typing was performed and the multiple antibiotic resistance confirmed by the Streptococcus Reference Laboratory, Colindale.

Nose and throat swabs were obtained from all the immediate family—father, mother, a 3 year old brother, a 2 year old sister—and the index patient. The resistant strain was grown from the nose swabs of the index patient and the 3 year old sibling. A sensitive pneumococcus was grown from the nose of the 2 year old, whereas no pneumococci were grown from the parents. The neonate's infected eye was treated for three weeks with topical penicillin (10×10^6 U/l) with clinical and bacteriological cure.

Comment

The family's travel history is of note. Though the father had made business trips to Johannesburg for two weeks in 1976 and again in 1984, of more interest was a two year residence in Spain by the family between 1983 and 1985. In a recent survey of pneumococcal carriage in children aged 4-5 years in Barcelona[4] two of 159 pneumococcal isolates were type 6 and possessed the same multiple resistance pattern as the Bristol isolate. Probably, therefore, the 3 year old sibling had acquired carriage during his time in Spain.

This infection was treatable because of the high concentration of penicillin obtainable at the site of infection with topical application. Systemic infection with this strain would be much more difficult to treat adequately, and treatment with penicillin might well fail. Eradication of carriage of multiply resistant pneumococci would not be easy; using a combination of rifampicin and fusidic acid in Johannesburg had only 65% efficacy.[5] Eradication of carriage was not attempted in our case in view of the mild nature of the infection. Evidence of spread in a susceptible population such as immunocompromised children would require a more aggressive approach. It is to be hoped that such strains do not become more widespread in Britain.

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Acquired factor VIII inhibitor associated with lung abscess

Haemoptysis in chronic bronchitis is common and may be worsened by any generalised bleeding disorder. We report on a patient who developed a lung abscess and was found to have a coagulopathy due to an acquired factor VIIIC inhibitor.

Case report

A 70 year old man with chronic bronchitis was admitted with haemoptysis; he had not noted abnormal bleeding previously. On admission his sputum was bloodstained and he had signs of consolidation of the right middle lobe, which was confirmed by chest radiography. Despite antibiotic treatment cavitation developed in the consolidated lobe; a lung tumour was excluded at bronchoscopy. Extensive superficial bruising and petechias and prolonged bleeding from venepuncture sites were noted.

Platelet count and morphology were normal. Activated partial thromboplastin time was prolonged to 84 s (normal range 21-37 s) and bleeding time to >20 minutes (normal range 2-9 minutes). Prothrombin time, thrombin time, and concentrations of fibrinogen and fibrin degradation products were within the normal range. Von Willebrand factor was 368% (normal range 60-160%); platelet aggregation and von Willebrand factor multimer pattern were normal. The prolonged activated partial prothrombin time was caused by a low factor VIIIC concentration (1%) (normal range 60-160%). His plasma contained a factor VIIIC inhibitor at a concentration of 37 Bethesda units.[1] The inhibitor had no activity against porcine factor VIIIC. He was treated with continuous antibiotics for two months; the haemoptysis resolved in six weeks, his temperature returned to normal in eight weeks, and clearance of the abscess took 23 weeks.

One week after abnormal bleeding was first noted he had an episode of profuse haematuria that required treatment with cryoprecipitate. This increased his factor VIIIC concentration to 10%, which was one fifth of the maximum rise predicted in the absence of a factor VIIIC inhibitor. The factor VIIIC concentration declined with a half life of one hour, compared with the normal half life of infused factor VIIIC of over eight hours. The bleeding time, however, was corrected for 12 hours, and the haematuria stopped immediately after the infusion. Eleven months later the bleeding disorder was not clinically evident and he was well. Factor VIIIC inhibitor was still detectable although much reduced. The table shows changes in the variables associated with clotting.

Changes in variables associated with clotting over the 11 months after illness

No of weeks after admission*	Bleeding time (min)	Activated partial thromboplastin time (s)	Factor VIIIC (%)	Factor VIIIC inhibitor (Bethesda units)
4	>20	84	1	37
8	8	49	5	19
13	8	50	5	3
23	5·5	54	8	12
48	5	49	12	2

*Abscess recognised at week 2; temperature settled at week 8; discharge from hospital at week 13; resolution of abscess by radiography at week 23.

Comment

Acquired inhibitors to factor VIIIC in non-haemophiliacs are rare but may develop in association with autoimmune disorders, malignancy, and pregnancy. Nearly half of all patients have no detectable underlying disorder.[2] In most cases the pattern of spontaneous haemorrhage is similar to that in patients with inherited factor VIIIC deficiency (haemophilia A).[3] The decline in factor VIIIC inhibitor concentration in our patient as the lung abscess resolved suggested an association, although, interestingly, the inhibitor persisted at a lower concentration when he was well.

A prolonged bleeding time is not characteristic of factor VIIIC deficiency even if inhibitors are present: in this our patient's illness resembled von Willebrand's disease, although the antigen structure and activity of von Willebrand factor were normal. This is analagous to the bleeding disorder in renal failure, in which the bleeding time may be corrected with cryoprecipitate,[4] although the plasma concentration of von Willebrand factor is usually already increased.[5] The mechanism whereby cryoprecipitate corrects bleeding time in renal failure is not fully understood, although it may increase the ratio of high to low molecular weight oligomers of von Willebrand factor. This mechanism may have operated in our patient, although as in renal failure the changes in the multimer pattern of von Willebrand factor were too subtle to be picked up by sodium dodecylsulphate electrophoresis.

Although haemoptysis is well recognised in patients with lung abscesses, basic coagulation screening should be carried out to exclude a generalised bleeding disorder. Identification of an associated coagulopathy in patients with other medical problems may substantially alter management.

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