Discussion

Staphylococcal pneumonia is an uncommon, but serious infection. Despite appropriate antibiotic treatment, mortality in staphylococcal pneumonia remains high.¹⁻³ Staphylococcal pulmonary infections are typically nosocomial and usually occur in older adults (sixth decade or older) with concomitant medical illnesses.¹ Watanakunakorn³ reported the cases of 44 patients with bacteraemic *S aureus* pneumonia (positive sputum and blood culture). All patients had at least one underlying condition (intravenous drug abusers were excluded from the study). Virtually all patients in a study by Kaye *et al*¹ had some underlying illness, particularly chronic pulmonary disease. Our patient was relatively younger, however she did have a history of chronic pulmonary airway disease.

No single radiological presentation is diagnostic of *S aureus* pneumonia. However, multiple cavitation or pneumatoceles are characteristically seen, particularly in children and intravenous drug abusers. These cavities are usually thin walled (2–4 mm) and are slightly indistinct on their outer border. Abscess formation has been reported in 23–70% of adult patients presenting with *S aureus* pulmo-

nary infections.⁴⁻⁶ However, in recent studies,^{1,2} abscess formation was seen in 16–20% of patients and multiple abscesses occurred infrequently. In the study by Kaye *et al*¹ all patients had single abscesses. The chest X-rays in these patients typically show multilobar infiltrates, predominantly in the lower lobes, and often bilateral.^{1,5,6} The frequency of pleural involvement is variable, with estimates ranging from 5% to 48%.^{1,5,6} Our patient had bilateral involvement on presentation and went on to develop multiple abscesses, however, she did not have pleural involvement.

Despite the availability of effective antibiotics, mortality continues to be 30–32%.^{1,2} This may reflect the fact that patients with *S aureus* pneumonia tend to be elderly and have other significant illnesses. Treatment is with a penicillinase-resistant penicillin such as nafcillin, cefazolin or vancomycin (penicillin allergic or methicillin-resistant *S aureus*), and 4–6 weeks of therapy is required to promote cure.

Final diagnosis

Multiple staphylococcal lung abscesses.

Keywords: pneumonia; *Staphylococcus aureus*

1 Kaye MG, Fox MJ, Bartlett JG, Bramn SS, Glassroth JG. The clinical spectrum of staphylococcus aureus pulmonary infection. *Chest* 1990;97:788–92.

2 Woodhead MA, Radvan J, MacFarlane JT. Adult community acquired staphylococcal pneumonia in the antibiotic era: a review of 61 cases. *Q J Med* 1987;64:783–90.

3 Watanakunakorn C. Bacteraemic staphylococcal pneumonia. *Scand J Infect Dis* 1987;19:623–7.

4 Hausmann W, Karlsh AJ. Staphylococcal pneumonia in adults. *BMJ* 1956;2:845–7.

5 Fisher AM, Trever RW, Curtin JA, Schultze G, Miller DF. Staphylococcal pneumonia: a review of 21 cases in adults. *N Engl J Med* 1958;258:919–28.

6 Gold JA, Davis JW. Adult staphylococcal pneumonia. *Med Ann DC* 1959;28:363–5, 417–8.

The chilling tale of a patient with thrombocytopenia

Nicholas R Balcombe

Department of Health
Care for the Elderly,
Queens Hospital,
Burton Hospitals NHS
Trust, Belvedere Rd,
Burton upon Trent,
Staffordshire, DE13
0RB, UK
N R Balcombe

