

Resolution of Pseudotumor Cerebri following surgery for morbid obesity

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ABSTRACT

The cause of pseudotumor cerebri (PTC) is poorly understood although there is strong evidence that obesity plays a role in its development. This report describes a patient with medically intractable PTC, who had continued symptoms despite undergoing a ventriculo-peritoneal (VP) shunt. Following significant weight loss, as a result of laparoscopic gastric banding, she has been symptom free and off all medications for 11 months allowing VP shunt removal. Bariatric surgery should be strongly considered in morbidly obese patients with PTC.

INTRODUCTION

Idiopathic Intracranial Hypertension, or pseudotumor cerebri (PTC), is a condition characterised by raised intracranial pressure not attributable to mass lesions, focal structural abnormalities or CSF flow in the orientated and alert patient. It presents most commonly with headaches, visual disturbances and tinnitus. It is a recognised complication of a diverse array of underlying medical conditions including morbid obesity, attributed to raised intra-thoracic pressure, itself secondary to raised intra-abdominal pressure (1-3). The most common surgical approach to PTC is ventriculo- or lumbo-peritoneal shunting. These techniques have high failure rates in the obese, however, hypothesised to be due to the high intra-abdominal pressure in these patients limiting the pressure gradient of the shunt (4).

Previous studies have demonstrated the role of bariatric surgery in the treatment of PTC, by the reduction of symptoms. This case report demonstrates the potential role of bariatric surgery in reversing PTC to such an extent that a VP shunt can be removed, thus providing an alternative to shunting as the surgical treatment of choice for PTC in the morbidly obese patient (5).

CASE REPORT

A 28-year-old lady, mother of two and a social work student undertaking her thesis, presented as an emergency to hospital, complaining of a bi-frontal headache, worsening over the last three months with associated nausea. Visual field disturbances had been noted for the previous two weeks. On the day of admission the worsening headache had been associated with vomiting.

In hospital, she described the visual disturbances as a blurring in the right upper quadrant of her visual field. She denied obscurations or diplopia. She used no regular medications, was an ex-smoker and drank alcohol in moderation.

On examination, she remained alert and orientated with a GCS of 15, she wore glasses and was clinically obese. The vision in her right eye displayed shimmering in her lateral visual field, and in her left there was clouding in the medial visual field. Visual acuity was 6/6 in the right and 6/9 in the left, correctable with glasses. Visual field analysis noted bilateral enlarged blindspots. Ophthalmoscopy revealed bilateral papilloedema. Other cranial nerve examination was normal. Peripheral neurology was similarly unremarkable.

A lumbar puncture (LP) was performed, showing an opening pressure of 40 cmsH₂O, and closing pressure of 14 cmsH₂O. Other investigations demonstrated: a CSF glucose of 3.8mmol/L (Blood glucose 5.4mmol/L), protein 0.016g/dL and no RBC or WBC on microscopy. A diagnosis of idiopathic intracranial hypertension was made and, after the therapeutic LP, she was discharged on acetazolamide 250mg TDS, diclofenac 50mg TDS and co-dydramol PRN.

Unfortunately, her condition was not controlled medically and she had a further ten therapeutic LPs (opening pressures 27-53 cmsH₂O) over a 2 year period, as well as various unsuccessful combinations of medications including simple analgesia, acetazolamide, spironolactone and chlorthalidone.

With symptoms intractable to medical therapy, she underwent a ventriculo-peritoneal (VP) shunt insertion and had complete resolution of symptoms for 18 months when she re-presented with headaches. Shunt malfunction was excluded on 2 occasions over the following year with an LP (opening pressure 23), CT scan, shunt series imaging and intra-cranial monitoring which showed no spikes in ICP outside normal pressures. She was re-started on chlorthalidone 25mg BD and discharged.

In January 2006, the patient was able to secure funding from the Primary Care Trust Exceptional Funding Panel to undergo gastric banding surgery. She had been overweight from childhood, with marked weight gain during her two pregnancies. She had multiple failed attempts at dieting and her body mass index (BMI) at the time of referral for bariatric surgery was 41.

She was fully assessed by the multi-disciplinary team including endocrinologist, specialist nurse counsellor, dietician and surgeon before undergoing laparoscopic gastric banding surgery using a MIDband (Medical Innovation Developpement, Limonest, France). Under the careful observation of clinicians and nurse specialists she underwent incremental band inflations resulting in 30kg weight loss over a 12 month period. This corresponded to a loss of 81% of her excess body weight. Significantly, during this period, her headaches abated, and the medications intended to control her PTC were reduced and ultimately stopped. Her weight has remained stable and her symptoms have not returned over a one year period leading to a decision to remove her VP shunt.

DISCUSSION

PTC has a multi-factorial and poorly understood aetiology. While there is undoubtedly a relationship with obesity the precise pathological mechanisms remains under debate. Sugarman et al (6). postulate that raised intra-abdominal pressures directly induce raised intra-thoracic pressures, thus raised central venous pressures and so PTC. However, Walker notes they did not explain why the pulmonary artery pressures were higher in their obese patients with PTC than in equivalently obese patients without PTC (7).

Bariatric surgery as treatment for PTC does not provide an immediate resolution of intracranial hypertension. Thus, the patient at acute risk as a result of PTC would be better served with alternative treatments - e.g. emergent optic nerve fenestration for those at risk of blindness (8). However, in patients with chronic symptoms who are not at immediate risk of complications, bariatric surgery should be considered.

This case report clearly demonstrates how laparoscopic gastric banding led to a reduction in the patients CSF pressure and resolution of symptoms sufficient to allow removal of her VP shunt. This occurred after 5 years of medical therapy and 12 lumbar punctures had failed to produce sustained improvement in her condition.

Weight loss surgery has been shown to be the only reliable method of producing significant and sustained weight loss in the morbidly obese population (9). Current laparoscopic approaches mean that such surgery can be performed with low morbidity and mortality even in the super-obese (BMI ≥ 50) (10). With the significant risk of complications and side effects in VP shunting, and with the potential to also relieve other obesity related co-morbidities, laparoscopic bariatric surgery is an exciting alternative to conventional treatment of PTC.

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