CASE REPORTS

Crohn’s colitis and idiopathic thrombocytopenic purpura

Michael S Boyne, Kevin R Dye

Abstract
A 17 year old girl with active Crohn’s colitis developed idiopathic thrombocytopenic purpura that was managed with intravenous immune globulins and cyclosporin A. The possible association between Crohn’s disease and immune thrombocytopenia is explored.

Keywords: Crohn’s disease; colitis; thrombocytopenia

Various autoimmune diseases have been associated with inflammatory bowel disease, with the majority of reports describing clustering of autoimmune haemolytic anaemia with ulcerative colitis. An unusual case of Crohn’s colitis with the subsequent development of idiopathic thrombocytopenic purpura (ITP) is described.

Case report
A 17 year old girl with a history of Crohn’s pancolitis without ileal involvement since age 8, was relatively well until the age of 16, when she had several hospitalisations for exacerbations of her Crohn’s disease. This necessitated a medical regimen of prednisone (30 mg/day), azathioprine (100 mg/day), metronidazole, and mesalamine but the symptoms of colitis persisted.

Apart from an anaemia of chronic disease (packed cell volume 0.247), her blood counts were normal until May 1996 when her platelet count was 3 x 10^9/l, haemoglobin 85 g/l, packed cell volume 0.258, and leucocyte count 9.2 x 10^9/l. She was also experiencing epistaxis and bloody diarrhoea. She had no history of recent viral infections, immunisations, recent drug transfusions, or use of recreational drugs. Physical examination revealed only mild cushingoid facies and there was no hepatosplenomegaly. Erythrocyte sedimentation rate was 60 mm/hour. Antinuclear antibodies, anti-HIV, Coombs’ tests, platelet antibodies, anti-HP, white cell differential, partial thromboplastin time, and prothrombin time were unremarkable. A peripheral blood smear showed marked thrombocytopenia with occasional giant platelets and no evidence of microangiopathic haemolytic anaemia. A bone marrow biopsy specimen revealed normocellularity with megakaryocytic hyperplasia compatible with peripheral platelet destruction.

The patient’s medications were discontinued and she was managed initially with methylprednisone (2 mg/kg/day) and platelet transfusions but without effect. She was then treated with intravenous gammaglobulin (1 g/kg/day) which raised her platelet count to 45 x 10^9/l and maintained on cyclosporin A (5 mg/kg/day) and prednisone (60 mg/day) as an outpatient. Her platelet counts remained between 400 and 680 x 10^9/l. A colonoscopy, one month after discharge, revealed no active Crohn’s disease. However, two months later, the return of active colitis prompted the addition of mesalamine to her regimen. One month later, she developed an inflammatory colonic mass necessitating left hemicolectomy. Histology of the mass was consistent with severely active Crohn’s colitis. She remained asymptomatic, with normal platelet counts and did not require immunosuppressive medication.

Discussion
There are multiple case reports in the literature describing the association of inflammatory bowel disease with extraintestinal autoimmune disorders. The actual prevalence of these associations is not known, as no controlled population studies, to date, have been performed. However, in the case-control study by Snook et al, there was a clustering of autoimmune disorders (including primary sclerosing cholangitis) with ulcerative colitis, with a prevalence of 8.2%–10.5%. The study showed little evidence of an association with

Table 1  Reported cases of Crohn’s disease and idiopathic thrombocytopenic purpura (ITP)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age</th>
<th>Sex</th>
<th>Duration between Crohn’s disease and ITP</th>
<th>Location of Crohn’s disease</th>
<th>Response to glucocorticoids</th>
<th>Platelet antibodies</th>
<th>Treatment of ITP</th>
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</thead>
<tbody>
<tr>
<td>4</td>
<td>65</td>
<td>F</td>
<td>(+) 28 years</td>
<td>Colon</td>
<td>Transient</td>
<td>Yes</td>
<td>Splenectomy, colectomy</td>
</tr>
<tr>
<td>5</td>
<td>19</td>
<td>F</td>
<td>On presentation</td>
<td>Colon, distal ileum</td>
<td>Transient</td>
<td>No</td>
<td>Splenectomy, LTF</td>
</tr>
<tr>
<td>6</td>
<td>43</td>
<td>M</td>
<td>(~) 2 months</td>
<td>Colon</td>
<td>Yes</td>
<td>No</td>
<td>Glucocorticoids</td>
</tr>
<tr>
<td>7</td>
<td>54</td>
<td>M</td>
<td>(~) 7 months</td>
<td>Colon, distal ileum</td>
<td>Yes</td>
<td>Yes</td>
<td>Glucocorticoids</td>
</tr>
<tr>
<td>8</td>
<td>22</td>
<td>F</td>
<td>(+) 6 weeks</td>
<td>Colon</td>
<td>Yes</td>
<td>No</td>
<td>Glucocorticoids, 6MP</td>
</tr>
<tr>
<td>Present case</td>
<td>17</td>
<td>F</td>
<td>(~) 9 years</td>
<td>Colon</td>
<td>Yes</td>
<td>No</td>
<td>IV IgG</td>
</tr>
</tbody>
</table>

