Britain and Ireland has recently published guidelines on artificial hydration and cardiopulmonary resuscitation for people who are terminally ill (*European Journal of Palliative Care* 1997;4(4):124, 125, 126-8 (discussion of guidelines)).

Funding: None.

Conflict of interest: None.

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(Accepted 27 February 1997)

Aseptic meningitis associated with high dose immunoglobulin: case report

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Aseptic meningitis is a recognised complication of high dose intravenous immunoglobulin. We report a case of aseptic meningitis diagnosed on the basis of eosinophilia in cerebrospinal fluid.

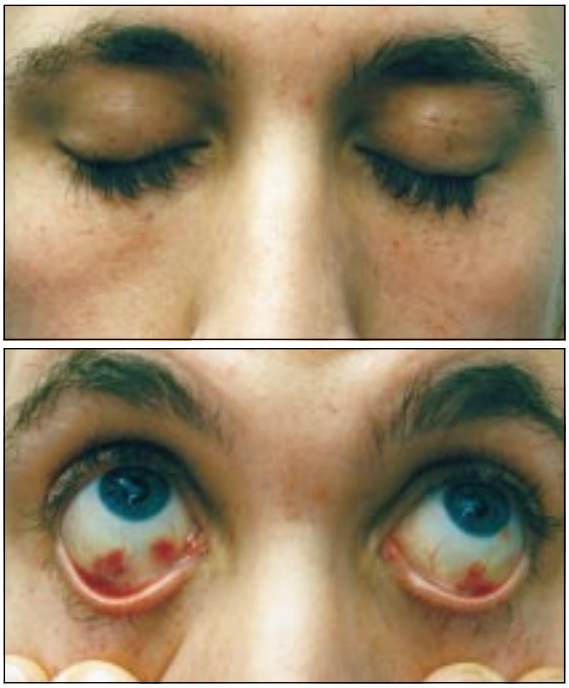
Case report

A 21 year old man with autoimmune thrombocytopenia was admitted with severe headache, photophobia, obvious neck stiffness, and vomiting. Symptoms began after taking high dose intravenous immunoglobulin for two days. This was his first exposure to intravenous immunoglobulin. On day 1 he received 24 g of immunoglobulin. On day 2 he received 60 g but complained of mild headache. He was given paracetamol and allowed home. He was admitted to hospital six hours later with worsening headache.

On examination he was drowsy and had a temperature of 37.4°C, a newly developed extensive purpuric rash, and bilateral subconjunctival haemorrhages (figure). The remainder of the examination gave normal results. The platelet count had not changed from pretreatment values (14×10^9/l), and a coagulation screen gave normal results. Lumbar puncture was delayed because he needed a platelet infusion to cover it, and intravenous cefotaxime was given in the meantime. His cerebrospinal fluid was clear and colourless and contained glucose 3.1 mmol/l (plasma glucose concentration 5.1 mmol/l), protein 0.54 g/l, and immunoglobulin 0.05 g/l. A chamber count showed 80 leucocytes/mm^3; no organisms were seen. Giemsa staining on a spun sample of cerebrospinal fluid revealed many disrupted and some intact eosinophils; a cell count (Cell-Dyn 3500 analyser, Abbott Diagnostics, CA) gave an absolute leucocyte count of 0.06×10^9/l and confirmed these were all eosinophils. The peripheral blood eosinophil count was normal (0.1×10^9/l). These findings excluded acute bacterial meningitis and supported the presence of aseptic meningitis secondary to immunoglobulin infusion. Antibiotic treatment was discontinued, and the patient recovered over the next 24 hours. Blood cultures, cerebrospinal fluid culture, throat swabs, and the polymerase chain reaction for meningococcal DNA all gave negative results.

Comment

High dose intravenous immunoglobulin is used for many conditions.[1] Common side effects include headache, fever, chills, and nausea; these usually resolve within an hour of stopping or slowing the infusion and respond to symptomatic treatment.[2] More serious effects are anaphylaxis, haemolysis, hepatitis, thrombosis, and aseptic meningitis.[3]



Purpuric rash and subconjunctival haemorrhages in patient with aseptic meningitis. Reproduced with patient's permission

