much more and found that through simple action planning I was able to start making the changes that improved my life and my health. I learnt pain management techniques and was able to improve my pain relief. I increased my exercise levels, improved my eating habits, and drank more water. I was able to maintain these improvements after completing the course and now, if they do slip, I notice straight away, feeling more fatigue and more pain. Living this healthy lifestyle also helps me to feel in control.

The judge's score

Although I do get frustrated quite often, and I am occasionally fearful for the future, I no longer have any anger. I am a very optimistic person and keeping up to date with current research helps reassure me. I hope that the self management techniques I have learnt through the volunteer programme and through over a decade of living with lupus has meant that I have a better relationship with my clinicians (I think it has) and feeling better and improved self esteem have improved my prognosis.

And, strangely, there have been benefits in having lupus. Without it, I would not have learnt so much about myself and about life in general; I would not have met some of the people I have met and become friends

Websites

- Lupus UK (www.lupusuk.org.uk)—the only British national charity that supports people with systemic lupus and discoid lupus and helps people approaching diagnosis
- Lupus UK's site for healthcare professionals (www.medical.lupusuk.org.uk)—provides information on the diagnosis of lupus and the treatment and support of patients with lupus and related connective tissue diseases
- The LUPUS Journal, published by Hodder Arnold in the United States (www.ingentaconnect.com/content/arn/lu)
- Lupus Patients Understanding & Support (LUPUS): for free information and free psychological support (www.lupus-support.org.uk; www.lupus-forums.org.uk/LuPUSMB)

with; and I would not have become involved with the Expert Patient Programme. I am genuinely grateful for all these things. With support from friends, family, and clinicians, dancing with the wolf is not all doom and gloom.

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Lesson of the week

New football boots and toxic shock syndrome

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Consider toxic shock syndrome in a child with rash, fever, and hypotension

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Toxic shock syndrome is unusual in childhood. Although it is easily recognised by those who have seen it before—and is an important diagnosis to make—few clinicians will have encountered a case. We present two similar cases. Both patients developed friction blisters over the area of calcaneal insertion of their Achilles tendons—the result of wearing new football boots. The blisters contained *Staphylococcus aureus*, which in one case was found to express the toxic shock syndrome gene TSS1. Both had a secondary rash during recovery.

Case reports

Case 1

A 13 year old girl played a competitive game of football in new boots and developed friction blisters over both heels. The next day she developed fever, lethargy, and vomiting but remained alert and oriented. Within a few hours, she had developed an erythematous, macular, coalescent rash over the trunk, back, and all limbs (figure). Twenty four hours later she developed diarrhoea, which lasted a few hours, and abdominal pain.

She was admitted to her local hospital, where the rash was recorded as having spread to the hands and feet, with oedema of the fingers. The buccal and labial

Case 1: Erythematous rash on patient's arm and trunk

mucosae were erythematous and the conjunctivae injected. On arrival at hospital her blood pressure was 106/46 mm Hg. She had no anaemia, lymphadenopathy, or hepatosplenomegaly and no abnormalities in the respiratory or central nervous systems. Her last menstrual period had been 10 days previously and she never used vaginal tampons.

She was treated empirically with intravenous ceftriaxone 3.2 g daily (she weighed 64 kg). A blood culture taken before she started taking antibiotics proved sterile. Over the next 48 hours she deteriorated and

Table 1 Early clinical findings in both cases

Findings	Case 1	Case 2
Fever	Yes	Yes
Diarrhoea and vomiting	Yes	Yes
Hypotension	Yes	Yes
Rash	Yes	Yes
Myopathy	Yes	No
Renal impairment	Yes	Yes
Mucosal erythema	Yes	Yes
Confusion	No	Yes

showed increasing muscle weakness. Her blood pressure ranged from 92 mm Hg to 103 mm Hg systolic and 48 mm Hg to 61 mm Hg diastolic, with a pulse rate of 100-130 beats/min and temperature fluctuating between 37°C and 39°C. There was laboratory evidence of worsening renal function, lymphopenia, derangement of liver enzymes, and raised plasma concentration of creatine kinase.