Key: 6MP = 6-mercaptopurine; LTF = lost to follow-up; (+) = Crohn’s disease occurred before ITP; (~) = ITP occurred before Crohn’s disease; IV IgG = intravenous gammaglobulins.
Crohn’s disease, irrespective of the extent of colonic involvement.

The association between ITP and ulcerative colitis has been well described, with an estimated prevalence of 0.1%–0.48%. However, there are only five other reported cases of patients with Crohn’s disease and ITP. The most notable common factor is the extensive colonic involvement in all the cases with active colitis being present during the development of the thrombocytopenia. The resolution of ITP, only after colectomy in the case by Kosmo et al, also lends some credence to the idea of the colon being involved in the pathogenesis. Hypothetically, during severe colitis, antigens of the colonic lumen (such as bacterial antigens) could generate a humoral response, leading to antibodies that cross react with platelet surface antigens, thus resulting in ITP. An alternative mechanism could be that severe colonic inflammation leads to local destruction and/or sequestration of platelets in the colonic vascular lumen, which could result in the absence of circulating platelet antibodies and resistance to glucocorticoids. Of course, one cannot totally dismiss a fortuitous association between Crohn’s colitis and a self-limiting ITP in these cases.

This patient, notably, despite having Crohn’s colitis for nine years, did not develop ITP until she had severe flaring of her colitis, raising the possibility of a temporal association. Also, her ITP developed while on glucocorticoids, and once established, the ITP remained steroid resistant. This steroid resistance was also noted in two of the other cases and has occurred in some cases of ITP associated with ulcerative colitis. The possibility of a drug induced mechanism seems unlikely. Azathioprine, in one series, caused the sudden onset of isolated thrombocytopenia in 1.4% of treated patients with Crohn’s disease, but the mechanism is related to bone marrow suppression and there are no reported cases of an immunological mechanism. Unlike sulfasalazine, which can cause immunological destruction of platelets, mesalamine causes thrombocytopenia through non-immunological mechanisms, that is, bone marrow suppression with hypocellularity. Also, this patient was rechallenged with mesalazine without recurrence of thrombocytopenia. There are no reported cases of ITP associated with metronidazole.

The patient was treated with immune globulins, which stimulated partial recovery of her platelet counts, and then continued on maintenance therapy with cyclosporin A in an attempt to maintain her platelet counts and induce remission of the Crohn’s colitis. The cyclosporin did induce remission of her colitis as shown by colonoscopy, but this effect was not sustainable, which is consistent with previous observations of similar low dose regimens.

Partial colectomy ultimately enabled treatment of the underlying Crohn’s colitis, discontinuation of the cyclosporin and there was no recurrence of thrombocytopenia. In the cases of ulcerative colitis and ITP, the thrombocytopenia resolved with glucocorticoids or immune globulins, although it was necessary to perform splenectomies in the steroid resistant patients. In three cases of Crohn’s colitis, glucocorticoids were therapeutic, but the other cases had only transient responses. These cases did not respond to splenectomy but one eventually responded to colectomy. Consequently, it would seem reasonable that Crohn’s associated ITP should be treated first with glucocorticoids and in resistant or severe cases immune globulins may be tried, which is similar to recognised consensus guidelines on the management of ITP. The efficacy of cyclosporin is questionable in this case and cannot be recommended at present. The present data in refractory cases seem to indicate that splenectomy may not be a successful therapeutic modality, but it is too early to routinely recommend colectomy as an alternative.

Learning points
- Idiopathic thrombocytopenic purpura can be an extraintestinal autoimmune manifestation of ulcerative colitis and less commonly, Crohn’s colitis.
- Medications used to control Crohn’s disease may cause drug induced thrombocytopenia and should be excluded as a potential aetiology of the thrombocytopenia.
- Crohn’s associated thrombocytopenia should be managed like other cases of ITP, but high dose glucocorticoids may not be effective.

