PRACTICE POINTER

Necrotising fasciitis

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Necrotising fasciitis is one of a group of highly lethal infections that cause rapidly spreading necrosis of fascia and subcutaneous tissues, sometimes involving muscles and skin. They were previously known by such names as hospital gangrene, gas gangrene, and Fournier's gangrene and are now referred to by the generic term "necrotising soft tissue infections." We review the clinical features and highlight the potential pitfalls in diagnosis.

Methods

We searched Medline and the Cochrane Library using terms such as "necrotising fasciitis" and "Fournier's gangrene." We also drew on our own experience. The overall quality of evidence is weak to moderate.

How common is necrotising fasciitis?

About 500 cases of necrotising fasciitis a year occur in the United Kingdom.[1] Although rare, the infection occurs often enough for most emergency department doctors and general practitioners to see a case in the course of a working lifetime. The key to successful treatment lies in early diagnosis and appropriate management.

LEARNING POINTS

Necrotising fasciitis is a lethal and rapidly progressive soft tissue infection, which can occur in healthy young patients

People with diabetes, those who inject drugs, and patients with haematological malignancy are particularly at risk

Diagnosis requires a high index of suspicion. Consider necrotising fasciitis especially when the presentation is "not quite right" or the patient is not responding to treatment

Early surgical exploration of the soft tissues has little morbidity and may be the only means to reach a definitive diagnosis and expedite treatment

In established necrotising fasciitis, surgery gives a 60-80% chance of survival. The earlier the first exploration and subsequent debridement, the less extensive the resection and postoperative morbidity is likely to be

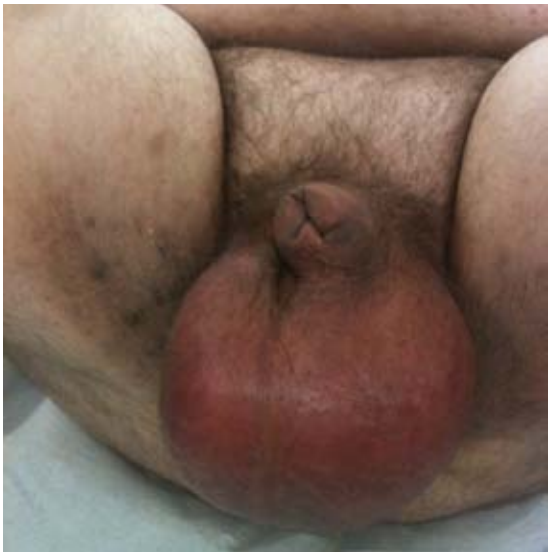

Fig 1 | Necrotising fasciitis of the scrotum, with erythema of the scrotum. Subtle necrosis can also be seen in the thigh area. Further images are also available at http://dermnetnz.org/doctors/bacterial-infections/necrotising-fasciitis.html

This condition is catastrophic if missed. Even with surgery, mortality is 20-40%.[2 3] Delay in diagnosis increases mortality,[4-6] and those who survive need more extensive surgery, reconstruction, and often amputation. With early diagnosis outcome is much improved[7 8] and significant long term disability is reduced or prevented.

What are the clinical features?

Necrotising fasciitis is notoriously difficult to diagnose. The initial symptoms are non-specific up to the point when the patient rapidly deteriorates, and septicaemia develops, often accompanied by shock or confusion. However, this clinical course is often slower than might be expected. Fever or pain develops first, so the patient often presents initially to primary care or the emergency department. The pain may seem to be disproportionate to the clinical findings.

