**THE ART OF MEDICINE**

“Safeguarding” in the community

As doctors, we follow structured guidance if safeguarding concerns arise clinically. But what about in the community? In a supermarket you see a parent strike a preschool child on the face—the child’s whimpering speaking of familiarity. Corporal punishment is potentially illegal if there is “actual body harm”: non temporary marking of the body. A mark on a child’s face from such a strike would lead to a child protection investigation if noted at school or by a health professional. But here, with neither evidence nor a way to trace the child, do you intervene? What could help the child?

In the structured “clinical” approach to safeguarding, doctors can instigate multi-agency involvement.

The General Medical Council’s guidance for good medical practice states, “You must inform an appropriate person or authority promptly of any reasonable concern that children or young people are at risk of abuse or neglect.” A moral obligation on doctors is also expressed: “You must offer help if emergencies arise in clinical settings or in the community, taking account of your own safety, your competence and the availability of other options for care.” How do we put these principles into action?

Corporal punishment in public is not common, but the bigger worry—what happens behind closed doors—unfortunately is. When concerns are obvious, reporting should be a given.

But when concerns aren’t glaringly obvious, when we have no means of following up, and when corporal punishment is not entirely prohibited, how can we reconcile our feelings of unease if we witness such a scenario, when it isn’t clear what we can do to ensure the child’s safety?

Ala Fadilah, Paediatrics Department, York Hospital, York, UK

We welcome contributions to this column.

Please email samuel.parker@bmj.com

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**CLINICAL UPDATE**

Type 1 diabetes in adults

All adults with type 1 diabetes should be offered a structured education programme of proven benefit six to 12 months after they are diagnosed, the National Institute for Health and Care Excellence (NICE) has said. Adults who have not attended a programme should be offered one “any time that is clinically appropriate and suitable for the person, regardless of duration of type 1 diabetes.”

Vaginal birth after previous caesarean

Most women who have had a lower segment caesarean delivery can go on to have a planned vaginal birth, the Royal College of Obstetrics and Gynaecology has said. However, planned vaginal birth should not be offered to women with previous uterine rupture or classic caesarean scar, and caution is needed for women with complicated uterine scars, the college says.

Duty of candour for surgeons

Surgeons may want to tell patients about near miss incidents or errors, even when patients are not harmed, the Royal College of Surgeons has said. In its guidance on surgeons’ duty of candour, the college says that there is no expectation to tell patients about near misses or incidents that have resulted in no harm. But, it adds, “Surgeons may decide to do so if they think that the patient may want to know this information and that the lack of disclosure may undermine the relationship of trust between surgeon and patient.”

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**FAST FACT – ABNORMAL LIVER FUNCTION TESTS**

The finding of incidentally abnormal liver function tests (LFTs) is common; studies have found that 5%-8% of the general population of Europe have raised liver enzymes. The most common causes of incidentally found abnormal liver enzymes in a UK primary care study of 1118 patients were:

- Non-alcoholic fatty liver disease (NAFLD) (26%)
- Alcohol related liver disease (ARLD) (25%)
- Unexplained (45%), many of these patients had metabolic risk factors and were likely to have NAFLD.

Other causes such as hepatitis B and C, haemochromatosis, and autoimmune liver diseases accounted for less than 1% each.

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You can gain CPD points from your reading by recording what you have read in your appraisal folder. You should try to link your reading back to a learning need and also consider how you plan to improve your practice as a result of your learning.

http://learning.bmj.com
WHAT YOU NEED TO KNOW

- Pityriasis rosea usually starts with a herald patch followed by smaller lesions along the lines of cleavage within three weeks (Christmas tree pattern)
- Reassure the patient that the rash is self-limiting, typically resolves within one to three months, will not scar, is not contagious, and usually needs symptomatic treatment only
- Skin biopsy is unnecessary in typical pityriasis rosea
- Refer to a dermatologist if pruritus is severe, rash persists beyond three months, or the diagnosis is uncertain

Pityriasis rosea is an acute self limiting exanthem that may cause patients great anxiety, but is self-limiting and resolves within one to three months. It is a distinctive erythematous oval scaly eruption of the trunk and limbs, with minimal constitutional symptoms.

