Increasing breathlessness, hyperkalaemia and thrombocytosis in a patient with chronic obstructive pulmonary disease

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CASE REPORT

A 72-year-old gentleman with no past history of chronic obstructive pulmonary disease (COPD) was admitted with a history of progressive shortness of breath, increasing over the preceding 2–3 months. His exercise tolerance was decreasing and he was unable to climb stairs without becoming breathless. He described a cough on most days productive of creamy white sputum but he had never coughed up any blood. He denied wheeze, ankle oedema, orthopnoea or paroxysmal nocturnal dyspnoea. There was no history of chest pain. He was not on any medication and had no allergies. He was an ex-smoker. He smoked an average of 20 cigarettes a day (one pack) for almost 30 years giving him a total of 30 pack-years of smoking. He denied drinking alcohol. There were no symptoms referable to any other system.

On examination, he was breathless, cyanosed and tachycardic with a pulse of 120 regular, blood pressure of 160/70 mmHg, and temperature of 37.7°C. His oxygen saturation on air was 77%. Heart sounds were normal. Jugular venous pressure was not visible and there was a slight pitting oedema of his right foot. He had widespread reduced breath sounds on respiratory examination with widespread inspiratory crackles. Abdominal and neurological examinations were unremarkable.

Initial investigations showed haemoglobin 11.9 g/dl (normal range (NR) 13–18 g/dl) with normal red cell indices. His white cell count was 16.9×10^9/litre (NR $4–11 \times 10^9$/litre), platelets 1339×10^9/litre (NR $140–440 \times 10^9$/litre), sodium 138 mmol/litre (NR 133–148 mmol/litre), potassium 6.9 mmol/litre (NR 3.5–5.5 mmol/litre). Urea, creatinine, bicarbonate, glucose calcium, creatine kinase and liver function were all normal. Blood gases on air showed marked hypoxia with a pH of 7.38, PaO_2 6.1 kPa and $PaCO_2$ 7.6 kPa. Oxygen saturation was 81.3% and his bicarbonate level was 32.6 mmol/litre. Chest X-ray showed normal heart size with prominent peri-hilar vasculature and there were some interstitial change in the right lower zone with a small right basal effusion. Subsequent repeat chest X-ray showed the lungs to be hyperinflated but clear, with the effusion having resolved. Electrocardiography showed a sinus tachycardia. On spirometry his forced expiratory volume in 1 second was 1.12 litres (40% of predicted), forced vital capacity was 2.1 litres (59% of predicted) giving a ratio of 53%. Gas transfer corrected to his haemoglobin (DLCO CORR) was 14.17 (59% of predicted) and the ratio of his gas transfer to alveolar volume (DL/VA) was 3.89 (101% of predicted).

He was initially thought to be having an infective exacerbation of COPD and was treated as such with steroids, nebulisers, antibiotics and prophylactic subcutaneous heparin. He had a good clinical response to these. However, on noting his raised platelet count, aspirin 150 mg was started and a perfusion lung scan arranged. The latter showed segmental defects, particularly in the left lung field, with the appearances suggesting pulmonary emboli (*Figure 1*). In view of his asymmetric ankle oedema a venogram of his right leg was arranged. This proved negative. His relatively raised potassium level was thought to be spurious and a repeat check on a lithium heparin blood sample gave a reading of 4.3 mmol/litre (NR 3.5–5.5 mmol/litre). A bone marrow trephine was reported as markedly hypercellular. There was hyperplasia of white cell series with numerous mature forms and reduced erythropoiesis which was normoblastic. Megakaryocytes were markedly increased in numbers, many occurring in small clusters with dysmorphic hyperlobated nuclei and abundant cytoplasm. There was no increase in the blastic component, lymphocyte or plasma cell population or of reticulin fibrosis within the marrow. The appearances were consistent with a myeloproliferative disorder and were in keeping with an essential thrombocythaemia. Ultrasound scan showed normal liver, kidneys and spleen. With the results of the perfusion scan the patient was anticoagulated with warfarin and started on allopurinol and hydroxyurea. He was mobilized and made an uncomplicated recovery.

INTRODUCTION

Thrombosis is a well-recognized complication of essential thrombocythaemia (ET). This paper discusses a patient with stable chronic obstructive pulmonary disease presenting with a rapidly progressive exertional dyspnoea. This was found to be caused by ET-related pulmonary occlusion and infarction, most probably related to in-situ thrombosis. He also had a high serum potassium level which proved to be spurious and most likely related to leakage from his abnormal platelets. The importance of promptly substantiating the diagnosis of ET and starting aspirin therapy in such patients is emphasized.

DISCUSSION

This case illustrates two interesting characteristics of ET. First and foremost is its association with an increased thrombotic tendency (Messinezy and Pearson, 1997). This is thought to be related to both the increased platelet count and reduced physiological blood anticoagulant mechanisms (Landolfi et al, 1997).