Table 1 | Percentages of patients who show signs and symptoms of necrotising fasciitis at presentation

Study and country	Severe pain	Fever	Tachycardia (with or without hypotension)	Skin erythema	Skin oedema	Skin tenderness	Blistering or bullae	Ecchymosis or skin discoloration	Crepitus
Wong et al[12] (n=89), Singapore	98	53	74 (18)	100	92	98	45	No data	14
Childers et al[11] (n=162), United States	100	70	No data	95	82	No data	16	49	25
Frazee et al[16] (n=122), United States	No data	44	59 (21)	80	66	54	12	No data	7
Angoules et al[17] (n=451), United Kingdom	63	15	(12)	73	49	No data	15	No data	7

Cellulitic skin changes may develop next. The presentation may mimic haematoma, bursitis, phlebitis, sciatica, cellulitis, septic arthritis, or deep venous thrombosis. The classic textbook picture of haemorrhagic bullae, crepitus, and skin necrosis often does not occur until day 5 or later.[9-15] The patient may seem systemically well until relatively late. Table 1 shows the frequency of signs and symptoms at presentation. Figure 1 shows necrotising fasciitis of the scrotum.

The patients who present the greatest diagnostic difficulty are those presenting with pain but without fever or systemic signs. Pain is caused by tissue necrosis, but the nerves can also be infarcted as perforating vessels to the tissues are occluded by thrombus during the necrotic process. This can result in exquisite pain and tenderness but also in sensory loss to the overlying skin. The area may be tender or tense. Pain is often very severe, preventing weight bearing or use of the limb but may be mild until late in the process. People who inject drugs often present without systemic signs.[16]

Even in patients with systemic signs, the severity of the skin infection is often not apparent initially. The skin may look normal, or there may be erythema suggestive of cellulitis. In true fasciitis there will be no ascending lymphangitis, but this may be present in other, more superficial necrotising soft tissue infections.

How is necrotising fasciitis diagnosed?

Necrotising fasciitis affects all age groups but is particularly rare in childhood. It is more common in patients with diabetes, chronic hepatitis, and malignancy (particularly leukaemia) and in people who inject drugs. Iatrogenic immunosuppression also increases the risk. Intra-abdominal malignancy or sepsis can lead to necrotising fasciitis of the abdominal wall. Varicella infection is a recognised risk factor in children.[18] Any puncture wound or surgical procedure can introduce infection, including such minor procedures as acupuncture or intramuscular injection. However, about 25% of cases occur in patients without comorbidity or precedent trauma.

Table 2 | Diagnostic scoring system of the Laboratory Risk Indicator for Necrotizing Fasciitis

Variable	Score
C reactive protein (mg/L):	
>150	4
White blood cell count (per mm^3):	
<15	0
15-25	1
>25	2
Haemoglobin (g/dL)*:	
>13.5	0
11-13.5	1
<11	2
Sodium (mmol/L):	
<135	2
Creatinine (µmol/L):	
>141	2
Glucose (mmol/L):	
>10	1

*The corresponding SI values for haemoglobin are >135 g/L, 110-135 g/L, and <110 g/L.

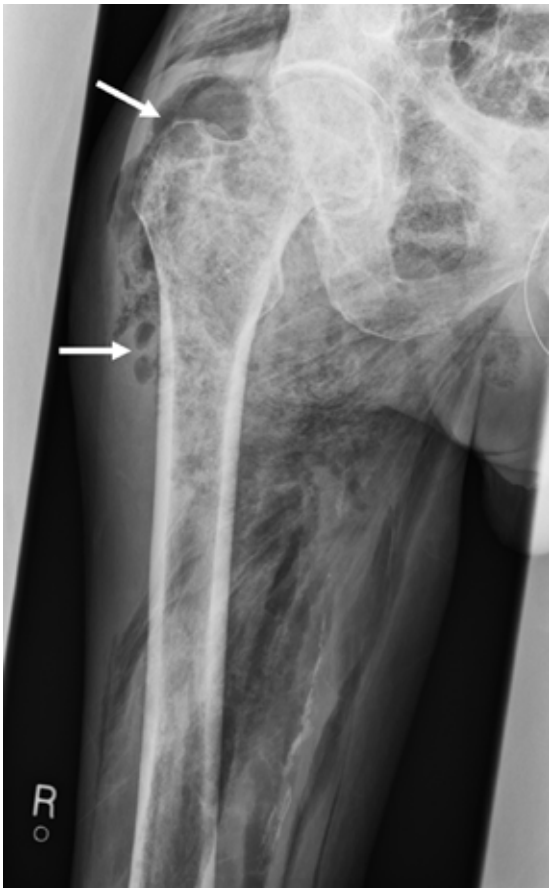

Fig 2 | Extensive subcutaneous gas in the thigh of a man with necrotising fasciitis of the buttock (arrows)