Osteomyelitis and possible endocarditis secondary to *Lactococcus garvieae*: a first case report

P Rachael James, Suzanna M C Hardman, David L H Patterson

Abstract
Although osteomyelitis is commonly caused by staphylococcal infection, the first case of a lumbar osteomyelitis secondary to *Lactococcus garvieae* is reported. The case was complicated by possible endocarditis of an aortic valve prosthesis.

Keywords: *Lactococcus garvieae*, osteomyelitis

Lactococci are often believed to be of low virulence. We report a first case of osteomyelitis secondary to *Lactococcus garvieae* in a previously well, middle aged woman.

Case report
A 56 year old woman was referred to a rheumatology clinic with a nine week history of lower back pain and a five week history of rigors and night sweats. She was anorexic and had lost approximately 3.5 kg in weight in six weeks. Systems review was unremarkable other than tenderness over L5/S1 and a soft early diastolic murmur at the left sternal edge in keeping with the aortic regurgitation. Results of routine tests were as follows: haemoglobin 99 g/l (mean corpuscular volume 92 fl), white cell count 6.1 × 10^9/l, erythrocyte sedimentation rate 74 mm/hour, and C reactive protein 12.6 mg/l; urea, electrolytes, creatinine, liver function tests, and a bone profile were all normal. The urine culture was negative, the chest radiography was normal, an electrocardiogram showed sinus rhythm with a normal axis and deep T wave inversion in the inferolateral territory. This was unchanged from earlier electrocardiograms (previous history of aortic stenosis). Thoracic and lumbar spine radiographs showed a thoracolumbar scoliosis with loss of disc height at L2/L3 and subchondral bone loss. A bone scan revealed increased tracer activity in the mid-lumbar region (fig 1).

Three days later she was admitted with increasing back pain and spiking temperatures. On day 6 after admission, pale splinter haemorrhages were noted in several nails of her hands and toes, not previously documented. There was no splenomegaly or microscopic haematuria and there was no other evidence of embolic phenomena. The murmur of aortic regurgitation was unchanged. A transthoracic echocardiogram revealed a well seated valve replacement in the aortic position, with thin mobile leaflets and mild to moderate transvalvular aortic regurgitation. Other valves appeared normal and no vegetations were identified. In view of the high suspicion of infective endocarditis, a transoesophageal echocardiogram was undertaken. No vegetations or changes consistent with an aortic root abscess were identified. The aortic regurgitation remained unchanged in severity and no further splinter haemorrhages subsequently developed.

All blood cultures, in addition to the biopsied bone, grew Gram positive cocci growing in chains on blood agar. They were identified as *Lactococcus garvieae* and were found to be indistinguishable from one another by API Strep (bioMérieux, Basingstoke, Hants, UK). Sensitivity testing and their identity was confirmed by the Streptococcal Reference Laboratory (Respiratory and Systemic Infection Laboratory, London, UK). Vancomycin had been started after the bone biopsy, which was subsequently replaced by teicoplanin to which the organism was sensitive. With antibiotic treatment her clinical course improved and her remaining admission was uneventful. She was discharged after one month of intravenous treatment, with a temperature chart, to complete a further two months of teicoplanin at home via a Hickman line. She has remained well and continues under active follow up.

![Figure 1: Bone scan showing increased tracer uptake in mid-lumbar region.](Image)
This is the first report of osteomyelitis secondary to \textit{L. garvieae}, an emerging zoonotic pathogen.\cite{1} Although first isolated from bovine sources, mainly involving cases of mastitis,\cite{2} \textit{L. garvieae} has subsequently been isolated from both fish and humans.\cite{3} Lactococci are facultatively anaerobic, catalase negative, Gram positive cocci that occur singly, in pairs, or in chains. They are most often confused with enterococcus species but can be differentiated by biochemical tests.\cite{4} In contrast to streptococci and enterococci, lactococci are unusual pathogens and are considered to be opportunistic and of low virulence in humans. They have previously been recovered from urine and blood.\cite{5} We are unaware, however, of any previous reports of osteomyelitis secondary to \textit{L. garvieae}, a condition where \textit{Staphylococcus aureus} remains the commonest causative organism.