Accepted 12 October 1998

An 86-year-old woman was admitted having been found collapsed at home. On examination, she was pale, dehydrated, confused and disorientated with a rectal temperature of 28°C. There was no lymphadenopathy or bruising. Her pulse was regular at 50 beats/min and blood pressure was 150/100 mmHg. Cardiorespiratory, abdominal and neurological examinations were all unremarkable. Review of this patient's previous hospital notes revealed no significant illnesses in the past and she was on no regular medication. There was no history of excess alcohol intake. Social assessment revealed that she lived alone with inadequate heating provisions. Investigations showed thrombocytopenia, with a platelet count of $88 \times 10^9/l$ (reference range $150\text{--}450 \times 10^9/l$), confirmed by analysis of two venous blood samples. Her haemoglobin was 14 g/dl, white blood count $8.2 \times 10^9/l$, with a normal differential count, and an erythrocyte sedimentation rate of 6 mm in the first hour. Serum urea was 11.1 mmol/l ($3.5\text{--}8$ mmol/l) with a serum creatinine of 127 $\mu\text{mol/l}$ ($50\text{--}105$ $\mu\text{mol/l}$). Serum electrolytes, liver function tests, coagulation studies, electrocardiogram and chest X-ray were all normal, as were vitamin B12 and folate levels. Urine microscopy and culture revealed no evidence of urinary tract infection. Serum thyroid-stimulating hormone was 18.93 mU/l ($0.5\text{--}5.0$ mU/l) and free thyroxine 14 pmol/l ($9\text{--}24$ pmol/l).

Questions

- 1 What is the cause of thrombocytopenia?
- 2 What complications may occur in this patient?

Answers

QUESTION 1

The potential causes of thrombocytopenia are numerous (box 1). In this patient, the cause of thrombocytopenia was hypothermia. Following admission, she was rewarmed and rehydrated and the following day, her resting oral temperature had risen to 34°C and although she remained lethargic, she was no longer confused or disorientated. On day three her resting oral temperature was normal. On day nine, her platelet count had risen to normal ($295 \times 10^9/l$).

QUESTION 2

Apart from the complications of thrombocytopenia, which include skin purpura, mucosal haemorrhage and prolonged bleeding after trauma, hypothermia may lead to other clinical features, shown in box 2.

Discussion

Thrombocytopenia occurring with low body temperature was first described 58 years ago.¹ Since then, animal studies have attempted to identify a cause for this phenomenon.^{2,3} In 1958, Villalobos used radioactively labeled platelets to demonstrate hepatic and splenic sequestration during hypothermia in dogs, with

80% of the platelets returning to the circulation on rewarming.² A later study showed the liver to be the major site of sequestration.³ Disseminated intravascular coagulation and bone marrow failure have also been postulated as causes of hypothermia induced thrombocytopenia.

Platelet sequestration as a result of hypothermia is rare and only a few recorded cases exist.^{4,5} This was thought to be the cause of our patient's thrombocytopenia. She never exhibited any clinical features suggestive of disseminated intravascular coagulation and clotting studies were normal. Her initial full blood count did not show any evidence of bone marrow suppression and the rapid return of the platelet count to normal, mirroring the rise in body temperature, would suggest a causal relationship.

Although our patient experienced no problems as a result of her low platelet count, this case illustrates that thrombocytopenia is a potentially serious complication of hypothermia.

Final diagnosis

Hypothermia-induced thrombocytopenia.

Keywords: hypothermia; thrombocytopenia

Causes of thrombocytopenia

Reduced platelet production

- viral infections: measles, varicella, infectious mononucleosis, congenital rubella, mumps
- drugs: thiazide diuretics
- aplastic anaemia
- leukaemia
- myelodysplasia
- myelosclerosis
- marrow infiltration: carcinoma, lymphoma
- multiple myeloma
- megaloblastic anaemia
- alcohol

Decreased platelet survival

Increased destruction

- auto-immune idiopathic thrombocytopenic purpura
- secondary immune thrombocytopenia: SLE, CLL, lymphoma
- post-transfusion purpura
- drug-induced immune thrombocytopenia: quinine, quinidine, sulphonamides, rifampicin
- heparin

Consumption

- disseminated intravascular coagulation
- thrombotic thrombocytopenic purpura
- haemangioma (Kasabach-Merritt syndrome)

Platelet sequestration

- splenomegaly
- hypothermia

Box 1

Complications of hypothermia

Haematological

- polycythaemia
- isolated thrombocytopenia or leucopenia
- bone marrow suppression
- disseminated intravascular coagulation

Nervous system

- listlessness
- confusion
- amnesia
- loss of consciousness
- motor paralysis
- sensory impairment

Cardiovascular

- initial rise in cardiac output and blood pressure and tachycardia
- later fall in cardiac output and hypotension and bradycardia
- arrhythmias: atrial fibrillation, ventricular fibrillation
- ischaemic necrosis