In patients with fever, suspicion may be aroused by something being "not quite right" for a diagnosis of cellulitis. The classic cyanotic and bullous skin changes may only appear late in the process; however, the site of infection may appear unusual. The pain may seem too severe for cellulitis, despite relatively mild skin signs, or there may be overlying sensory loss. The patient may seem disproportionately unwell for the degree of skin involvement. The progression of the illness also suggests the diagnosis. The patient may seem relatively well initially, but will deteriorate despite treatment with antibiotics. Close observation is important for identifying those patients whose disease is not progressing as expected.

In patients presenting with pain alone, the severity of the pain and absence of trauma may suggest the diagnosis of necrotising fasciitis. In patients with unexplained pain, especially severe or rapidly progressing pain, a search for covert sepsis and an investigation of inflammatory markers is advisable.

What investigations are useful?

No investigations are diagnostic, but blood test abnormalities such as a raised C reactive protein concentration occur relatively early, reflecting the systemic inflammatory response. The most accurate diagnostic scoring

system to date is the Laboratory Risk Indicator for Necrotizing Fasciitis (table 2).[19] A score of ≥6 was 93% sensitive and 92% specific for necrotising fasciitis in a Singaporean population but achieved only 74% sensitivity and 81% specificity in a UK validation study (H Y Sultan et al, unpublished UK data, 2011). Blood cultures take too long to influence immediate management but have a role in guiding further antibiotic treatment.

Although the validity of this score for a UK population may be in doubt, it demonstrates the relative importance of certain laboratory tests. Hyponatraemia in the presence of sepsis and clinical signs of soft tissue infection should be considered highly suspicious for a necrotising soft tissue infection.

Computed tomography can show fascial swelling, inflammation, and sometimes soft tissue gas and is sensitive (100% in one small series) but less specific[20]; magnetic resonance imaging is also sensitive[21] but often not feasible or available. Ultrasonography can be diagnostic but requires a highly skilled operator.[22] Although plain radiography is not the imaging of choice when the diagnosis of necrotising fasciitis is suspected because sensitivity and specificity are low, it may show subcutaneous gas (fig 2).

The mainstay for investigation and treatment remains surgical exploration. The decision to explore the soft tissues should be made early. An incision over the site of maximal skin change is needed to assess the underlying tissues. Healthy subcutaneous fat and fascia indicates that further resection is not needed, and the morbidity to the patient is limited to a short scar.

However, if exploration shows necrotic fascia, fat, or the "dishwater" appearance of liquefied necrotic tissue, resection can be done until healthy tissues are reached. This can be facilitated by the "finger sweep test" (necrotic fascia loses its adherence to surrounding tissues and the plane opens abnormally easily until the limit of the disease is reached). Where doubt over the appearances persists, send specimens for histology to look for evidence of necrosis and microbiology for urgent Gram staining.

Necrotising soft tissue infections used to be considered streptococcal, but they are now understood to be often caused by mixed pathogens, including gas forming bacteria such as *Clostridium* species.

How is necrotising fasciitis managed?

Early referral to a surgeon and an early decision to explore and debride is the cornerstone of treatment. In established sepsis, debridement does not bring about a rapid change in the condition of patients. However, over the following hours they tend to stabilise but then often spend several days needing invasive support in intensive care and have an overall hospital stay averaging 33 days.[23] Subsequently, the patient will be referred to the nearest plastic surgery or burn centre for ongoing wound care and reconstruction.