The complicating issue in this case was the finding of splinter haemorrhages in the presence of bacteraemia and a prosthetic heart valve. There should be a high index of suspicion for infective endocarditis in the presence of heart valve replacements, but there was no evidence on transthoracic or transoesophageal echocardiography of valve prosthesis or aortic root infection. The aortic regurgitation had previously been noted and was transvalvular rather than paraprosthetic. Using the criteria published by the Duke endocarditis service, the diagnosis of endocarditis in this case remains possible (see boxes 1 and 2).\cite{6} This method for assessing the likelihood of infectious endocarditis employs clinical, microbiological, serological, and echocardiographic parameters as major and minor criteria rather similar to the Jones criteria used in the diagnosis of acute rheumatic fever. This yields a high specificity and hence a low chance of a false negative result.\cite{7}

We should like to acknowledge the help of Dr Andrew Mackay MRCP, MRCPath, MA, MSc, Consultant Microbiologist and Lead Clinician in Pathology, Microbiology Department, Greenwich District Hospital, Vanbrugh Hill, London for his assistance with this report.
Phaeochromocytoma unearthed by fluoxetine

A S Kashyap

Abstract
Non-specific noradrenaline reuptake inhibition by high dose selective serotonin reuptake inhibitors, along with catecholamine release from phaeochromocytoma, may lead to a hypertensive paroxysm. This may unmask a clinically silent phaeochromocytoma. Hypertensive paroxysm induced by paroxetine leading to detection of phaeochromocytoma has been reported. The first patient in whom fluoxetine unmasked a phaeochromocytoma is reported.

Keywords: selective serotonin reuptake inhibitor; fluoxetine; phaeochromocytoma

Case report
A 29 year old man was prescribed fluoxetine (selective serotonin reuptake inhibitor) 20 mg/day for depression. In view of his poor response the fluoxetine dose was doubled to 40 mg/day. A few days later he presented with paroxysmal attacks of palpitations, nausea, headache, pallor, perspiration, and headache. His other medication was diazepam. On examination the patient was agitated, apprehensive, and had marked peripheral vasoconstriction. His blood pressure varied between 250/140 and 80/30 mm Hg. There was no postural hypotension. Phaeochromocytoma was considered in view of paroxysmal nature of symptoms and fluctuations of blood pressure. Blood pressure recorded earlier was normal. Twenty four hour excretion of noradrenaline was 10.3 nmol/day (normal range 0.06–0.47), adrenaline 32 nmol/day (normal <0.016), and vanillylmandelic acid 134 nmol/day (normal <30).

Magnetic resonance imaging of the abdomen for a suspected phaeochromocytoma revealed a 3 cm diameter mass in the right adrenal gland. After surgical removal of the right adrenal gland, histological examination confirmed the presence of a phaeochromocytoma. The patient has been normotensive since then, and has no symptoms.

Discussion
Tricyclic antidepressant drugs lead to increased concentrations of noradrenaline due to inhibited presynaptic reuptake of noradrenaline. Although this effect is beneficial, it can lead to haemodynamic abnormalities, particularly when used with monoamine oxidase inhibitors or in a patient with phaeochromocytoma.1 Similar haemodynamic effects are seen when selective serotonin reuptake inhibitors are used along with a monoamine oxidase inhibitor.2

A difference in selectivity has been seen in animal studies between various serotonin reuptake inhibitors, regarding dose dependent inhibition of noradrenaline uptake. In humans, such inhibition in therapeutic dosage has not been shown.3

Activity of cytochrome P-450 (CYP2D6) determines the rate of fluoxetine metabolism; this differs between poor and prolific metaboliser patients.4 Non-specific noradrenaline reuptake inhibition by high dose serotonin reuptake inhibitors, along with catecholamine release from phaeochromocytoma, may explain the haemodynamic abnormalities seen in this patient. Inhibition of serotonin reuptake by platelets may result in increased plasma serotonin concentrations. This in turn leads to higher sensitivity of noradrenaline receptor, thus providing an alternative explanation for paroxysms of haemodynamic abnormalities.5

Paroxetine has been implicated in such adverse effects in a patient with phaeochromocytoma.6 There is no report of fluoxetine leading to such effects until now. Clinicians should suspect phaeochromocytoma or drug interactions when hypertension is detected during treatment with selective serotonin reuptake inhibitors.
Reversible sensorineural hearing loss after non-otological surgery under general anaesthetic

H Pau, D Selvadurai, G E Murty

Abstract

Acute sensorineural hearing loss can occur after both otolaryngological and non-otolaryngological procedures. The mechanism of such hearing loss remains unproved; but nitrous oxide has been implicated and where used, attendants should be aware of its potential damage to hearing. It is essential that patients with sudden hearing loss are identified as soon as possible as the recovery rate had been shown to be higher in those who presented early; and as our case demonstrates, complete recovery is possible. Anaesthetists, non-otolaryngological surgeons, and ward nurses must be aware of this early postoperative complication if appropriate treatment is to be instituted.