Aseptic meningitis after high dose immunoglobulin has been reported in several conditions, including idiopathic thrombocytopenic purpura,[4] chronic inflammatory demyelinating polyneuropathy,[3] and other immune related neuromuscular diseases.[1] In two separate studies the incidence ranged from 11% to 17% of 137 patients. [1 4] At least six immunoglobulin preparations have been implicated.[2] Symptoms often develop after several courses, beginning six to 48 hours after infusion and clearing within three to five days. Corticosteroids are non-protective. Recurrent symptoms usually develop on rechallenge despite varying the rate of infusion, spreading the treatment over more days, or using different immunoglobulin products.[1]

Cerebrospinal fluid analysis commonly shows a leucocyte pleocytosis with raised protein and IgG con-

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BMJ 1997;315:1203–4

centrations. In most reports the pleocytosis has not been examined further by differential count. A mild (3%) cerebrospinal fluid eosinophilia has been documented with immunoglobulin and in aseptic meningitis after other drug treatment.[1 5] In our case cerebrospinal fluid was specifically analysed and stained to provide an accurate differential count. The presence of eosinophilia enabled aseptic meningitis to be diagnosed and antibiotic treatment to be stopped, also avoiding extensive contact tracing.

Funding: None.
Conflict of interest: None.

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(Accepted 20 May 1997)

Dentists' agreement on treatment of asymptomatic impacted third molar teeth: interview study

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BMJ 1997;315:1204

The main indications for removal of a third molar tooth were outlined at a consensus development conference of the National Institutes of Health in 1979.[1] These are (*a*) acute or chronic infection in a third molar tooth, (*b*) damage to adjacent teeth, (*c*) irreparable decay in the tooth, and (*d*) a cyst or space occupying lesion in the tooth. Currently a quarter of third molar teeth are removed without being diseased,[2] and the need for their removal has been questioned.[3] We measured the variation in and reliability of decisions made by a random sample of dentists about the treatment of asymptomatic impacted third molar teeth.

Subjects, methods, and results

We prepared case notes for 25 patients that contained details of the patient's age and sex, a colour intraoral photograph of one asymptomatic lower third molar tooth, and a monochrome glossy print of a radiograph of the lower jaw. All of the patients attended a dentist regularly, and none of them had any coexisting medical or dental conditions to influence the removal or retention of the tooth. The photographs in two cases were poor quality, so only 23 cases were included in the study. A random sample of 90 dentists was selected from the 391 dentists listed by the family health services authorities in two district health authorities in the north west of England. We made an appointment with each dentist to view the case notes and record his or her recommendation. A second assessment was carried out one month later. To prevent dentists from memorising individual cases we asked them if they would repeat the exercise only after they had completed the first assessment. The agreement within each dentist (individual reliability over time) was calculated with the κ statistic. Interexaminer agreement was calculated with multiexaminer κ.[4] Significance was taken as $P < 0.05$.

Seventy four dentists agreed to take part in the study; 16 had left or retired when we tried to contact them. All 74 completed the first and second assessments.

At the first assessment the dentists suggested extraction of 0 to 19 teeth (median 6; mean 7.05 (95% confidence interval 5.91 to 8.19)). At the second assessment they suggested extraction of 0 to 21 teeth (median 6; mean 6.77 (5.62 to 7.92)). Agreement between dentists was fair at the first assessment ($\kappa = 0.22$ (0.21 to 0.23)) and poor at the second ($\kappa = 0.11$(0.10 to 0.11)). The reliability of the dentists' decisions over time varied from excellent ($\kappa = 1.00$) for 10 dentists to extremely poor (negative κ score; worse than chance) for one dentist. For 17 dentists reliability was excellent ($\kappa = 1.0$ to 0.80), for 10 good ($\kappa =$ 0.79 to 0.6), for 22 moderate ($\kappa = 0.59$ to 0.4), for 19 fair ($\kappa = 0.39$ to 0.2), and for 6 poor ($\kappa < 0.2$).

Comment

This study highlights the poor agreement between dentists making decisions on the extraction of asymptomatic lower third molar teeth. Uncertainty in predicting the clinical outcome of leaving an asymptomatic impacted third molar in situ may encourage elective removal.

The surgical removal of teeth is not without risk, especially the risk associated with general anaesthesia. Surgery is also associated with postoperative pain and facial swelling, leading to time lost from work. Up to 6% of patients have paraesthesia of the tongue or lower lip, and 1% have permanent nerve damage.[5]

We suggest that referrals of asymptomatic third molars could be reduced by improved education and the introduction of clinical guidelines.

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(Accepted 8 April 1997)