What causes pityriasis rosea?
The cause of pityriasis rosea is uncertain but epidemiological (seasonal variation and clustering in communities) and clinical features suggest an infective agent. Light and electron microscopy findings suggest infection with human herpesviruses 6 and 7 (HHV-6/7).

Box 1 | Medications reported to be implicated in pityriasis rosea-like eruptions

- Antibiotics/antifungals: metronidazole, pristinamycin, terbinafine
- Antidepressants/anxiolytics: nortriptyline, barbiturates, bupropion
- Antiepileptic: lamotrigine
- Antihypertensives: angiotensin converting enzyme inhibitors (captopril), clonidine, hydrochlorothiazide, atenolol
- Antipsychotics: asenapine, clozapine
- Biological agents: adalimumab, rituximab
- Metals: arsenic, bismuth, gold
- Vaccines: hepatitis B, H1N1 influenza, yellow fever, BCG, diphtheria, smallpox, pneumococcus, human papillomavirus
- Others: isotretinoin, non-steroidal anti-inflammatory drugs, omeprazole

Drugs and pityriasis rosea
A pityriasis rosea-like eruption has been attributed to several drugs (box 1), mostly in single case reports. In the drug induced form there is no herald patch, individual lesions tend to be violet-red in colour, pruritus is more severe, and eosinophilia may be present.

If the rash lasts longer than two months, consider if medication may be responsible (box 1). If a drug is suspected but is medically indicated, refer to a dermatologist and, if appropriate, another relevant specialist (such as a neurologist about antiepileptic therapy), to assist with decisions on stopping the drug.

Who gets pityriasis rosea?
Pityriasis rosea mainly affects adolescents and young adults aged 10-35 years. A meta-analysis recorded an incidence of 0.68/100 dermatology patients in specialised settings and prevalence has been estimated at 1.3% people in the community.

How does pityriasis rosea present?
Pityriasis rosea begins in 40-76% of people with a single herald patch—an asymptomatic thin oval scaly plaque often on the trunk (fig 1). Multiple herald patches may also occur. The patch is usually well demarcated, 2-4 cm in diameter, erythematous, salmon coloured, or hyperpigmented. A fine collarette of scale is attached to the periphery of the plaque with its free edge extending internally. Within days to three weeks the second phase begins—the appearance of numerous smaller lesions, which are similar in configuration but occur along the
lines of cleavage of the trunk (Christmas tree pattern; figs 2, 3 and 4). The rash typically lasts five weeks; it resolves within eight weeks in 80% of patients but can last for five months. After recovery, the affected skin may be hyperpigmented or hypopigmented but will not scar.

About 20% of patients present with atypical disease, including unilateral, inverse, lichenoid, vesicular, purpuric, erythema multiforme-like, and urticarial types (fig 4).

Constitutional symptoms are usually absent but a recent case series of 52 patients found prodromal symptoms (fever, headache, arthralgia, cough, vomiting, or lymphadenopathy) in 59.6%. Pruritus varies in intensity and frequency and may affect 50% of patients. Topical treatments of all forms have been reported to exacerbate the pruritus.

**Box 2: Proposed diagnostic criteria for pityriasis rosea**

A patient is diagnosed as having pityriasis rosea if:
- On at least one occasion or clinical encounter, he or she has all the essential clinical features and at least one of the optional clinical features, and
- On all occasions or clinical encounters related to the eruption, he or she has none of the exclusional clinical features.

The essential clinical features are:
- Discrete circular or oval lesions
- Scaling on most lesions, and
- Peripheral collarette scaling with central clearance on at least two lesions

The optional clinical features are:
- Truncal and proximal limb distribution, with less than 10% of lesions distal to mid upper arm and mid thigh
- Orientation of most lesions along skin cleavage lines, and
- A herald patch (not necessarily the largest) appearing at least two days before eruption of other lesions, noted from patient history or from clinical observation

The exclusional clinical features are:
- Multiple small vesicles at the centre of two or more lesions
- Two or more lesions on palmar or plantar skin surfaces, and
- Clinical or serological evidence of secondary syphilis

*This outline was proposed by Chuh and Zawar; reproduced with permission from Chuh.*
Relapse rates of 1.8–3.7% have been reported, but these are probably underestimates.\(^6\) Multiple recurrences are rare.\(^1\)

**How do you diagnose pityriasis rosea?**

Pityriasis rosea can be difficult to diagnose, especially at the onset of symptoms. The differential diagnosis is wide (table). Diagnosis is clinical and no non-invasive tests can confirm diagnosis. A Cochrane review noted that studies have used different inclusion and exclusion criteria, making systematic reviews and meta-analyses difficult.\(^3\) Thus diagnostic criteria have been proposed for typical and atypical pityriasis rosea (box 2).\(^6\) Their reliability and applicability in all ethnic groups is as yet uncertain.