Keywords: surgery; sensorineural hearing loss

Cases of sudden sensorineural hearing loss after non-otological surgical procedures are rare. Most of these were surgical complications after cardiopulmonary bypass, and to date there have been only 29 cases (table 1) of sensorineural hearing loss after non-otological and non-cardiopulmonary surgery. We present a patient who suffered unilateral sensorineural hearing loss after varicose vein surgery under general anaesthetic.

Case report

A 29 year old male smoker presented to the ear, nose and throat department two days after undergoing long saphenous vein stripping and avulsions under general anaesthetic. The general anaesthetic involved induction with intravenous propofol, droperidol, fentanyl, and atropine. Ventilation was via a laryngeal mask airway size 4. Maintenance was achieved by spontaneous ventilation with a gas mixture of 40% oxygen in nitrous oxide and isoflurane vapour. Postoperative pain was controlled by a diclofenac suppository and by infiltrating a solution of 0.5% bupivacaine with adrenaline (epinephrine) to the wounds.

On awakening, he complained of sudden deafness in the right ear and described right sided tinnitus. He was otherwise healthy with no significant past medical history and on no medications. On physical examination he was afebrile; the cardiovascular system was normal with no carotid bruits and examination of the remaining cranial nerves and peripheral nervous system revealed no abnormalities. The appearance of the pinna, the external acoustic meati, and the tympanic membrane was unremarkable. Rinne’s test produced a false negative in the right ear and Weber’s test lateralised to the left. A pure tone audiogram showed profound sensorineural hearing deficit in the right ear (fig 1). A full blood count, plasma viscosity, clinical chemistry, and autoantibody screen were normal and no abnormalities were detected.

On examination he had painless, tender, red and swollen areas in the region of the cutaneous blood supply posterolateral to the external ear. There were no signs of infection such as cellulitis or lymphadenopathy. The patient was diaphoretic and his pulse rate was 94 and his blood pressure 150/90mm Hg. His temperature was 37.4°C and his respiratory rate was 22 per minute. He had a significant amount of bleeding and oozing from the incisions of both thighs. He was in pain and refused to give his medications. On physical examination he was otherwise healthy with no significant past medical history and on no medications.

Table 1 Twenty nine cases of sensorineural hearing loss after general anaesthetic after distant site surgery and non-cardiopulmonary surgery

<table>
<thead>
<tr>
<th>Study</th>
<th>No</th>
<th>Surgery</th>
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<th>Hearing improved ?</th>
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<tr>
<td>Patterson and Barlett (1979)</td>
<td>1</td>
<td>Bunionectomy and arthroplasty</td>
<td>Yes</td>
<td>Yes</td>
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<td>Pyloroplasty and vagotomy</td>
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<td>Hockerman and Reimer (1989)</td>
<td>3</td>
<td>Rectal prolapse</td>
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<td>Yes</td>
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<td>Adrenalectomy</td>
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<td>Mastectomy</td>
<td>N/A</td>
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<td>Pupilloplasty</td>
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<td>N/A</td>
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<td>Jaffe (1967)</td>
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<td>Thyroidectomy</td>
<td>N/A</td>
<td>N/A</td>
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<td>9</td>
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<td>10</td>
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<td>Inguinal hernia</td>
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<td>Lumbar discectomy</td>
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<td>N/A</td>
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<td>16</td>
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<td>Yes</td>
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<td>De la Cruz et al (1998)</td>
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<td>Appendicectomy</td>
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<td>Present case (1999)</td>
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<td>Varicose vein surgery</td>
<td>Yes</td>
<td>Yes</td>
</tr>
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</table>

N/A = not available.
were normal. Magnetic resonance imaging of the posterior cranial fossa showed no evidence of an acoustic neuroma.

He was admitted for regular carbogen (a mixture of 95% oxygen and 5% carbon dioxide) inhalation hourly for the first 24 hours, intravenous infusion of Dextran 70 solution at a rate of one litre every 12 hours for the first four days, oral flucloxacillin 250 mg four times daily for five days, and oral prednisolone treatment, starting with 60 mg once daily which was later reduced gradually and eventually stopped on the 11th day. Pure tone audiograms were repeated at daily intervals and gradually returned to normal by day 5. He was then discharged from the ward and his follow up pure tone audiogram at six weeks was normal.

