LETTER TO THE EDITOR

Idiopathic thrombocytopenic purpura associated with scarlet fever

EDITOR.—Idiopathic thrombocytopenic purpura (ITP) is common in children and is very often associated with or follows recovery from a viral infection, accounting for about 90% of the paediatric cases of immune thrombocytopenia.1 2 Scarlet fever generally results from a pharyngeal infection with a streptococcal strain that elaborates erythrogenic toxin. Thrombocytopenia is not reported as a complication of scarlet fever.1 3

To the best of our knowledge there are no reports about association between scarlet fever and ITP in children.

Case report
A healthy 5 year old boy whose personal and familial history was unremarkable, was admitted to our department because of high fever and sore throat, associated with red exanthem, lasting for 24 hours. At admission physical examination showed the presence of fever and pharyngeal inflammation associated with satellite lymphadenopathy. There was a diffuse red exanthem with many points of deeper red, blanching on pressure, spreading from the upper part of the chest to the remainder of the trunk, neck and extremities, sparing palms, soles, and perioral area. Moreover, the patient showed a petechial exanthem, not blanching on pressure, involving face, trunk, neck, upper limbs, and oral and pharyngeal mucosa. No spleen or liver enlargement was detected. Blood examination showed: white cell count 17·5 x 10⁹/l (70% neutrophils), platelets 5 x 10⁹/l, haemoglobin 128 g/l and 0·5% reticulocytes. Moreover, there was a slight increase of acute phase reactants (erythrocyte sedimentation rate 34 mm/hour and C reactive protein 33·2 mg/l). Coagulation profile, liver and renal function, and urinalysis were normal. Bone marrow aspirate demonstrated qualitative and quantitative normality of megakaryocytes and erythroid series, with increased megalakocytes showing immature aspect. Pharyngeal swab showed the presence of a group A β haemolytic streptococcus. Serological studies performed before any treatment with the child and his father for antiplatelet IgG (CAPTURE-P). Antibodies against exanthematic viruses, HIV and toxoplasma were negative, antinuclear antibodies were also negative, while serum immunoglobulins, C3 and C4 values were in the normal range. Antibodies against cytomegalovirus and Epstein-Barr virus suggested previous infection.

Antibacterial treatment (oral clarithromycin at 15 mg/kg/day) and intravenous immunoglobulins (200 mg/kg/day) were immediately started. His clinical condition rapidly improved with regression of pharyngeal inflammation and exanthem, without appearance of new haemorrhagic elements. Antibiotics were administered for a total of 10 days and immunoglobulins for five days. After three days of immunoglobulin treatment platelet count was 85 x 10⁹/l and two days after withdrawal of immunoglobulin platelets rose to 165 x 10⁹/l. After antimicrobial treatment the pharyngeal swab was negative, white cell count was 7·7 x 10⁹/l (44% neutrophils), and platelets were 233 x 10⁹/l; haemoglobin and reticulocytes were unchanged from the onset. Renal function and urinalysis were also normal. One month after treatment the patient was in good general condition, white blood cell count, haemoglobin, platelet count, renal function, and urinalysis were within the normal range. Pharyngeal swab was negative. Moreover, antiplatelet and anti-nuclear antibodies were negative.

In this patient, affected with acute streptococcal infection and scarlet fever, thrombocytopenia was typical of acute ITP, with positivity of direct and indirect antiplatelet IgG and negativity for markers of systemic autoimmune disease. Although approximately 90% of children with acute ITP make an uneventful recovery with normalisation of the platelet count within a few weeks to months,1 we administered immunoglobulin because of the high risk of intracranial haemorrhage due to severe thrombocytopenia associated with infection, fever, toxin induced capillary fragility, and polymorphonuclear exudate.4

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Chloral hydrate defended

Chloral hydrate was first synthesised more than 150 years ago and has been used in clinical practice for over 120 years. Trichloroethylene, which is metabolised to chloral hydrate, was shown to cause cancer in rodents after long term administration of high doses and was banned by the American Food and Drug Administration in 1986 and 1992 have shown an increase in liver cancer in mice given chloral hydrate. The September 1993 issue of Pediatrics contains two articles discussing the use of chloral hydrate in children (Alfred D Steinberg, 1993; 92: 442-6, and American Academy of Pediatrics, Committee on Drugs and Committee on Environmental Health 1993; 92: 471-3).

There is no evidence relating the use of chloral hydrate to carcinogenesis in people and both of the articles come out in favour of continuing to use the drug. Newborn babies metabolise chloral hydrate slowly and its prolonged use in the newborn has led to excessive sedation, conjugated hyperbilirubinaemia, and metabolic acidosis. It is therefore suggested that repeated doses is best avoided. The American Academy makes a five point conclusion: (1) chloral hydrate is a useful sedative, (2) repeated doses especially in the newborn are ‘of concern’, (3) data on carcinogenicity do not warrant changing from chloral hydrate to alternative sedatives, (4) there is no evidence that chloral hydrate is less safe than the alternatives and clinicians are better to stick to the sedative with which they are most familiar, and (5) more studies are needed (of course).