Respiratory

- depressed respiration
- tissue anoxia
- respiratory acidosis
- pneumonia

Metabolic

- hyperglycaemia
- hyperkalaemia
- renal failure

Gastrointestinal

- gastric erosions
- haemorrhage
- pancreatitis

Box 2

- 1 Tocantins LM. The mammalian blood platelet in health and disease. *Medicine* 1938;17:155.
- 2 Villalobos TJ, Adelson E, Riley PA, Crosby WH. A cause of the thrombocytopenia and leukopenia that occurs in dogs during deep hypothermia. *J Clin Invest* 1958;37:1–7.
- 3 Pina-Cabran JM. Hepatic and splenic platelet sequestration during deep hypothermia in the dog. *Haemostasis* 1974;2:235.
- 4 Vella MA, Jenner C, Betteridge DJ, Jowett NI. Hypothermia induced thrombocytopenia. *J R Soc Med* 1988;81:228–9.
- 5 Chan KM, Beard K. A patient with recurrent hypothermia associated with thrombocytopenia. *Postgrad Med J* 1993;69:227–9.

Hypotension and intractable vomiting in the first trimester of pregnancy

C Usalan, E Özarslan

A 24-year-old woman, gravida 1, para 1, presented at 12 weeks gestation with abdominal pain, weakness, fatigue, nausea and vomiting of several days duration. Ten days earlier she had been admitted to another hospital with the same complaints; she had been diagnosed as having hyperemesis gravidarum and hospitalised for 3 days, after which she was stabilized. Her medical history was unremarkable and she had no family history of relevant illness. On initial evaluation, she was in rather poor clinical condition and was pigmented to an abnormal degree, particularly around the eyes and in the skin creases of the hands; buccal pigmentation was also pronounced. Blood pressure was 100/60 mmHg, pulse 108 beats/min and body temperature was 38.7°C. Physical examination was unremarkable except for dehydration and hypotension. The laboratory examinations are summarised in the table. The results of urinalysis were: specific gravity 1.016, pH 5, 2+ protein, and urine sediment examination revealed a large number of erythrocytes and leukocytes. The urine contained Gram-negative bacteria.

Within two hours, the patient was confused, sweating and tachypnoeic. A repeat blood sugar level was 52 mg/dl. She was given a rapid infusion of intravenous 5% dextrose in 0.9% sodium chloride, but did not improve. Arterial blood gases showed a profound metabolic acidosis. Her acid-base status was as follows: pH 7.12, HCO₃ 6 mmol/l, pO₂ 154 mmHg, pCO₂ 20 mmHg, base excess –22.

Table Initial laboratory findings

Variables	Patient	Normal range
Haemoglobin (g/dl)	14.6	12–18
Mean corpuscular volume (fl)	84	80–100
White cell count ($\times 10^9/l$)	16.6	3.6–10
Differential count (%)		
Neutrophils	78	40–70
Band forms	10	0–5
Lymphocytes	12	20–40
Platelet count ($\times 10^9/l$)	452	150–450
Sedimentation rate (mm/h)	90	0–15
Urea nitrogen (mg/dl)	28	8–23
Creatinine (mg/dl)	1.0	0.6–1.2
Total protein (g/dl)	6.8	6–7.8
Albumin (g/dl)	3.8	3.2–4.8
Uric acid (mg/dl)	6.2	2.7–7.8
Glucose (mg/dl)	62	70–110
Sodium (mmol/l)	124	136–147
Potassium (mmol/l)	5.6	3.5–5.5
Alanine transaminase (IU/l)	28	5–40
Aspartate transaminase (IU/l)	32	8–33

Division of Internal Medicine, Hacettepe University, Ankara, Turkey
C Usalan
E Özarslan

Correspondence to
C Usalan, Sinan Cad. 49/12,
Dikmen, 06450 Ankara,
Turkey

Accepted 18 February 1999

Questions

- 1 What is the differential diagnosis compatible with the history, clinical and laboratory findings?
- 2 Which clues suggest an underlying endocrinologic illness?
- 3 What could be a precipitating factor leading to the aggravation of the underlying illness?