Adjuvant measures include systemic support in an intensive care setting and antimicrobial treatment. Broad spectrum antibiotics such as a benzylpenicillin and flucloxacillin are used. Clindamycin has an additional role owing to its bacteriostatic mechanism. It inhibits the production of the streptococcal superantigen, which greatly contributes to septic shock.[24]

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A PATIENT'S JOURNEY

Kidney dialysis—the need for humanity

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This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

Renata Carey provides a patient's perspective on chronic kidney disease, being considered for transplantation and starting dialysis

From my perspective as a patient, dialysis can appear to lack imagination and kindness. My kidney failure was diagnosed in 2005. At that time, I was on another journey, one of indescribable and agonising sadness. My husband was dying. For many months I cancelled appointments. Eventually I went, not because of any symptoms but because I thought it best to find out what I should do. I was 74 and was referred to the low clearance clinic.

This was a kind and decent set-up with excellent nurses monitoring the gradual deterioration of people's kidneys. Dialysis was never mentioned. Some time later came the first appointment with the consultant. Now a transplant was proposed: I was put on the transplant list. I started attending the low clearance clinic in 2007, and at the end of 2009 the superb consultant said that dialysis must now begin.

So dialysis began in January 2010. It was particularly unfortunate that the first thing I passed on my way to the new dialysis unit was the mortuary: a sad low brick building with lots of little windows—maybe for the corpses to breathe better. Then followed a tiring uphill climb to the unit. And when at last I got there, a closed door greeted me. "Any chance of you kindly opening it when you arrive at 6.30 am?" I asked one of the nurses.

"We change into our uniforms, we've got all the machines to get ready, we've . . ." "But wait a moment," I pleaded politely. "It's freezing out here in the dark in the winter. We would only go into the warm waiting room—obviously not into the machine rooms until we're allowed. We'd just sit there warmly till everything was ready." End of conversation. And how I did hate having to suggest an idea to "someone higher up."

Other things were much more complicated to suggest or complain about. Kindness, thought, imagination—all these should have been the order of the day. But those depended on the personalities of the nurses and doctors involved. The kidney consultants and nurses at the hospital before dialysis were mostly excellent. But the dialysis doctors were from a different world—both the consultants and the staff who rushed around keeping an eye on patients' blood results and general progress. Fantastic invention that the dialysis machine was, how hard it must have been for those doctors not to be in an active, positive, curing role—instead they were watching their patients, immobile and attached to machines for four or five hours, three times a week, patients who are hardly over the moon about that. Not quite true, that last bit: a GP had once told me that one of his patients who had at last received a transplant actually missed the jolly camaraderie of it all and the routine of coming for dialysis.

A DOCTOR'S PERSPECTIVE

Renata was first referred to the renal clinic in 2005 with a previous history of a hemicolectomy for carcinoma of the bowel, type 2 diabetes, ischaemic heart disease, and deteriorating kidney function. She had been noted to have hypertension and abnormal kidney function in 2001, but these had been appropriately managed in the community and her care was transferred to me in the context of her renal function and proteinuria. The initial assessment was of a well woman with chronic kidney disease stage 3-4, presumably caused by her diabetes, hypertension, and atherosclerosis. It became apparent that she needed coronary artery bypass graft surgery. She had the surgery, successfully, and for the next two years she attended routine clinics where the focus was on treating her hypercholesterolaemia, hypertension, and monitoring of her renal impairment.

In 2006 Renata lost her husband, which deeply saddened her and she was very reluctant to contemplate dialysis and initially not keen to consider any active treatment. However, she began to explore the possibilities of transplantation and was assessed for this. In our practice at the time it was extremely unusual for a patient in her 70s with diabetes to be considered for transplantation because of the high risk associated with the procedure and the scarcity of deceased donor kidneys. However, her recent successful bypass surgery, which she had tolerated well, was very much in her favour, as was her remarkable level of fitness, enthusiasm, and engagement in the medical aspects of transplantation. There were no obvious live donors that she was willing to consider, and after a few additional tests, her name was added to the transplant list. However, as part of an agreed back-up plan, a forearm fistula was formed.

Over the next two years, without any offers of a deceased donor kidney, her renal function gradually and predictably declined, and towards the end of 2009, we felt that her symptoms of malaise and fatigue resulted from her renal failure, and she was urged to start haemodialysis three times a week. This started in January 2010.