On day 4 of her illness the patient was transferred to the regional paediatric renal unit because of worsening renal function. She maintained a good urine output and hydration, but her blood pressure in the first 12 hours after transfer ranged from 93 mm Hg to 100 mm Hg systolic and 30 mm Hg to 47 mm Hg diastolic. Urine analysis (with Ames Multistix) showed blood 3+, protein 2+ and leucocytes 2+, and urinary nitrite was not detectable. Further examination showed a blister (containing yellow pus and measuring 2 cm in diameter) over each of her Achilles tendons. There was no fasciitis and little surrounding inflammation. Toxic shock syndrome (TSS) was diagnosed, and she was treated with clindamycin at an initial dose of 300 mg every six hours and cefotaxime 3 g every six hours.

Over the next 48 hours she showed profound muscle weakness. Electrocardiography and echocardiography were normal. By day 5 her temperature had settled and her blood pressure had improved to give diastolic readings consistently above 55 mm Hg and systolic readings consistently above 105 mm Hg. By day 7 she was able to walk about, the rash had largely cleared, and her mucosae were normal. *Staphylococcus aureus* was cultured from the pus in the blisters, and the cefotaxime was discontinued. The *S aureus* was shown by multiplex polymerase chain reaction to possess the TSST gene that encodes for toxic shock syndrome toxin 1. The patient was discharged on day

10 but was readmitted on day 12 after developing a transient recurrence of rash. Clindamycin was discontinued. When reviewed 19 days after onset, she had extensive peeling of her skin, particularly hands and feet. She has since made a full recovery.

Case 2

A previously healthy 11 year old boy played football in a new pair of boots, causing a blister on his right heel. Over the next two days he developed fever, vomiting, and diarrhoea. Two hours before admission to hospital (on the second day of his illness) he became confused and developed a generalised macular rash. On initial examination he was 5% dehydrated and his blood pressure was 99/63 mm Hg. He was drowsy (Glasgow coma score 12). He had a centripetal erythematous macular rash all over his body, with blanching discrete spots (1-2 mm diameter) on the posterior aspects of his thighs and on his trunk and arms. He had hyperaemic mucosae. His temperature was 40°C. Abdominal examination showed a 3 cm non-tender hepatomegaly. A blister was noted on his right heel. Intravenous cefotaxime was started empirically.

Within nine hours his condition had deteriorated and his blood pressure had fallen to 86/42 mm Hg despite resuscitation using 70 ml/kg of intravenous fluid since admission. His paediatricians recognised the features of TSS and added intravenous flucloxacillin. The blister on the heel was deroofed. Pus from this later grew *S aureus*, sensitive to flucloxacillin. Faeces and throat virus cultures were negative as was measles IgM antibody titre. Throat culture and blood cultures were negative. A dopamine infusion was started at 5 µg/kg per minute, and he was transferred to the regional paediatric intensive care unit. His blood pressure stabilised and he was transferred back to the local hospital 48 hours later.

On day 7 he developed a maculopapular rash over his torso and arms associated with an increase in temperature to 38.5°C. Chlorphenamine was given. He was discharged home on day 9 to continue oral co-amoxiclav for a further five days; the rash was still florid at this time. After discharge the skin of his hands and feet peeled.

On review three months later he had made a full recovery. The allergen specific IgE antigen test (IgE RAST), skin prick tests, and a formal flucloxacillin challenge were all negative, confirming that he did not react to flucloxacillin.

Table 2 Laboratory findings in both cases

Laboratory findings	Case 1		Case 2	
	On admission	12 hours after admission	On admission	9 hours after admission
Haemoglobin (g/l)	117	121	143	116
White cell count (10 ⁹ /l)	10.6	16.0	13.4	17.5
Neutrophil (10 ⁹ /l)	9600	13 100	12 900	16 500
Lymphocytes (10 ⁹ /l)	300	1700	100	200
Platelets (10 ⁹ /l)	180	169	180	149
Serum sodium (mmol/l)	134	137	126	133
Serum urea (mmol/l)	19.6	16.1	7.9	6.7
Serum creatinine (µmol/l)	188	136	88	71
Serum bilirubin (µmol/l)	56	62	NA	21
Creatine kinase (IU/l)	NA	5536	NA	270
Aspartate aminotransferase (IU/l)	NA	145	NA	37
C reactive protein (mg/l)	353	494	4	104

NA=not available.

