Should we use bath emollients for atopic eczema?

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Regular topical application of an emollient cream or ointment is key in the management of patients with atopic eczema and is thought to help the skin maintain a defensive barrier effect, which is defective in atopic eczema.1 Support for such treatment comes from one (non-blinded) randomised controlled trial, which found that regular application of emollients direct to the skin reduced the amount of topical corticosteroid cream needed for atopic eczema in infants.2 Long clinical experience also suggests that directly applied emollients are safe and effective in atopic eczema.

People with atopic eczema are commonly also advised to use an emollient substitute for soap (such as aqueous cream or emulsifying ointment), as soap can irritate the skin (as can bubble bath preparations); patients are also often prescribed a bath emollient to add to their bath water.3 Bath emollients typically consist of liquid paraffin plus another emollient (usually wool fat or isopropyl myristate); a few also contain an antimicrobial drug.

Some prescribers recommend bath emollients to avoid use of bubble bath preparations. Some believe that using a bath emollient is an easy way to apply an emollient to a large area of skin, particularly for children, who may not cooperate with having topical emollients applied frequently. Also, some treatment guidelines argue that complete emollient therapy (a combination of creams, ointments, bath emollients, and soap substitutes) will provide maximal effect.4 Of note, the National Institute for Health and Clinical Excellence’s guideline on atopic eczema in children (applicable to England, Wales, and Northern Ireland) suggests the additional use of bath emollients for some children “to ensure that adequate amounts of emollient are absorbed into their skin.”5 These influences have prompted very common use of bath emollients.

What is the evidence of the uncertainty?

We searched PubMed, the Cochrane Library, Clinical Evidence, and the Current Clinical Trials database to identify published and ongoing randomised controlled trials and systematic reviews that have assessed the efficacy of bath emollients in patients with atopic eczema. This search followed previous wide consultation among specialists, as well as drug companies, to identify relevant published evidence while preparing an article on bath emollients for the Drug and Therapeutics Bulletin.6

No published randomised controlled trials have specifically assessed the clinical efficacy of bath emollients in atopic eczema.7 8 Also, we are not aware of any longstanding clinical experience of benefit from bath emollients to match that for directly applied emollients. The quantities of emollient deposited on the skin during bathing are likely to be far lower than with directly applied emollients. These points highlight the weakness of the case for using bath emollients.

Additionally, we found no published evidence that “complete emollient therapy” has a “maximal effect.” Moreover, the unproved concept of “complete emollient therapy” has fostered assumptions that each of the individual components (including bath emollients) contributes to a worthwhile benefit, despite the absence of confirmatory data.

On current evidence, bath emollients could be offering little or no benefit. If so, people who use them in place of directly applied emollients are unknowingly receiving substandard emollient therapy.

Is ongoing research likely to provide relevant evidence?

Trials on the clinical efficacy of bath emollients in atopic eczema are feasible but no such studies seem to be in progress (www.controlled-trials.com/mrct/). The box outlines possible research that could be conducted. Such research might facilitate development of products that produce bubbles for young children to enjoy in the bath but do not irritate the skin and which can be bought over the counter; this would obviate the need to use bath emollients to fill this gap.

What should we do in the light of uncertainty?

Robust clinical trial data could reassure patients and prescribers that bath emollients are worth using for atopic eczema or, alternatively, show that spending by the NHS in the UK on these products (about £15.5m (£17m; $25.4m) in England alone in 2008)9 could be put to better use.
In the absence of confirmatory data, no basis exists for asserting that patients who successfully apply emollients directly to the skin but do not also use bath emollients are using substandard treatment.

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LESSON OF THE WEEK

Osteoarticular infection of the symphysis pubis and sacroiliac joints in active young sportsmen

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Athletic patients may be susceptible to staphylococcal infection of their cartilaginous non-synovial joints

We describe two cases of young sportsmen who presented to our emergency department nine months apart with pyrexia, pain exacerbated by movement, and blood cultures positive for Staphylococcus aureus, indicative of infection of the cartilaginous joints. This unusual cause of febrile illness in athletes should not be overlooked, particularly as localising signs may not be evident at presentation. An entry site for infection is not always apparent.

Case report 1

A previously fit and well 24 year old fitness trainer presented with a three day history of increasing leg weakness and pain in his groin and gluteal regions, accompanied by fever and rigors. He had had a dental extraction one month before, in his groin and gluteal regions, accompanied by fever and rigors. Two days later he was bedridden and remained feverish. Repeat examination showed that he had no true leg weakness but had painful restriction of movement secondary to point tenderness over the symphysis pubis. Blood cultures were positive for Staphylococcus aureus and he was given intravenous flucloxacillin and rifampicin. An echocardiogram and abdominal ultrasound scan showed no source of infection, but an isotope bone scan showed increased uptake over both sides of the symphysis pubis. Magnetic resonance imaging of the pelvis was consistent with an inflammatory abnormality centred on the symphysis pubis (fig 1).