Skin biopsy is not advocated in typical pityriasis rosea; however, histological examination shows non-specific but similar changes in the herald patch and secondary lesions.\(^8\)

**Pityriasis rosea in children and pregnancy**

Reports suggest that 6–10.5% of patients with pityriasis rosea are under 10 years of age. Children may present with papular lesions (33%) and in those with darker skin, hyperpigmentation (48%) may persist.\(^5\) HHV-6 or HHV-7 reactivation may occur with immunosuppression. Owing to the altered immune response in pregnancy, there is a risk of viral reactivation. A series of 61 patients found an increased rate of miscarriage in women who develop pityriasis rosea in the first 15 weeks’ gestation, so closer follow-up of these women is recommended.\(^1\)

**How can you treat it?**

Explain to patients that pityriasis rosea is a self limiting condition that is not contagious and usually resolves within one to three months. Treatment is usually symptomatic, with no good evidence for one specific treatment. A 2007 Cochrane review found insufficient evidence for or against the effectiveness of erythromycin, systemic corticosteroids, systemic antihistamine, and intravenous glycyrrhizin.\(^6\) There is also no evidence of benefit from natural sunlight, ultraviolet A1 phototherapy, topical corticosteroids, topical antihistamines, dapsone, and macrolides.\(^5\)\(^1\)\(^4\)\(^5\)

Data from a small randomised controlled trial suggest that taking high doses of the antiviral agent aciclovir may speed resolution,\(^1\) but these are insufficient to recommend its use. If symptoms are severe, consider testing an area with topical therapy (class 2 or 3 topical corticosteroid or topical menthol); if this is tolerated and improves symptoms, it can be applied more extensively.

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**WHEN SHOULD YOU REFER?**

Pityriasis rosea can be managed in primary care. Refer to a dermatology clinic if the rash persists for more than three months, symptoms are severe, the diagnosis is uncertain, or the patient is distressed or requests referral. Although evidence is contradictory, many dermatologists will consider a monitored trial of ultraviolet B phototherapy for more aggressive eruptions.

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**10-MINUTE CONSULTATION**

Reducing the risk of type 2 diabetes

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This is part of a series of occasional articles on common problems in primary care. The BMJ welcomes contributions from GPs.

**WHAT YOU NEED TO KNOW**

- People with glycated haemoglobin between 42 and 47 mmol/mol or fasting plasma glucose between 5.5 and 6.9 mmol/L are at high risk of diabetes
- Weight loss and lifestyle change can reduce this risk considerably
- Consider metformin or orlistat for prevention of diabetes in people at high risk, if intensive lifestyle intervention is not sufficient or suitable

A 45 year old white man comes to see you to discuss his blood glucose result. This was measured because he had an elevated QDiabetes risk score of 15%,\(^1\) as well as a brother with diabetes. His glycated haemoglobin (HbA\(_1c\)) is 43 mmol/mol (6.1%).

**What you should cover**

This man is at high risk of developing diabetes. Determine the risk of diabetes by using a computer based risk assessment tool such as the QDiabetes risk calculator (http://qdiabetes.org/), Cambridge diabetes risk score, or Leicester practice score.\(^2\) If the risk assessment tool suggests elevated risk (for example, >10% over 10 years), you should offer him a fasting plasma glucose or HbA\(_1c\) test. Fasting plasma glucose of 5.6-6.9 mmol/L or HbA\(_1c\) between 42 mmol/mol and 47 mmol/mol indicates a high risk of diabetes (table).

With the added information from your patient’s HbA\(_1c\) result, you now estimate that his risk of progressing to type 2 diabetes is around 5% per year.\(^2\)\(^1\) Intervention at this stage may reduce his risk.