Discussion
Box 1 shows a list of some common causes of acute sensorineural hearing loss. However in some cases, the aetiology remains unknown.1 Idiopathic acute sensorineural hearing loss has been reported to have an incidence of between five to 20 new cases per 100 000 population per year.2 Several theories including viral, autoimmune, vascular, embolism, and inner ear membrane rupture have been postulated.

Sensorineural hearing loss has been reported in several specialties after surgery: (1) local surgery including neuro-otological and dental; (2) cardiopulmonary; and (3) distant site surgery including ophthalmic, gastrointestinal, gynaecological, urological, orthopaedic, and endocrine (table 1).

In neuro-otological surgery, local trauma in acoustic neuroma resection can lead to sudden sensorineural hearing loss.3 In dental procedures, local factors including opening the jaw widely, and the noise intensity and duration of drilling were thought to be responsible.2 There have also been a number of cases of sudden hearing loss after cardiopulmonary bypass surgery.4–8 Twenty nine cases of sudden sensorineural hearing loss after distant site surgery including our case are listed in table 1. There were five orthopaedic, one ophthalmic, one cardiac pacemaker implantation, 13 general surgical, one nasal, one urological, three endocrine, and three gynaecological operations. The type of operation in one case was not recorded. Nitrous oxide was administered in 17 of the cases but information regarding the length of the operations were not available. Hearing

Box 1: Common causes of acute sensorineural hearing loss
- Trauma (direct, surgical, barotrauma, or noise)
- Ototoxic drugs
- Acoustic neuromas
- Meniere’s disease
- Multiple sclerosis

Figure 1 Pure tone audiogram showing profound sensorineural hearing deficit in the right ear.

<table>
<thead>
<tr>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air conduction true threshold</td>
<td>○ X</td>
</tr>
<tr>
<td>Air conduction shadow of opposite ear</td>
<td>● ●</td>
</tr>
<tr>
<td>Air conduction masked but the same</td>
<td>○ ○</td>
</tr>
<tr>
<td>Unmasked bone conduction</td>
<td>△ △</td>
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<tr>
<td>Masked bone conduction</td>
<td>○ ○</td>
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<tr>
<td>Loudness discomfort levels</td>
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</table>
improvements were noted in 10 patients, but whether complete reversibility was achieved in these patients was not clearly demonstrated.

The type of anaesthetic has been postulated to have a causative role in these patients. It is well known that nitrous oxide can affect middle ear pressures \(^1\) and sensorineural hearing loss can be caused by a formation of a perilymph fistula secondary to a ruptured round window. Goodhill \(^2\) and Goodhill et al. \(^3\) postulated an “implosive” and an “explosive” route for rupture of the round window membrane. The former creates rupture by increased pressure in the middle ear and the latter by an increase in the cerebrospinal fluid. Segal et al. \(^4\) stated that a rupture of the round window by the “implosive” route can take place during induction of anaesthesia with nitrous oxide by outward movement of the tympanic membrane and thus the stapes. \(^5\) However, four of the cases shown in table 1 certainly did not receive nitrous oxide during their operations. The other anaesthetic agents used may act indirectly on the auditory system by altering the general haemodynamics—for example, isoflurane will increase intracranial pressure, propofol at therapeutic dose increases cerebral vascular resistance by 50% and decreases systolic blood pressure by 20%–30%, and fentanyl causes resistance by 50% and decreases systolic blood pressure.

The modification of this. Dextran 70 has been used by Fisch, \(^6\) and inhaled carbogen to improve the oxygenating loss is largely empirical due to the number of potential causes. The use of systemic steroids, for example, prednisolone, to combat the inflammatory effect of any viral infection and inhaled carbogen to improve the oxygen tension in the perilymph has been advocated by Fisch, \(^7\) and the management of our case was a modification of this. Dextran 70 has been used as a plasma expander to improve the cochlear circulation. \(^8\) In our case, fluocoxacinil was given prophylactically against possible secondary bacterial labyrinthitis. However it is difficult to judge whether a suggested treatment would result in a higher recovery rate than a spontaneous recovery. Wilkins et al. showed that there was no statistically significant difference between the patients who received treatment and those who did not. \(^9\) Further evaluation of our current methods of treatment is important.

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**Learning points**

- Acute sensorineural hearing loss can occur after both otolaryngological and non-otolaryngological procedures.
- Patients with sudden hearing loss postoperatively should be identified as soon as possible as the recovery rate had been shown to be higher in those who presented early.

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