Diabetes and hypertension are very common causes of chronic kidney disease and renal failure, and Renata's history will be echoed by thousands of other patients with diabetes and renal impairment.

Mark Harber, renal consultant

USEFUL RESOURCES FOR PATIENTS AND HEALTHCARE PROFESSIONALS

- British Kidney Patient Association (www.britishkidney-pa.co.uk)—A UK registered charity that gives information and advice for those with kidney disease, grants to help patients and families needing financial help with domestic costs, hospital travel, education, and holidays, and financial support to UK kidney units
- British Renal Society (www.britishrenal.org)—Promotes patient centred multiprofessional care for people with kidney failure and their families and carers; advances education about renal disease and replacement therapy in the UK; and helps fund multiprofessional research into kidney disease and management
- National Kidney Foundation (www.kidney.org)—A US voluntary, non-profit organisation dedicated to preventing kidney and urinary tract diseases, improving the health and wellbeing of individuals with kidney disease and of their families, and increasing availability of organs for transplantation
- Kidney Health Australia (www.kidney.org.au/)—A not for profit organisation focusing on improving kidney health and developing initiatives that reduce the incidence of kidney disease in Australia

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It is not a good idea to say to a patient, as the dialysis doctor said, "I'm being very honest. I mean, I'd never have put you on the transplant list: you're much too old—you would probably die on the table. People always think all will be fine when they've got that kidney. But indeed not, masses can go wrong."

"Can you take me off the list?" I inquired gently. "Unfortunately not. But your consultant could." *My* consultant? Yes, he was, I supposed. It was at my last consultation that he'd said "I see you have two children." And I sensed what was coming. "You know live donors are far better than dead ones," he said, adding, "Can you ask them if they would give you a kidney?"

"I gave birth to my children; I nurtured them; I tried with all that is in me to let them have fulfilled and happy lives. They might need kidneys for their own children; for their wives or husbands; they might have accidents that destroy their kidneys. Do you have children?"

"Yes I do."

"Then think!"

This conversation was not a good way to embark on a relationship—but by god I was shocked by his suggestion.

And then there were the masses of other machine people. Vaguely, one said good mornings into the air on entering the waiting room. Apart from that, I probably conversed with only three patients, of different nationalities, all of whom gave me interesting lectures on the history and politics of their countries.

And now there is a new development: self care, which they try to foist on everyone. Ghastly for patients to have to think "dialysis" all the time and struggle with tricky technical procedures ("leave it to the professionals" we might prefer). And the end aim—to have our own machine at home. At home alone, within four walls and with a machine and bags.

There are other things that I could mention. Firstly, I feel we should be forewarned that eventually we stop passing urine; happily it hasn't yet happened to me. But the idea is pretty horrid physically and psychologically. And secondly, what I call the first world war scenario. (The students I tutor one to one are often doing projects on this war: the terrible slaughter, the trenches.) Anyway, throughout dialysis, patients suddenly appear with amputations; and very often with heavily bandaged feet, rapidly followed by crutches and then wheelchairs.

To summarise: dialysis is brilliant, of course, but deeply horrendous. The people running it must be selected for heart and intelligence and imagination. Above all, they need heart and humanity.

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A PATIENT'S JOURNEY

Irreversible renal damage from accidental mushroom poisoning

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This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

Nicholas Evans, author of *The Horse Whisperer*, tells the story of his journey through acute renal failure to successful transplantation

We were visiting family in the north of Scotland. We thought the mushrooms in the woods behind the house were ceps and chanterelles, but we were wrong. The "ceps" turned out to be *Cortinarius speciosissimus*, a cousin of the more widely known deadly webcap. I have picked mushrooms all my life and never before eaten anything without getting 100% confirmation from my two guide books. Our error with the supposed ceps was the result of two people each trusting the expertise of the other—and the consequences were catastrophic.

A blessing was that none of the children who sat down to lunch wanted to taste the mushrooms I'd so proudly cooked. Had they done so, they would almost certainly have died. The guide book, consulted only the next morning when their parents were starting to feel ill, showed a skull and crossbones. It said what we had eaten was "deadly poisonous."