- Consider whether the patient has any condition that may render HbA\(_1c\) inaccurate. Certain medical conditions may falsely lower HbA\(_1c\) (for example, haemoglobinopathy, blood loss) or raise it (for example, iron deficiency anaemia, renal failure, HIV treatment, hypertriglyceridaemia, hyperlipidinaemia).
- Check his blood pressure, weight, and body mass index.
- Discuss other risk factors for diabetes, such as drugs (for example, thiazide diuretics, β blockers, atypical antipsychotics, and steroids), comorbidities (for instance, cardiovascular disease or mental health problems), shift working, stress, and smoking.
- Review his physical activity levels and dietary habits.
WHAT TO DISCUSS WITH PATIENTS

- Ask what he understands about diabetes and its potential complications
- Advise that the screening test for diabetes shows that he is at high risk of developing the condition
- Consider discussing other risks such as certain cancers, cardiovascular disease, arthritis, and sleep apnoea
- If he is overweight or obese, discuss how the loss of 6-7% of one’s body weight can lead to a 58% reduction in the risk of developing type 2 diabetes over three to five years. Even small increases in physical activity are beneficial. For example, using the stairs at work or getting off the bus one stop earlier
- Advise him to reduce his portion sizes and intake of saturated fat and refined sugar. He could increase his intake of wholegrains and vegetables.
- If he smokes, offer smoking cessation advice.
- Explain that diabetes is a lifelong condition that may lead to complications and reduce his quality and quantity of life and that good control can reduce the risk of retinopathy, nephropathy, and neuropathy
- Explain that long term lifestyle change may be more effective than drugs in preventing or delaying type 2 diabetes

Cite this as: BMJ 2015;351:h4595

Diagnostic criteria for diabetes and impaired glucose regulation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Fasting plasma glucose (mmol/L)</th>
<th>2 hour plasma glucose (mmol/L)</th>
<th>Random plasma glucose (mmol/L)</th>
<th>Glycated haemoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;5.5</td>
<td>&lt;7.8</td>
<td>&lt;7.8</td>
<td>&lt;42 mmol/mol (6.0%)</td>
</tr>
<tr>
<td>Impaired fasting glucose</td>
<td>6.1-6.9</td>
<td>&lt;7.8</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Impaired glucose tolerance</td>
<td>&lt;7.0</td>
<td>7.8-11.0</td>
<td>-</td>
<td>42-47 mmol/mol (6.0-6.4%)</td>
</tr>
<tr>
<td>Diabetes mellitus (if asymptomatic, two tests required)</td>
<td>&gt;7.0</td>
<td>≥11.1</td>
<td>≥11.1</td>
<td>≥48 mmol/mol (≥6.5%)</td>
</tr>
</tbody>
</table>

Even small increases in physical activity are beneficial. For example, using the stairs at work or getting off the bus one stop earlier
Diabetic ketoacidosis in adults

Shivani Misra,1,2 Nick S Oliver1,3

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WHAT YOU SHOULD KNOW

- Diabetic ketoacidosis (DKA) is a common, serious, and preventable complication of type 1 diabetes, with a mortality of 3-5%. It can also occur in patients with other types of diabetes
- It can be the first presentation of diabetes. This accounts for about 6% of cases
- The diagnosis is not always apparent and should be considered in anyone with diabetes who is unwell
- Diagnosis is based on biochemical criteria. However, hyperglycaemia may not always be present and low blood ketone levels (<3 mmol/L) do not always exclude DKA
- Immediate treatment consists of intravenous fluids, insulin, and potassium, with careful monitoring of blood glucose and potassium levels to avoid hypoglycaemia and hypokalaemia
- Knowledge of the type of diabetes at the time of DKA does not affect immediate treatment, and all patients with DKA should be advised to continue with insulin on discharge
- Subsequent management should focus on patient education and support to avoid recurrence
- Patients should be managed by a specialist multidisciplinary team during and after an episode of DKA

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

Patients gave perspectives on DKA through a live TweetChat on 15 July 2015 organised by the Great Britain Diabetes Online Community (#GBDOC, www.gbdoc.co.uk), which undertakes a weekly TweetChat on issues relevant to people with diabetes (box 4). We also used opinions from patient contributors who had experienced DKA. These discussions led to the inclusion of a new section on issues for health professionals to consider.