This patient had relatively stable chronic obstructive pulmonary disease, but reduced pulmonary blood flow and pulmonary infarction led to worsening respiratory symptoms with a relatively rapid deterioration in exercise tolerance and breathlessness. It is tempting to speculate that infarction in this gentle-

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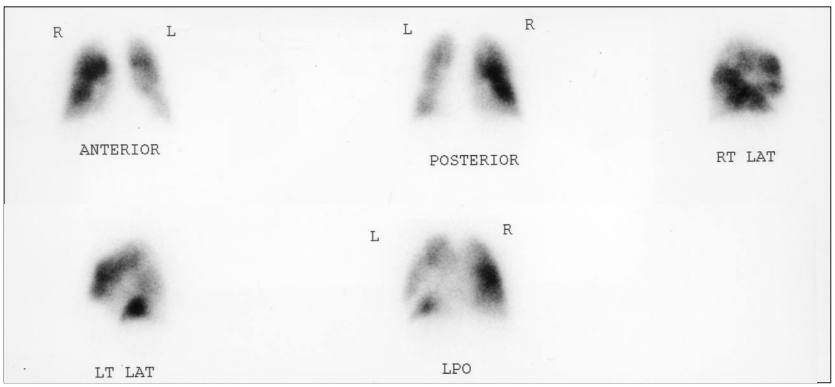

Figure 1. Multiple segmental perfusion defects in patient's ventilation/perfusion lung scan.

man was primarily related to pulmonary vascular thrombosis and occlusion in-situ. Early bone marrow examination to confirm ET (to distinguish this from reactive thrombocytosis) is vital.

Clonogenic assays, cytogenic and clonal analysis are future tools that might help in the earlier diagnosis of this disorder (Harrison et al, 1998). Prompt therapeutic intervention with specific myelosuppressive and cytoreductive therapy and particularly with antiplatelet drugs would then be indicated as delays can be fatal (Randi et al, 1990).

The other learning point in this patient is pseudo-hyperkalaemia. Platelets in ET are abnormal, rather fragile and leak potassium. Promptly rechecking potassium level on a lithium heparin blood sample saved this gentleman from being prescribed unnecessary potassium-lowering treatment. HM

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In the Public's View...

Any cure for blocked tubes?

Bristol, Ledward, Shipman, body parts: I can honestly say that none of these nor their political fallout has had more than minimal direct effect on how my patients respond to me. Now, all of a sudden, my patients are worried.

It's this story about blocked oxygen tubes.

I can talk sensibly about all the other stories. If patients had worried about them — which they didn't as far as I could tell — I could have made encouragingly reassuring noises and given them my views. It surprised me that patients didn't seem to worry about misplaced injections, but again I do understand how they can happen and I can be reassuring.

However, at least as reported by the media, I could not and still don't understand what exactly happened when a 'blocked oxygen tube' killed a boy having surgery. First reports made no sense at all. The only tubes that supply purely oxygen come from the wall into the anaesthetic machine and don't reach the patient at all. Early on in the story the police were informed because of suspected sabotage. Blocking the oxygen pipeline supplies or the piping within the anaesthetic machine requires skilled interference and is glaringly obvious. It seemed unlikely. When the accident was also blamed on tubes that are disposable but re-used to save money, I presumed the 'oxygen tube' to be part of the anaesthetic circuit, the endotracheal tube, or the various bits of connecting plastic. From what I've heard since, the obstructions (now rumoured to be at four different hospitals) have been small plastic caps jammed in a plastic connector.

Such incidents do occur completely by accident. Anaesthesia journals carry case reports of all sorts of things jammed down tubes, including cockroaches (not recommended even when sterilized by autoclave). There are all sorts of unobtrusive small plastic bits — needle cases, protective caps from intravenous sets — lying around in the operating theatres that could easily find their way to where they shouldn't be.

But how could an anaesthetist miss one? If these connectors are blocked, artificial ventilation fails and the pressure alarms go off. The failsafe dictum 'If in doubt, take it out' is central to anaesthesia training, so if all else fails you take out all tubes and pick up the good old mask. By that stage, a blocked connector is obvious — and you can always blow into the patient; I've done that myself once or twice. As so often with media reports, we do not have enough, or the correct, information to know what happened. So I have not been able to give anything like a satisfactory answer to the patients who have asked me about it in the last few days.

The general lesson is that the politicians are wrong (and probably know it) when they tell us how the medical profession has been damaged (to which 'irreparably' is usually attached) by recent medical misfortunes. Patients don't generalize; they particularise. They don't see incidents as symptoms of a deeper malaise, but see each incident as something that might affect them if they were in the same circumstance. In that, as in so much else, the public have more sense than the politicians. HM

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