Within 48 hours my wife and I were in the local hospital, and by the end of the week we were in Aberdeen Royal Infirmary with tubes in our necks and having our first experience of dialysis. We had black diarrhoea and nausea and retched every few minutes until all that came was blood and bile. I thought we were going to die. And there were times, in the dark, early hours of another sleepless night, when but for the thought of my children, I rather wanted to. I called my solicitor and had him courier me a new draft of my will. After a few days, two of my older children flew up to take our young son home to Devon.

We remained in hospital in Aberdeen for about three weeks. Our nephrologist worked deep into the night deciphering Scandinavian and German medical papers on the treatment of *C speciosissimus* poisoning. The toxin in this mushroom is only interested in the kidneys, and some research suggested that massive doses of antioxidants could prevent the total annihilation of the kidneys. We tried it, but it didn't do the trick. Our creatinine levels soared from the normal of around 70 µmol/L to well over 1000 µmol/L. Biopsies showed extensive cell necrosis. My wife retained a minimal amount of renal function, but I had eaten more of the mushrooms than she had, and I had almost none. I stopped peeing entirely. I used to have regular peeing dreams and wake up thinking I was cured.

A MEDICAL PERSPECTIVE

Nicholas Evans eloquently describes the devastating consequences of poisoning by the mushroom *Cortinarius speciosissimus*. The toxin from the mushroom causes severe acute tubular necrosis, which meant that Nicholas became dependent on dialysis within a few days. The renal damage is irreversible; renal transplantation was his only option if he wanted to get off dialysis and improve his quality of life. Reading Nicholas's article, I was struck by how physically and mentally overwhelming his "crash land" on to dialysis was, going from health to organ failure in a matter of days.

Patients who have a slow decline in their renal function have the benefit of an early referral to a nephrology clinic and time to adjust to their situation, symptoms, and psychological state, aided by the multidisciplinary team.

The value of predialysis education is enormous in preparing patients. Some element of choice is introduced in terms of modality. Indeed, the procedure of having a fistula constructed or a peritoneal dialysis catheter placed is a psychological milestone in the journey towards dialysis. A home visit from a specialist nurse, in the patient's own environment rather than a sterile clinic room, can help the patient think about the practicalities of renal replacement therapy: showering with a tunnelled line; parking and transport at the dialysis unit; what happens if it snows? These conversations may be better held with front line staff, who appreciate the social perspectives without a clinician's agenda in mind. Patients have an opportunity to visit a dialysis unit and to speak to other, more experienced patients as part of their preparation process.

"Crash landers" have none of these benefits, and a multitude of uncertainties, including recovery, finance concerns, and temporary access to dialysis that may be suboptimal.

Nicholas describes dialysis as gruelling. He would have been an obvious candidate for home haemodialysis. In a satellite dialysis unit some patients build a new social network, having dialysis in the same slots week in and week out, and become friendly with the staff and other patients—even romantic unions can take place. Home haemodialysis might have given Nicholas more control over his dialysis but perhaps less engagement with the dialysis community. Haemodialysis requires "ultra pure" water, and unfortunately the age, rurality, water supply, and sewerage of his home made this impractical to achieve.

Another option would have been peritoneal dialysis, which in Nicholas's case might have been less of a burden on his cardiovascular system, and given him far greater independence and travel possibilities.

Unfortunately no renal replacement therapy is perfect, but, as with many of our patients, transplantation became the preferred aim for Nicholas. Transplantation, whether living or cadaveric, remains an ideal way of avoiding the need for dialysis (along with its complications) and providing a cost effective long term solution. Clearly no patient has a completely unchequered course after transplantation, but Nicholas's piece emphasises the benefits for him of a live donation.

Alexander Hamilton, renal registrar; **Maria J Bello-Villalba**, consultant nephrologist; **Coralie Bingham**, consultant nephrologist

We flew back home to Devon, where my kids had rigged up balloons and a big "welcome home" banner across the driveway. My wife and I had tried to hide the monstrous, Frankenstein tubes in our necks with bandanas but to no avail. I'll never forget the shock on our young son's face as he surveyed his new parents. We looked like the walking dead.