Table 1 | Guidelines for diagnosis of diabetic ketoacidosis (DKA) in adults

<table>
<thead>
<tr>
<th>Definition of DKA by guideline</th>
<th>Ketones used to define severity?</th>
<th>Resolution of DKA</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Diabetes Association 20097</td>
<td>Glucose &gt;13.9 mmol/L</td>
<td>Blood glucose ≥11 mmol/L and two of three: bicarbonate &gt;15 mmol/L, pH ≥7.3, or anion gap ≤12 mmol/L</td>
</tr>
<tr>
<td>Bicarbonate/pH &lt;18 mmol/L; pH &gt;7.3</td>
<td>Ketones Positive result for urine or serum ketones by nitroprusside reaction</td>
<td>No</td>
</tr>
<tr>
<td>Joint British Diabetes Societies 20133</td>
<td>Glucose &gt;11 mmol/L or known diabetes</td>
<td>Yes, a level &gt;6 mmol/L</td>
</tr>
<tr>
<td>Bicarbonate/pH &lt;15 mmol/L or pH &lt;7.3, or both</td>
<td>3-hydroxybutyrate &gt;0.6 mmol/L, pH &gt;7.3, and bicarbonate &gt;15 mmol/L</td>
<td></td>
</tr>
<tr>
<td>Ketones &gt;7 mmol/L or result of urine dipstick testing ++</td>
<td>Testing ++</td>
<td></td>
</tr>
</tbody>
</table>

Box 1 | Signs and symptoms

- Polyuria
- Polydipsia
- Dehydration
- Weight loss
- Nausea and vomiting
- Weakness and lethargy
- Altered mental state
- Kussmaul respiration (a characteristic deep hyperventilation)
- Acetone on breath (smell of pear drops)

How is DKA diagnosed?

DKA is usually diagnosed in the presence of hyperglycaemia, acidosis, and ketosis. However, hyperglycaemia may not be present (euglycaemic ketoacidosis), and low levels of blood ketones (<3 mmol/L) may not always exclude a diagnosis. Clinical judgment therefore remains crucial.

Guidelines differ on the exact biochemical thresholds for diagnosis (table 1).

Glucose

The Joint British Diabetes Societies9 10 recommend a glucose cut-off of >11 mmol/L. The higher cut-off recommended by the American Diabetes Association (>13.9 mmol/L)7 may fail to identify euglycaemic ketoacidosis.

Ketones

Internationally there is little consensus on how ketones should be assessed, the cut-off used, or whether ketones have a role in monitoring for resolution of DKA11 (table 2).12 11
The evidence in favour of a specific DKA diagnostic threshold using 3-hydroxybutyrate is also difficult to evaluate. The more recent observational studies show a variation in 3-hydroxybutyrate levels that mean using a cut-off of 3 mmol/L risks missing patients with lower levels. Taken together these data mean that a ketone value of less than 3 mmol/L may not always exclude the diagnosis of DKA. Other variables and clinical judgment should be taken into consideration.

What is the main approach to management?

The mainstay of treatment is carefully monitored delivery of intravenous fluids and insulin. Fluids correct hyperglycaemia, dehydration, and electrolyte imbalances such as hypokalaemia. Insulin reduces glucose levels and suppresses ketogenesis. This approach coupled with the treatment of the precipitating cause and appropriate patient education before discharge should in most cases result in good outcomes.

Intravenous fluids

The initial fluid of choice in most guidance is 0.9% saline, despite hypotonic fluid losses, as it restores intravascular volume while preventing a rapid change in extracellular osmolality. Subsequent fluid administration depends on the patient’s haemodynamic status and which guideline is being followed, with the American Diabetes Association recommending 0.45% saline if the sodium level is normal or high and the Joint British Diabetes Societies recommending continued use of 0.9% saline. The randomised trial evidence to guide fluid choice is limited. The risk of hyperchloremic metabolic acidosis from continued use of 0.9% saline has prompted the use of isotonic electrolyte solutions in some studies.

Box 2: What precipitates DKA?