The patient improved on antibiotics and his C reactive protein concentration normalised after treatment. A diagnosis of osteomyelitis of the symphysis pubis was made in view of the scan findings, fever, and positive blood cultures. He recovered well after a six week course of antibiotics and returned to normal training activities.

Fig 1 | MRI STIR sequence showing symphysis pubis and superior pubic rami. Increased signal around the symphysis pubis and superior pubic rami indicates marrow oedema, and subperiosteal fluid is visible. This pattern suggests aggressive inflammation consistent with infection.
Case report 2
A 20 year old rugby player presented to the emergency department with lower back pain. Blood tests including urea and electrolytes, full blood count, and liver function tests were normal, and his C reactive protein was 5 mg/l. He was discharged with analgesia. Two days later he presented again with worsening lumbar pain radiating to the groin, accompanied by fever, sweating, and dysuria.

On examination his abdomen was soft but left renal angle tenderness was evident. Results on urine dipstick testing were protein ++, nitrates + and bilirubin +++. His liver function tests were abnormal (bilirubin 45 µmol/l, alkaline phosphatase 168 U/l, alanine aminotransferase 91 U/l, and γ-glutamyl transpeptidase 168 U/l), but renal function was normal. His C reactive protein was raised at 248 mg/l, and he was treated for likely pyelonephritis.

Twenty four hours later on clinical review he could raise his left leg to only 70°, but no neurological deficit was found. Blood cultures were positive for *S aureus* and urgent magnetic resonance imaging of the spine was arranged to exclude osteomyelitis or discitis. He was given flucloxacillin and rifampicin intravenously, and his symptoms improved over the next four days. By then the pain had localised to the left sacroiliac joint, and a bone scan showed increased isotope uptake over this region (fig 2), consistent with infection at this site. He was discharged after 1/4 days of intravenous antibiotics; one month later his C reactive protein was 3 mg/l and liver function was normal. He took oral flucloxacillin and rifampicin for four more weeks, and six months elapsed before he could resume normal training.

Both patients were followed up for about six months after discharge and remained well once the initial illness had settled. They made a good recovery and were able to resume their normal training.

Discussion
These cases represent the variable presentation of patients with infection in their cartilaginous joints, and the importance of a high index of clinical suspicion and repeated review of symptoms to reach the correct diagnosis.

Osteomyelitis pubis is a rare condition with a variety of presentations. It should be distinguished from the inflammatory condition osteitis pubis, which is more common and can occur in athletes whose training involves rapid changes in speed or direction. The common sheering forces result in inflammation, sclerosis, and bony changes in the region and can be picked up on magnetic resonance imaging. Osteomyelitis pubis is far less common and is caused by infection.³ The most common causative organism is *S aureus*, as in our cases; others are *Pseudomonas aeruginosa* and *Escherichia coli*.¹ The diagnosis is ideally confirmed by biopsy and culture of the site, but positive blood cultures together with painful hip movements, fever, and localised pain are strongly suggestive. The diagnosis of osteomyelitis pubis is also supported by the response to appropriate intravenous antibiotics.

Osteitis pubis usually presents with pain in the groin and should be treated with rest and non-steroidal anti-inflammatory drugs.³ Fever or refractory symptoms should alert the doctor to the possibility of osteomyelitis pubis, which is treated with prolonged intravenous and oral antibiotics. A total course of six weeks of treatment is suggested,⁴ but an evidence base for optimum antimicrobial treatment is lacking.⁵,⁶ Previous osteitis pubis may predispose to subsequent bacterial infection at this site.⁷

In a review of 18 cases of osteomyelitis pubis in athletes, the diagnosis was delayed in most cases (mean 13 days).¹ The prognosis is generally good despite the delay,¹ but potential complications include chronic pain at the site and pelvic instability, both of which would be devastating for young athletes.³

Acute bacterial infection of the sacroiliac joints in adults is less commonly documented.⁷ It presents with a triad of fever, antalgic gait, and buttock pain. Delay in diagnosis is common, and tests are non-specific. Cases are most commonly caused by *S aureus; P aeruginosa* infection is more likely in intravenous drug users.⁸ Both our patients denied using performance enhancing drugs. Complications include abscess formation, septicaemia, and haematogenous spread to distal sites.⁹ In case series, treatment was as for osteomyelitis pubis.⁵

In case 2, abnormal liver function tests on arrival in hospital normalised with treatment. In a similar case reported in 2006, a man was diagnosed with staphylococcal osteomyelitis of the left sacroiliac joint.¹⁰ His localising signs were preceded by jaundice, which resolved completely with antimicrobial treatment.