We started dialysis at the Royal Devon and Exeter Hospital, then in Newton Abbot, where the ancient dialysis unit, perched on top of a hill, overlooked the town's otherwise derelict hospital. We then moved to a new unit, in Torbay. Our consultant nephrologist and all the friendly staff there looked after us with great care and attention.

Life on haemodialysis is a gruelling business. Occasionally your blood pressure plummets and you tumble into a slough of nausea. I had to do five hours every other day. My wife found she could only bear three hours. At first I thought that 15 hours of enforced stillness a week would provide an excellent chance to finish my new novel. It didn't. Dialysis blurs the mind, making it hard to concentrate. I discovered that typing with only one hand (as I had one arm needled to the machine and out of action) disrupted the rhythm of my thoughts. Usually when I came home I felt too sick and disoriented to work.

In fact, you feel only half well all of the time. Even after all those hours on a machine, your blood is never more than a quarter cleaned of all the toxins that healthy kidneys normally get rid of. Your lifestyle is blighted too. If you want to travel, particularly at holiday times, you have to fix up dialysis for where you want to go many months in advance. The units are often unable to take you. Europe, in my experience, was fine. I've had dialysis in the Netherlands and Italy several times in good and friendly units—and free, with my European health insurance card. In the United States, where I used to travel often and widely, you have to pay for dialysis and it's extremely expensive. In New York it cost me $1000 a session. It was a stark reminder of how marvellous our own NHS is.

However, there was always hope. I had no shortage of offers of a new kidney. All four children offered. But a parent's every instinct is to protect his children. Putting them at risk, however slight it might be, seemed unconscionable. Anyway, I had other offers. Seven friends, amazingly, offered me a kidney. It still moves me enormously that they should do so. Without knowing what I now know, would I have done the same for them? I'd like to think so, but I can't be sure.

But, one by one, they failed the tests. I'm blood group O, and oddly nearly all of them turned out to be group A. ABO incompatible transplants can be made to work but they are not ideal. And some of these wonderful friends found during the tests that they had medical problems of their own, which made it unwise to proceed.

I've always liked to keep active, but by the beginning of last year I found I couldn't run more than a few hundred yards without having to stop. My heart had always been healthy, but tests showed it was now being damaged, probably by the fistula. My pulse, normally about 45 beats per minute, was 70.

This was the moment when my daughter, Lauren, by now 29, said: "Dad, it's time to get serious. I want to give you one of my kidneys." I repeated what I'd been saying for two and half years: I couldn't do it. She said I shouldn't see it as a sacrifice on her part, that it was entirely selfish: she wanted me to live long enough to meet her children when she had them. She's a scientist and had done all the research. She said the risks were tiny. She had gone ahead and had herself tested.

USEFUL RESOURCES

- Give a Kidney, One's Enough (www.giveakidney.org)—A charity promoting altruistic living kidney donation
- Kidney Research UK (www.kidneyresearchuk.org)—A charity that funds research aimed at finding better treatments, and ultimately a cure, for kidney disease

We're quite alike and have always been very close. It was no surprise to discover she was an excellent match. Four out of the six criteria boxes were now ticked.

Reluctantly, I went with her to the Hammersmith Hospital in west London, where a nephrologist spent many hours with us patiently going through the research. My greatest worry, despite several studies showing no risk to conception or childbirth, was that it might reduce Lauren's ability to have kids. We were put in touch with a young woman who had given her mother a kidney and gone on to have two healthy children with no complications. I spoke to the mother (who'd had all the same misgivings) and Lauren spoke to the daughter. They were immensely positive and reassuring.

"So, Dad, shall we do it?" Lauren said, as we left the hospital, my arm around her shoulders. I couldn't speak. We were both in tears. I hugged her and managed to nod and whisper okay.

The transplant procedures took place in July 2011. The surgeon had told us what to expect, and the first few days were a bit of an ordeal for both of us. I got very sick and Lauren was in a lot of pain. But the good news was that her kidney, nestling on the right in my lower abdomen, was working like a Ferrari. I had not peed for three years, so my poor, shrivelled, old bladder took quite a while to get up to speed.