- There may be no obvious precipitant, for example, in ketosis-prone diabetes (an atypical form of type 2 diabetes), in which DKA is the presenting condition but insulin can later be discontinued.
- Infection
- Discontinuation of insulin, whether unintentional or deliberate. A variety of factors may contribute to deliberate insulin omission: fear of weight gain or hypoglycaemia, financial barriers, and psychological factors, such as a needle phobia and stress
- Inadequate insulin
- Cardiovascular disease: for example, stroke or myocardial infarction
- Drug treatments: steroids, thiazides, sodium-glucose cotransporter-2 inhibitors
- Consider the diagnosis in any unwell patient with diabetes

Box 3: Complications

- Thromboembolism (DKA is a prothrombotic state)
- Arrhythmias and cardiac arrest (secondary to hyperkalaemia at presentation)
- Iatrogenic: hypokalaemia, hypoglycaemia, cerebral oedema (rare in adults)

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**SPOT DIAGNOSIS**

**Characteristic sign of a colonic submucosal lesion**

A 58 year old woman presented with lower abdominal pain, constipation for three months, and intermittent small bleeds per rectum. She had no abdominal distension, vomiting, weight loss, or family history of colonic cancer. She had hypertension and type 2 diabetes and was taking regular drugs. Blood test results were unremarkable. Ultrasound imaging of her abdomen was normal. Colonoscopy was performed and a soft yellowish polyp in the ascending colon (fig A) was biopsied. Figure B shows the appearance after biopsy.

What is the sign used to describe fig B and what is your diagnosis?

Submitted by Pazhanivel Mohan and Abdoul Hamide

Patient consent obtained.

Cite this as: BMJ 2015;351:h5181

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**CASE REVIEW**

**A case of unusual hand pain in general practice**

A 34 year old white man presented to his general practitioner with a one year history of progressive bilateral hand pain. There was no trauma history or previous arthropathy. He consumed 84 units of alcohol a week, lived alone, and had lost his job as a plasterer owing to reduced hand function. He had three children. On initial examination of the hands and wrists, he had no swelling or bony tenderness and he had normal power, reflexes, and sensation of the upper limbs. Pain was elicited when he unclenched his fists.

Vitamin supplements were prescribed, he was referred to the alcohol team, and a fit note was issued with review planned after a full set of blood tests. Full blood count, renal profile, bone profile, thyroid function, erythrocyte sedimentation rate, and C reactive protein were within normal ranges. Serum alanine aminotransferase was raised (59 U/L; reference range 0-40) and rheumatoid factor was negative.

At follow-up review, bilateral ptosis, frontal balding, and myopathic facies were noted. Bilateral percussion myotonia and distal wasting of the hand muscles were also found on examination. Finger extension was restricted and slowed bilaterally. Cranial nerve examination was otherwise normal, as was cardiorespiratory examination.

1 What do these clinical findings suggest?

2 On the basis of these clinical findings, what further investigations are indicated in general practice?

3 Should this patient be referred for specialist input?

4 How should the patient be counselled after confirmation of the diagnosis?

Submitted by Faraz Mughal, Ahmed Rashid, and Rajnish C Mishra

Patient consent obtained.

Cite this as: BMJ 2015;351:h5005

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We welcome contributions that would help doctors with postgraduate examinations. We also welcome submissions relevant to primary care. See thebmj.com/endgames
Potassium supplementation

Hypokalaemia is a major and potentially fatal complication of DKA. All guidelines recommend potassium replacement after the first litre of fluid (or in the first litre if hypokalaemia is present). The Joint British Diabetes Societies recommend potassium monitoring at one hour and two hours after admission and every two hours thereafter.

Bicarbonate

Bicarbonate is not routinely recommended. It should be considered only under specialist supervision in patients with severe acidosis (pH <7) in whom the effects of acidaemia on myocardial contractility and cardiac output may be life threatening. Even in these patients the benefits are unclear. Harmful effects may include exacerbation of existing hypokalaemia. They may also include a late metabolic alkalosis, with a shift of the effects of acidaemia on myocardial contractility and cardiac output may be life threatening.

What dose of insulin?

Both the Joint British Diabetes Societies and American Diabetes Association recommend a weight based rate of delivery of 0.1 units/kg/h. An initial bolus dose of insulin is not advised, based on a randomised controlled trial that found no benefit.