Athletes who train in activities involving sudden changes in speed or direction are predisposed to infection in their cartilaginous, non-synovial joints, which suffer chronic, repetitive strain. In our patients it took 10 days (case 1) and two days (case 2) for the localising features of osteomyelitis to develop. We recommend frequent review of symptoms and signs in such cases, so that this important diagnosis is not overlooked.

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Patient consent obtained.
Corrections and clarifications

Exercise on prescription for women aged 40-74 recruited through primary care: two year randomised controlled trial

About a year after publication of this paper by Beverley A Lawton and colleagues (BMJ 2008;337:a2509, print publication 10 Jan 2009, pp 88-91), we have been alerted to an error in the title of the table. The title states that the values in the table are means and standard deviations, whereas in fact they are means and standard errors.

Income inequality, mortality, and self rated health: meta-analysis of multilevel studies

One of the figures in this research article by Naoki Kondo and colleagues contained an error (BMJ 2009;339:b4471, print publication 21 Nov, pp 1178-81). At the bottom of figure 2, 9.6% represents the percentage of total adult mortality (not of “total OECD population”).

Continuation of metformin after introduction of insulin in type 2 diabetes

Some small discrepancies occurred between the data reported in this editorial by Adriaan Kooy (BMJ 2009;339:b4227, print publication 14 Nov, pp 1093-4) and those reported in the linked research paper by Søren S Lund and colleagues in the same issue (BMJ 2009;339:b4324, pp 1121-5). These discrepancies arose from an error in the editorial department: when invited to write the editorial, Kooy was given the accepted (but unedited) manuscript of the research paper, but we failed to pass on to him the information about the subsequent changes in data introduced by the authors during the editing process. Kooy has become aware of these changes only since publication. All the discrepancies occurred in the second paragraph of the editorial and are explained below. None of the discrepancies affect the validity of any conclusion or point made by Kooy.

The editorial reported a fall in glycated haemoglobin level in the metformin plus insulin group from 8.15% to 6.71% (but this level should have been reported as 6.72%). The difference in the reduction of glycated haemoglobin concentration between the metformin plus insulin group and the repaglinide plus insulin group was therefore P=0.177 (not P=0.125).

The editorial also reported that in the patients with a negative glutamic acid decarboxylase 65 antibody status the difference in glycated haemoglobin level between the two treatment groups was −0.29% (95% confidence interval −0.56% to −0.02%, P=0.034); in fact, the values were −0.27% (−0.55% to 0.00%, P=0.052). Hence, the small difference in favour of metformin plus insulin was of borderline significance. As the interaction of treatment and antibody status was statistically significant (P=0.037), Kooy’s original statement that differences might exist in glycemic response in the metformin plus insulin group compared with the repaglinide plus insulin group on the basis of autoimmunity is still valid.

Finally, the difference in weight gain between the metformin plus insulin group and the repaglinide plus insulin group was reported in the editorial as −2.51 kg (95% confidence interval −4.10 kg to −0.95 kg; P=0.002), whereas the correct lower confidence limit is −4.07.

Selection bias explains seasonal vaccine’s protection

A percentage was wrong in the second paragraph of this letter by Naveed Z Janjua and colleagues (BMJ 2009;339:b972, print publication 28 Nov, pp 1214-5). In the penultimate sentence (starting “Therefore, with a higher rate of chronic conditions...”) the final percentage should read “about 15%” [not “9%”]. Also, references 5 and 6 were published the wrong way round (they should be switched).

Vertigo

In the section headed “Vertigo without red flags: confirmatory tests” in this Practice article last year by Kevin Barraclough and AdolfoBronstein (BMJ 2009;339:b3493, print publication 26 Sep 2009, pp 749-52) we wrongly said that videoclips were available on bmj.com showing two repositioning manoeuvres (the Epley manoeuvre and the Semont manoeuvre) for treatment of benign positional vertigo. These are not on bmj.com, but the authors have told us that they are available from http://www1.imperial.ac.uk/medicine/research/researchthemes/neuroscience/movement_balance/mabdandv/.

Cools to Newcastle?

In preparing this letter for print from the original online rapid response, we wrongly gave Sheila Sherlock as an author (BMJ 2010;340:c287, print publication 23 Jan 2010, p 167). We apologise for this. The sole author of this letter is Humphrey J F Hodgson, who holds the Sheila Sherlock Chair of Medicine UCL and is senior censor and vice president, education and training, Royal College of Physicians of London.