to failure, or frankly inappropriate.

Stefan Timmermans is a Belgian healthcare sociologist who spent time in American emergency departments observing the rituals surrounding CPR. His book questions the notion of CPR for all, and the over-optimistic programme of survival from out of hospital cardiac arrest that is portrayed in the media, and by some medical authorities. The book describes the attitudes and feelings of doctors, nurses, and paramedics, their definitions of good and bad resuscitation attempts, and the way in which they feel constrained by guidelines and lawyers.

The chapters are wide ranging and include the evolution of resuscitation techniques, death awareness, and what constitutes a “good” death, as well as discussion on advance directives and the presence of relatives during resuscitation attempts. The author divides resuscitation attempts into four distinct categories: the legal death trajectory, where resuscitation is performed mainly as a legal matter; the elite death trajectory, where the victim is presumed to have high social viability and receives aggressive resuscitation irrespective of clinical viability (for example, the young); the temporary stabilisation trajectory, in which the patient is resuscitated despite the fact that the short term prognosis is poor; and the stabilisation trajectory, in which prompt resuscitation leads to a better outcome.

The book is written from a sociologist’s perspective, and therefore does not aim to provide answers—just observations. Yet despite the North American setting, it raises questions that are highly applicable to UK practice, and this book should be required reading for all ALS providers.

J E France

Handbook of paediatric emergency medicine


Knowledge is a process of piling up facts; wisdom lies in their simplification. Martin Luther King, Jr (1929–1968)

Upwards of two million children will attend accident and emergency departments in the United Kingdom every year. Many thousands more will attend general practice for advice or treatment after acute illness or injury. Large numbers of practitioners in many different settings therefore need to be prepared to deal with children with a variety of urgent and emergency conditions. As an old Chinese proverb states “Small children do not pretend to be sick.” The problem is that the vast majority of children have minor to moderate illness, much of which is self limiting. Indeed many of the injured children require little more than symptomatic relief and general supportive care.

The problem therefore is identifying the wheat from the chaff. In other words, how does one identify the critically ill child, or the child who is brewing something serious? Age and experience help. Certainly knowledge is useful. More often the wisdom of Solomon is required. There is no doubt that experience brings greater wisdom, and with it ability to deal with children effectively. I suppose that is really what I like about this book. The authors have brought their collective experience and wisdom, gathered over the years (I am not brave enough to state how many, but I know it is considerable!) to produce an extremely readable text that is well laid out and well presented. The salient features are highlighted in boxes and the use of diagrams is good. I personally would have liked to have seen more radiographs and clinical pictures, but then again this may not be the purpose of the handbook. This may best be left to a colour handbook. The Swan Ganz catheter is placed in its correct context, alongside alternatives including the pulse induced continuous cardiac output monitor. I was also pleased to see the role of corticosteroids set out in accordance with current thinking on the treatment of sepsis.

There is a review of the evidence on non-invasive ventilation in COPD. Activated protein C is (to this reviewer at least) a very new treatment in septic shock, and its brief role is considered in the context of child intensive care.

T Beatlie

Core cases in critical care


In 230 pages and a few monochrome illustrations this paperback covers the top 20 clinical cases that are likely to occur in the top 20 clinical cases that are likely to occur in every intensive care unit. The authorship is a reassuring collection of UK intensivists, a who’s who of the Intensive Care Society. I liked the standardised format; case histories are followed by discussion on the main issue with reference to pathophysiology, treatment options, and outcome. A panel of key learning points rounds off each chapter, and the recommended further reading is appropriate and proportionate.

A number of the cases bear upon emergency care and many are set in the resuscitation room. The importance of securing the ABCs is emphasised before discussion of theoretical concepts, not always the case in books of this sort. This reflects the interests of the authors, many of whom are active in education at the interface between intensive care and emergency medicine. Relevant cases include burns, trauma, and overdose, but pyrexia is included uncomfortably in the chapter on status epilepticus. The chapters are up to date; the roles of inhaled nitric oxide and platelet aggregation inhibitors are highlighted. And there is a review of the evidence on non-invasive ventilation in COPD. Activated protein C is (to this reviewer at least) a very new treatment in septic shock, and its brief role is considered in the context of child intensive care.

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