Lauren went home after about a week, and I stayed about a week longer. It took her about three or four weeks of rest and recuperation to feel well again. A couple of weeks more and she was out running again. My recovery took a little longer. I caught a couple of minor infections that put me back in hospital for a few days each time. But within seven or eight weeks I was feeling well and increasingly mobile.

Now, a year later, my daughter is living in Kenya doing fieldwork for a PhD. She is fit and strong, runs every day, and has 100% kidney function. Her one kidney has grown and is doing the same work that two did before. We talk almost every day. The bond between us is stronger than ever. As for me, I feel every bit as well as I did before the poisoning. I run about 12 miles a week, I can eat anything I like (nuts, bananas—all those high potassium foods that were banned on dialysis). On my son's 10th birthday, I even went down the "death slide" at our local theme park.

I recently went back to the dialysis unit. In one year, six dear friends there have died. Some people manage dialysis for 20 years, but the average life expectancy on dialysis is five to eight years. And as I now know, being on dialysis isn't really a life. It's not even half a life.

Just over 12 months after my transplant, my wife also had one—from a family friend. She is doing well. Four years after the poisoning, we have our lives back. We have learnt a lot about our families and friends and the extraordinary generosity at large in the world. It has been quite a journey.

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ANSWERS TO ENDGAMES, p 50 For long answers go to the Education channel on bmj.com

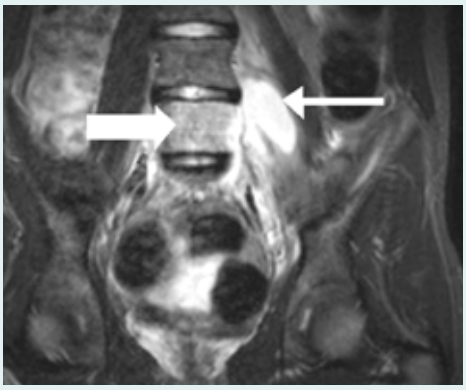
PICTURE QUIZ A sinister cause of back pain in a young man

1 This STIR (short T1 inversion recovery) MRI scan shows high signal in the L5 vertebral body, with a large paraspinal high signal area that displaces the body of the left psoas muscle laterally (figure). There is no abnormality of the intervertebral discs. The most likely diagnosis is a psoas abscess with associated L5 osteomyelitis.

2 Immunocompromise, inflammatory bowel disease, and diverticulitis are possible underlying causes. Send blood cultures and percutaneous aspiration for microscopy, culture, and antibiotic sensitivity. Perform staining and culture for acid fast bacilli and a chest x ray to check for signs of pulmonary tuberculosis. Consider colonoscopy and barium enema to investigate the underlying cause.

3 The most common causative organisms in psoas abscess are *Staphylococcus aureus* and *Escherichia coli*. Start broad spectrum antibiotics while awaiting antibiotic sensitivity test results. Fusidic acid and bed rest are also indicated because of the associated osteomyelitis. Also consider percutaneous drainage or open drainage of the abscess.

4 Bacteraemia, mycotic aortic aneurysms, seeding of the abscess, mass effects on vessels, sepsis, and organ failure.



STIR (short T1 inversion recovery) magnetic resonance imaging scan showing high signal in the L5 vertebral body (large arrow), with a large paraspinal high signal area that displaces the body of the left psoas muscle laterally (small arrow)

STATISTICAL QUESTION Observational study design

Answer *d* best describes the above study design.

CASE REPORT Skin rash in a preterm infant

1 The characteristic facial distribution of the rash and the presence of oral and buccal mucosal lesions make infection with herpes simplex virus (HSV) the most likely diagnosis.

2 Perinatal transmission.

3 Disseminated disease, severe hepatitis, infection of the central nervous system (CNS), ocular disease, and neurodevelopmental sequelae.

4 Intravenous aciclovir for three weeks for disseminated disease or infection of the CNS.