A steady delivery of low dose insulin adequately suppresses lipolysis (and thus ketogenesis). With concomitant intravenous fluids, glucose levels may normalise rapidly. However, ketoadamosis corrects more slowly: on average it takes six hours of treatment before glucose decreases to less than 14 mmol/L, compared with 12 hours before ketoadamosis is corrected. Adequate insulin should therefore continue beyond the resolution of hyperglycaemia to ensure the eradication of ketones. This has led to the shift away from a “sliding scale” that titrates insulin against glucose levels, to fixed rate intravenous insulin infusion.

How should patients be monitored?

Some of the major complications of DKA are related to its treatment (box 3). Blood glucose and potassium levels must be closely monitored and patients must have regular review, as too much insulin results in hypoglycaemia and hypokalaemia whereas not enough may fail to adequately suppress ketogenesis.

The Joint British Diabetes Societies recommend high levels of care and central venous access for those with severe DKA: people with severe metabolic derangement (pH <7.1, bicarbonate <5 mmol/L, blood ketones >6 mmol/L or hypokalaemia on admission (K+ <3.5 mmol/L)), a reduced Glasgow coma score, or haemodynamic instability. However, the guidelines are not prescriptive and people at extremes of age or with comorbidities may also require higher levels of care.

There is no substitute for careful monitoring and responding to the patient as treatment progresses. Mortality from DKA has improved considerably, but still persists at between 3% and 5%. Death is most often associated with the precipitating illness (for example, cardiovascular disease) and increasing age.

When should patients transition from intravenous to subcutaneous insulin?

There is no consensus on what marks the biochemical endpoint of DKA (table 1), so transition to subcutaneous insulin is advised when patients are eating and drinking. If patients are not eating and drinking but ketones are suppressed, a variable rate intravenous insulin infusion can be considered until oral intake is resumed. Crucially in such cases, there should be overlap between intravenous and subcutaneous insulin in order to prevent any period of insulin deficiency that risks recurrent ketogenesis. UK guidance recommends continuation of intravenous insulin for at least 30–60 minutes after the initiation of subcutaneous long acting insulin. In people with established type 1 diabetes, there is some evidence that continuing subcutaneous long acting insulin throughout the admission prevents rebound hyperglycaemia, and local practice may vary. Transition to subcutaneous insulin is best supported by members of the specialist diabetes team in line with national guidance.

How can DKA be prevented?

Patients with established type 1 diabetes should be given as much information as possible about risk factors for DKA and how to monitor their own glucose and ketone levels. Discussions from the accompanying TweetChat (box 4) show the need for better education. Some participants were unaware of the importance of ketone testing or the difference between ketosis and ketoacidosis.

Structured educational programmes provide advice on how to avoid omitting insulin; sick day rules, including increasing insulin doses if unwell; and when to test ketones. They can reduce rates of DKA. However, programmes are not always offered, and uptake can be poor.

Patients should be advised to measure their ketone levels if they are unwell as this may identify incipient ketosis, which can be dealt with by increasing insulin doses. They should be encouraged to seek medical attention if levels are increased.

Testing ketones in capillary blood has not been shown to be better for preventing DKA than urine testing. People with recurrent DKA may have underlying precipitants, and psychological support may be helpful.

Drugs such as sodium-glucose cotransporter-2 inhibitors should be used with caution in people at high risk of DKA, although these associations are still being elucidated.

Cite this as: BMJ 2015;351:h5660

Box 4 | Patients’ perspectives on DKA, from TweetChat

“Considering it’s such a major diabetic issue, it’s shocking and scary how many people don’t know what DKA actually is…”

“When I was diagnosed, I was in what is termed a semi-coma for three days. It was totally missed by my GP that I had T1…”

“It’s horrible and still fills me with panic that I was so very close to death, GP misdiagnosed three times!”

“When I was diagnosed, impact on mental health was never considered”

“Yep was drifting in and out of a coma. Now I panic when I get high sugars, would rather hypo any day”

Issues for healthcare professionals to consider, from structured and unstructured discussions between patient contributors:

• DKA is a frightening experience. Consider the need to address patients’ fears and concerns during recovery
• Consider your behaviour and language when talking to someone with a new diagnosis of type 1 diabetes. Ensuring the patient gets positive messages about type 1 diabetes is critical, as is ensuring access to support
• Provide advice about how to access further educational resources, including the importance of structured educational programmes
• Ensure that the episode of DKA is not viewed as a failure of self care, and that a personalised care plan is in place to prevent further episodes.
Massive diaphragmatic hernia causing shortness of breath and abdominal pain