Discussion

Toxic shock syndrome in adults has become less common since the association with tampon use was recognised in the 1980s. In children, for whom this association does not apply, TSS is rare and mostly a complication of skin burns affecting less than 10% of the body surface in the very young.¹ Children seem to have a better prognosis than adults, but a paediatric mortality of up to 5% for TSS induced by *S aureus* is currently quoted.² In a series of 13 referrals to a paediatric burns unit, however, none required ventilatory support or died.¹ A perinatal, incomplete form of TSS has been described in which babies develop a systemic exanthema and thrombocytopenia but not multiorgan failure and hypotension. This incomplete form of TSS seems to be attributable to carriage of methicillin resistant *S aureus*.³

These cases illustrate the typical speed of onset and range of symptoms and findings (tables 1 and 2). Rash, fever, and hypotension are central to the diagnosis. Hypotension is not as abrupt in onset as is usually seen in meningococcal sepsis. Lymphopenia is common. In one case, the doctors did not initially make the diagnosis and missed the clinical importance of the unimpressive blister.

Primary rash of TSS is characteristic.⁴ A secondary rash occurring during the convalescence stage has been described previously but is under-reported⁵ and may be mistaken for antibiotic allergy. We confirmed in the second case that the mechanism was not due to a

reaction to penicillinase resistant semisynthetic penicillin. We suggest that the appearance of a rash during recovery is unlikely to be due to an allergy to this type of penicillin. Antibiotic therapy should, therefore, not be discontinued or the regimen modified unnecessarily, and the patient should not be told they are allergic to penicillin.

TSS may follow relatively trivial skin trauma, and in a patient with signs of the syndrome a thorough search for the source is essential. Arnold and colleagues suggested that the foot is an unlikely site for infection,⁶ but we question that assumption.

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Ter die sumendus

In 1964 I was in one of the last years of UK medical students trained at the old Charing Cross Medical School in the art of making mixtures and the use of the pestle and mortar. Our pharmacology classes took place in what had once been a fashionable restaurant across the road from the hospital, by then converted into laboratories. The ornate pressed ceilings and chandeliers had been left, which gave the laboratories a rather bizarre atmosphere—a mixture of Georgian elegance and Neo-Gothic pillars fused with Bunsen burners and a modernistic skyline of distillation flasks.

I have the printed pharmacology lecture notes in front of me. I see that I was assigned to make up aspirin mixture for children (Mist. Acid. Acetylsal pro infans B.P.C.). The formula was Acid. Acetylsal 2 grains, Pulv. Tragac Co 1 grain, Syr. Rub Idaei 15 minims, Liq. Amaranth $\frac{1}{2}$ minim, Aq. ad 1 drachm. There is a note to say that compound powder of tragacanth is a thickening agent consisting of a mixture of gum of tragacanth 15%, gum acacia 20%, starch 20%, and sucrose 45%. I had to use the pestle and mortar to “tritrate” the aspirin and tragacanth into a smooth cream while adding the syrup of raspberry (rub. idaei) and solution of amaranth. I should think the restaurant had seen a similar process when the chefs were making up Hollandaise sauces.

I also remember my father, who was a village GP on the west coast of England before and after the second world war, compounding mixtures with his pestle and mortar and then leaving the mixtures on a shelf outside the surgery for patients to collect. When it got busy my mother was roped in from the kitchen to help

make up the Mist Pot. Iod. among the jeroboams at the back. When she was finished she would return to the kitchen to continue mixing the sauce for supper. Those were the early days of multitasking.

I have also in front of me the book we used—*Aids to Pharmaceutical Latin*, first published in 1921, by Bailliere, Tindall, and Cox of 7 and 8 Henrietta Street, Covent Garden. The pages are redolent with memories of fourth form Latin. You could order fomentations ter die sumendus (tds or three times a day), omni nocte (o.n or every night) or pro re nata (prn or as necessary). I realise that we will soon have to give up these few remaining Latin abbreviations in the interests of clarity and uniformity, but I will miss that touch of mystery and apothecary artistry of the pen as it writes, “signa: sumat aeger hora decubitus alterno die” (label: let the patient take at bedtime on alternate days).

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