Sudden Sensorineural Deafness after Unilateral Total Knee Replacement
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Citation

Abstract
We present a report of an individual who sustained a sudden onset of sensorineural hearing loss following a unilateral total knee replacement. Phillips et al reported a case in 2003 from the same hospital, following bilateral simultaneous knee replacements. It was previously concluded that the most probable cause of the hearing loss was fat embolism. This is the first recorded case of a sudden bilateral hearing loss following a unilateral knee replacement. We conclude that a prospective investigation of the incidence of hearing loss after joint replacement is may be warranted.

INTRODUCTION
Few studies exist of hearing loss after joint replacement. We present a case of a unilateral sensorineural loss after total knee replacement, review the literature and discuss the likely aetiology.

CASE REPORT
An 80 year old farmer presented with a severe left sided hearing loss 4 weeks after undergoing a left total knee replacement for osteoarthritis. He was otherwise well, with the exception of essential hypertension. This was well controlled by amlodipine and bendroflumethazide. He took 7.5mg of meloxicam bd as required for his osteoarthritic pain. He had no prior history of otological surgical procedures. He had noted no problem with the hearing in either ear pre-operatively.

He received routine peri-operative thromboprophylaxis with subcutaneous enoxiparin. Combined spinal-epidural anaesthesia was planned, but the epidural catheter could not be sited. He therefore had a spinal anaesthetic using 20mcg of fentanyl and 2.2mls of 0.5% Marcain (R) heavy (Bupivacaine hydrochloride 0.5% and Glucose monohydrate 80mg/ml). A 16 gauge Tuohy needle and 27 gauge spinal needle were used.

Surgery to site a ‘Genesis II’ total knee replacement (Smith and Nephew) went smoothly.

His blood pressure remained stable throughout the procedure. Approximately one litre of blood was collected in the first 6 hours post operatively, using a Bellovac® ABT drain (AstraTech AB, Mölndal, Sweden). 300mls was replaced as an autologous transfusion. He received 2 litres of Hartman’s solution and 500mls of Gelofusine®, intra and immediately post-operatively, followed by a further litre of Hartman’s solution overnight. A morphine patient controlled analgesia system was used.

On the first post operative night he was noted to have a slightly irregular pulse. No cardiovascular compromise was noted; his pulse, blood pressure and oxygen saturations remained stable. An electrocardiogram showed occasional ventricular ectopics. He was a little disorientated at night, but otherwise made a good postoperative recovery. He first noted that his hearing had deteriorated on the second post operative day, but this was not investigated during his inpatient stay.

He was reviewed in the ENT outpatient clinic three weeks after hospital discharge. Otoscopy revealed normal tympanic membranes. Audiometry revealed a bilateral sensorineural hearing loss, more marked on the left (Figure 1). Repeat audiometry five months later showed no improvement in hearing thresholds.

He was fitted with bilateral digital hearing aids, and was very happy with the benefit these provided.
DISCUSSION

The patient reported no previous hearing problems; post operatively he was sure that the hearing in his left ear had become markedly worse. At 80 years old a degree of pre-existing hearing loss might be expected due to presbyacusis.(2) We feel his right audiogram probably is representative of his pre operative hearing. There was no pre-operative audiogram for comparison. The post operative loss appears to be most marked in the lower frequencies.

We could find no studies of the incidence of hearing loss after joint replacement. The only previous case report of sudden hearing loss after knee replacement was at the senior author’s institution in 2003.(1) Those individuals who suffer a lesser degree of hearing loss may not present immediately to otolaryngology or audiology, and the association of hearing loss with previous joint surgery may not be as apparent. It may hence be under diagnosed and under reported.

In the previously reported case of deafness after knee replacement, it was concluded fat microemboli occluding the delicate cochlea circulation was the most likely aetiology.(1) Since this report, a histological examination of right atrial and arterial blood samples taken during knee replacement surgery intra-operatively has shown microscopic fat emboli in 50% of cases.(3) Animal studies of fat emboli suggest that small (15-20µm) emboli may traverse the lung vasculature.(4)

The internal auditory artery (labyrinthine) artery supplies both the cochlea and labyrinth and has a diameter of 200 to 300µm.(5) The cochlea is supplied by the common cochlea artery, an end artery, with poor co-lateral circulation.(6, 7) The modiolar branch supplies the apex of the cochlea and a vestibulocochlear branch supplies the base of the cochlea, posterior semicircular canal and sacculle. The modiolar artery has a diameter of approximately 60 µm.(8) It has smaller branches that supply the stria vascularis.(9) Obstruction of these vessels by fat micro-emboli could lead to low frequency hearing loss without vertigo. An analogous situation occurs in the retinal vasculature, where the end effects are more open to clinical examination via fundoscopy. Intra-arterial fat globules and retinal haemorrhages can be seen. They are found in approximately 50% of individuals with fat embolism syndrome (FES).(10) The patient in our case did not undergo fundoscopy, which can often help clinch a clinical diagnosis of fat embolism.

FES is caused by more extensive fat emboli. It is classically characterised by a petechial rash, cardiopulmonary compromise and neurological dysfunction. It is a diagnosis of exclusion with a differential diagnosis including myocardial infarction, pulmonary embolism, cardiogenic shock, sepsis, transient ischaemic attack, cerebrovascular accident and metabolic disturbance.(11-13) The incidence after major orthopaedic procedures or long bone fractures varies from 11% to 29%.(12-15)

Autologous transfusion is now common practice after joint replacement surgery.(16) The collected blood carries particulate debris and high levels of pro-inflammatory cytokines. A 40µm filter was used in our case to remove debris from the blood, but this would still leave microemboli of a size that could compromise the cochlea microcirculation.

Interleukin-1 Beta (IL-1β) is present in autologous blood, at levels 26 times higher than normal blood.(17) Interestingly, high levels of IL-1β causes sensorineural deafness in Muckle-Wells syndrome, with reports of reversal of the loss using anakinra (an IL-1β antagonist) therapy.(18, 19)

Spinal anaesthesia was used in this case, following an abandoned attempt to site an epidural catheter for a combined spinal-epidural.

Communication exists between the CSF and the perilymph via the cochlear aqueduct. A decrease in cerebrospinal fluid (CSF) pressure can, in theory, result in decreased perilymphatic pressure, relative endolymphatic hydrops, and hearing loss that is usually low frequency and temporary.(20) A study of hearing thresholds after neurologic surgery and non-neurologic procedures involving spinal anaesthesia showed the degree of hearing loss correlated with the amount of CSF lost.(20) Epidural anaesthesia carries a small (less than 1%) risk of dural puncture, hearing loss is very rare, but there are case reports
CONCLUSIONS

This is the first case report of sudden, permanent, unilateral deafness following a unilateral total knee replacement. We believe this condition may be more common than reports suggest, as we have seen a similar presentation relatively recently at the same institution.

We feel the most likely aetiology was again fat embolism. Other factors that may have played a role include spinal anaesthesia and autologous transfusion.

Fundoscopy may help in the diagnosis of otherwise subclinical fat emboli.

A future study might compare pre and post operative hearing thresholds, in those that have joint replacements with and without spinal anaesthesia. The histological examination of arterial blood samples for emboli, fundoscopic examination, and IL-1β levels may be areas for further investigation.

References

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