A 42 year old woman presented with chronic shortness of breath and left upper quadrant pain. On chest auscultation bowel sounds were audible up to the axillae. Chest radiography showed bowel extending into the thorax. Computed tomography showed almost complete lung collapse due to a diaphragmatic hernia containing small and large bowel. Diaphragmatic herniation is an uncommon cause of shortness of breath but should be considered particularly in patients who also have non-specific abdominal symptoms. A tentative diagnosis could have been made on clinical examination alone. This case highlights the importance of thorough clinical examination and discourages over-reliance on imaging in today’s culture.

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Patient consent obtained.

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It eats your brain and grows out of your eyes

*Cordyceps sinensis* is an unpleasant fungus, and quite a rare one, visible above ground on certain Himalayan plateaux where it grows out of the heads of subterranean beetles. Cochran reviewers have just trawled the mainly Chinese literature for evidence that it might have beneficial effects on kidney transplant recipients (*Cochrane Library* 2015, doi:10.1002/14651858.CD009698.pub2). If it does, it has yet to be demonstrated.

Critic and sage

“Why Most Published Research Findings Are False” was the 2005 paper that propelled John Ioannidis to fame, and he remains one of the most cogent critics of the published literature. This month he used his opening address at the annual Cochrane Colloquium in Vienna to argue that most systematic reviews are pointless. But his positive contributions are equally remarkable, and anyone interested in how medical knowledge is generated needs to read his latest paper (*PLoS Biol* 2015;13:e1002264; doi:10.1371/journal.pbio.1002264). It is about meta-research, which means how to do, report, verify, correct, and reward science.

The heart of prognosis

The hormones that the heart releases under stress are powerful prognostic markers, and a study by oncologists in Vienna finds that this applies not just to heart disease but also to cancer (*Heart* 2015, doi:10.1136/heartjnl-2015-307848). Markers like natriuretic peptides and copeptin were raised in an unselected group of cancer patients about to undergo cardiotoxic chemotherapy; concentrations rose even more with disease progression and were strongly related to all cause mortality.

Forgetting that you forget

Ronald Reagan is said to have joked that it was nice to have Alzheimer's disease because you meet so many interesting new people. Although most people with memory problems worry more than that, a study in *Age and Ageing* (2015, doi:10.1093/ageing/afv136) found that as memory deteriorates people no longer worry about memory loss because they are unaware of it.

De-adoption

“It is difficult to get a man to understand something, when his salary depends upon his not understanding it.” Minerva was reminded of Upton Sinclair’s comment when she saw a paper entitled “Towards understanding the de-adoption of low-value clinical practices: a scoping review” (*BMC Med* 2015;13:255, doi:10.1186/s12916-015-0488-z). Financial interest may not be the only reason why the Choosing Wisely campaigns are showing so little success in North America, but it is surely one of them. This review of the full literature is a valuable resource for everyone interested in making healthcare effective and sustainable.

Socially patterned asthma

Where once there was talk of the diseases of poverty, now there is talk of socially patterned phenotypes. Kids in the Avon Longitudinal Study of Parents and Children were more likely to have stand alone asthma if they came from poorer families, especially if their mothers smoked, than children from higher social class families, who were more likely to have asthma and atopy (*Am J Epidemiol* 2015, doi:10.1093/aje/kwv045). The authors conclude that current inequalities among children who have asthma but not atopy can be prevented by eliminating exposure to tobacco smoke.

Azathioprine pancreatitis

A study of patients taking azathioprine for inflammatory bowel disease in 37 centres around Germany (*J Crohns Colitis* 2015, doi:10.1093/ecto-jcc/jfv188) found a 7.3% incidence of pancreatitis—about twice that reported previously. However, most cases were mild and half did not require hospital admission. All patients made a full recovery.

There are no snakes in Iceland

Niels Horrebow’s famously short 1752 chapter about the absent snakes of Iceland is actually a bit longer than that, as is an article called “Child health in Iceland before and after the economic collapse in 2008” (*Arch Dis Child* 2015; doi:10.1136/archdischild-2014-307196). But the message is also one of absence: the economic collapse seems to have had no more effect on child health in Iceland than it did on the incidence of snakebite.