

Flank pain, haematuria and a rash

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Emergencies can spring up at any time and in many incarnations. Are you adequately equipped to deal with them? Each month we present a case study in emergency medicine based on real cases and events.

It is a typically chaotic shift in the local hospital's emergency department where you regularly work as a GP. As usual, inpatient beds are not available and the log jam puts the emergency department into a shambles.

The department's computer system shows your first patient has been classified at triage as a category 3, i.e. requiring to be seen within 30 minutes. The only place available for examination is a couch – not a bed – in the treatment cubicles, which are usually used for dealing with the walking wounded, such as those with lacerations. You promise the nurse you will be quick as when these treatment rooms get jammed up with admissions the ship is well on its way down.

Presentation and history

The triage nurse had done a great job – all the essentials were documented on this 53-year-old woman. 'Presented with blood "+++" in urine, right iliac fossa pain, red rash lower legs to abdomen.' The basic observations were, 'Well perfused, not distressed, Glasgow coma score 15/15 (normal). Urinalysis done (pH 7.5, large blood, large protein), urine sent for culture'. A nurse has also taken blood and sent it to

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the laboratory before you see the patient. You are fortunate to work with nurses who are happy to take blood and do procedures – this is definitely a great improvement over what used to happen in 'cas'.

You see your patient – a pleasant, overweight, anxious woman – an hour after her arrival in the department. You feel she is happy to see an older doctor as she quickly lets it be known she is frightened of hospitals and tests, and asks whether it is really necessary for her to be seen. You reassure her. In the back of your mind, you are expecting to diagnose a simple urinary tract infection and a simple unconnected rash.

You quickly learn from the patient that her GP, a friend of yours, had told her to come to the emergency department when she rang him that morning. Just like most patients nowadays there are several aspects to her medical history. She does not give a clear story but it includes a 24-hour history of right loin pain (a dull ache) that was not relieved with paracetamol. Over the same time the rash spread from her calves, up her thighs to her abdomen. Questioning revealed she had painful urination, with frequency every two hours – all through the night.

Her past history included appendicectomy, caesarean section, cholecystectomy, partial hysterectomy and arthritis. Eight years ago she had been assaulted and robbed by juveniles, an incident that left

her with panic attacks, agoraphobia and depression. About 12 months ago she had fallen and sustained a fractured ankle. While in hospital because of her ankle, a deep venous thrombosis (DVT) occurred; the ensuing warfarin had been ceased four months ago. The DVT had made her chronic ankle oedema worse.

Her current medications include sertraline (Zoloft), celecoxib (Celebrex) and budesonide (Rhinocort), frusemide (Lasix) for the past eight weeks and, for the past week, quinine sulfate at night for leg cramps. She is allergic to penicillin and sulfur-containing drugs.

She is divorced, on a pension and lives alone. She has a boyfriend. She then remembers to tell you that she has had



Figure. The petechial rash on the patient's calves also covered her thighs and abdomen.

Table. Possible causes of decreased platelet count

Marrow failure – e.g. aplastic anaemia or malignancy (marrow biopsy abnormal)
Peripheral destruction or sequestration – e.g. idiopathic thrombocytopenic purpura (increased platelet IgG), hypersplenism (clinical splenomegaly), disseminated intravascular coagulation (abnormal coagulation)

Source: Dodds A. Emergency department haematology. In: Fulde GWO, ed. Emergency medicine: the principles of practice. 3rd ed. Sydney: MacLennan & Petty, 1998: 398.

major dental procedures over the past few weeks.

Not a straightforward diagnosis

You scan her vital observations – temperature 36°C, blood pressure 170/95 mmHg, pulse rate 88 beats per minute, oxygen saturation 96% – and then she shows you her rash (Figure).

Now you lose any hope of a simple diagnosis. The rash extends from her calves to her abdomen, and is petechial but not painful or pruritic. General examination reveals right loin tenderness and very noticeable bruising on the chest where the ECG electrodes have been, as well as at the tourniquet site for the blood tests.

The laboratory results come back:

- haemoglobin 134 g/L (normal range 115 to 165 g/L)
- white cells $2.1 \times 10^9/L$ (normal range 4.0 to $11.0 \times 10^9/L$)
- platelets $2 \times 10^9/L$ (normal range 150 to $400 \times 10^9/L$).

These results indicate neutropenia with profound thrombocytopenia.

Clotting studies are normal, as is most of the biochemistry except for a slightly raised blood glucose level of 8.5 mmol/L (normal range 3.0 to 7.8 mmol/L) and raised liver enzymes (gamma glutamyl transferase and alkaline phosphatase).

Spontaneous haemorrhage a possibility

The rapport you built up with the patient earlier helps now in convincing her to come into hospital. You tell her she needs ‘packing in cottonwool’ for the moment because she is at serious risk of spontaneous haemorrhage. This is usually a risk at a platelet level below 20 to $30 \times 10^9/L$; this patient has a platelet level of $2 \times 10^9/L$.

You consider the possible causes of a decreased platelet count (Table 1).

You have, however, seen a case similar to this that was caused by quinine. Not being able to think of any other clever or obvious cause, you make a provisional

diagnosis of reduced platelet count caused by quinine. A drug dependent platelet antibody immunofluorescence assay by flow cytometry is organised.

A phone call to the patient’s GP confirms the history. You then call the haematologists who, on assessment of the patient, agree with you completely but note the leucopenia and comment on possible toxic changes in the film. They add the differential diagnosis of acute leukaemia or lymphoproliferative disorder.

A CT scan ordered to look for any possible retroperitoneal blood is normal.

The consultant institutes intravenous steroids (dexamethasone 8 mg immediately and 4 mg six-hourly) and also immunoglobulin, normal (human) 24 g daily, and a platelet transfusion. The antiarthritis COX-2 inhibitor celecoxib is ceased and, of course, the quinine sulfate is not charted. A bone marrow aspiration is arranged and performed.

The next few days

The gross haematuria clears by the next day, and the patient tells you the bone marrow aspirate site hurts.

It then becomes evident that the steroids have made the patient hyperglycaemic – fasting blood glucose level 17.7 mmol/L. Neutral insulin (Actrapid) is commenced (12 units daily).

The next day the urine culture results come back, showing *Escherichia coli*. Oral cephalexin 500 mg three times daily is commenced.

No tonic water allowed

The provisional diagnosis is confirmed by the immunofluorescence assay: quinine is the culprit.

The patient is discharged the next day on oral prednisone after being fully briefed about avoiding quinine – i.e. not drinking tonic water or bitter lemon drinks.

You are impressed by a very clever test but most of all by the patient doing so well with one of the lowest platelet counts you have ever seen.

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