CASE REPORT

Posterior reversible encephalopathy syndrome (PRES) after bariatric surgery—a potential consequence associated with rapid withdrawal of antihypertensive medications

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SUMMARY

A 61-year-old woman with a medical history of intracerebral haemorrhage, hypertension, hyperlipidaemia and carotid stenosis presented to the emergency department with altered mental status 3 weeks after undergoing a vertical sleeve gastrectomy for severe obesity. She presented with a hypertensive emergency and a National Institutes of Health Stroke Scale of 4. CT of the head was unrevealing. MRI showed an abnormal signal within the bilateral posterior border-zone areas, with several foci in the parietal and occipital lobes, and thalami, suggestive of posterior reversible encephalopathy syndrome (PRES). The patient was initially placed on a labetalol drip and her preoperative antihypertensive medications—amlodipine, captopril, triamterene and hydrochlorothiazide—were gradually reintroduced. She returned to her baseline and was stable on discharge. Rapid withdrawal of antihypertensive medications in the early postoperative period of bariatric surgery was the aetiology of PRES in this patient. This case report discusses postoperative care of bariatric surgery patients having hypertension.

BACKGROUND

While patients may have dramatic improvement in their metabolic profile in the immediate postoperative period of a bariatric surgery, blood pressure (BP) medications should be titrated off in a gradual fashion to reduce the likelihood of adverse reactions.

CASE PRESENTATION

A 61-year-old woman with a history of hypertension, hyperlipidaemia and carotid stenosis status post bilateral carotid endarterectomy (CEA) (right in 2000, left in 2001), with a reported history of an intracerebral haemorrhage in 2010, presented to the emergency department (ED) after an episode of word-finding difficulty 3 weeks after discharge home after undergoing a vertical sleeve gastrectomy (VSG) for class III obesity (preoperative body mass index (BMI) of 40.2 kg/m²).

Prior to admission to the hospital for her VSG, the patient was on several antihypertensive medications: (1) amlodipine 10 mg/benazepril 40 mg orally daily, (2) triamterene 37.5 mg/hydrochlorothiazide 25 mg orally daily and (3) prazosin HCl 5 mg orally two times per day. Because of the risk of severe dehydration, diuretics are routinely

stopped following bariatric surgery. Other antihypertensive medications are held and added back as needed to maintain a normal BP. Prior to her discharge home, the patient's BP was 112/62 mm Hg. Owing to her perceived relative normotensive BP, this patient with a history of resistant hypertension, who required five BP medications preoperatively, was sent home only on amlodipine 10 mg orally daily.

Three weeks after her initial discharge post-bariatric surgery, the patient stated that she went to bed on the night prior to her readmission, feeling anxious and stressed, and awoke on the day of admission, 'not feeling right'. She stated that she attempted to do a crossword puzzle, and noted difficulty performing this task. She went over to a neighbour's house, who immediately took her to the ED. While in the ED, she was noted to have an initial BP of 216/92 mm Hg and received labetalol with a goal systolic BP (SBP) <185.

At admission at 10:00, her initial National Institutes of Health Stroke Scale (NIHSS) score was 4—questions, partial L gaze, R facial, mild word-finding difficulty. The NIHSS is a 15-item clinical stroke assessment tool utilised to both evaluate and document neurological status in patients with acute stroke by their level of consciousness, language, neglect, visual field and sensory loss, extraocular movement, motor strength, ataxia and dysarthria. The tool has been validated to predict lesion size, and serves as a measure of stroke severity.

Initial head CT did not show an acute haemorrhage. The CT was followed by MRI and CT angiogram (CTA) of the head and neck. Owing to a concern for seizure activity, the patient was placed on lorazepam followed by a loading dose of levetiracetam. However, the levetiracetam was discontinued after the patient was not noted to have any additional seizure-like activity.

INVESTIGATIONS

MRI showed multiple areas of restricted diffusion in the parietal and occipital lobes, with associated fluid-attenuated inversion recovery signal (figure 1A, B). CTA of the head and neck was obtained, given the patient's history of bilateral CEA, and found to have moderate left internal carotid artery stenosis with calcified and non-calcified plaque proximally, as well as a potential area of narrowing of the mid left middle cerebral artery M1 area and mild right

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Figure 1 Axial MRI of the brain demonstrating increased signal on fluid-attenuated inversion recovery (A) and diffusion-weighted (B) images involving the occipital lobes (arrows).

internal carotid artery stenosis proximally. A neurology consult was placed to help discern the significance of these findings.

DIFFERENTIAL DIAGNOSIS

On the basis of the patient's presentation and medical history, the differential diagnosis included: (1) cerebrovascular accident, (2) posterior reversible encephalopathy syndrome (PRES) and (3) multiple embolic infarcts. The consulted neurologist believed that this patient's presentation was more consistent with PRES secondary to high SBP on presentation in conjunction with MRI findings.

TREATMENT

During her 6-day hospitalisation, the patient was gradually placed back on several of the antihypertensive medications she was on prior to her VSG. Her BP normalised on the four drug regimen to treat her hypertension (amlodipine 10 mg orally daily, triamterene 37.5 mg/hydrochlorothiazide 25 mg orally daily and captopril 75 mg orally three times per day). She was discharged home on obesity medicine, and scheduled for follow-up with internal medicine.

OUTCOME AND FOLLOW-UP

At the follow-up visit with her primary care physician 1 month after discharge, the patient was noted to have an acute kidney injury (AKI) denoted by an elevated blood urea nitrogen/creatinine (39 mg/dL/2.53 mg/dL) from her baseline values of 17 mg/dL/1.3 mg/dL. Nephrology was consulted, and her AKI was thought to be associated with renal hypoperfusion due to relative hypotension (the patient was noted to have several low BP readings after being restarted on the four BP agents) and renal autoregulation secondary to ACE inhibitor. Her captopril was held, and the patient's kidney status returned back to baseline over the course of 1 year. She has had several antihypertensive medication adjustments, and her current regimen consists of amlodipine 10 mg orally daily, benazepril 40 mg orally daily and spironolactone 100 mg orally daily.

The patient had an exceptional weight loss response to VSG. Her initial weight was 261 lbs with a BMI of 40.2 kg/m². At writing, her weight is 139 lbs and a BMI of 21.9 kg/m² with 47% of total body weight loss and 120% of excess body weight loss since surgery. On average, patients who undergo a VSG lose 50–60% of their excess body weight. Despite her postoperative course, the patient notes that she is content with her decision to undergo weight loss surgery due to her dramatically improved quality of life.

DISCUSSION

Bariatric surgery is an effective tool for weight loss and resolution of obesity comorbidities in patients with moderate-to-severe obesity.^{1–6} Recent studies have sought to delineate how bariatric surgery procedures (eg, Roux-en-Y gastric bypass (RYGB) and VSG) compare with regard to improving a patient's cardiovascular status. Benaiges *et al*⁷ and Zhang *et al*⁸ concluded that RYGB and VSG are equivalent in reducing obstructive sleep apnoea, hyperlipidaemia, hypertension, type 2 diabetes and musculoskeletal disease. Despite the significant potential improvement in obesity comorbidities in patients who undergo bariatric surgery, it is important to provide optimal care in the perioperative period.

Postoperative bariatric surgery guidelines provide clear information on surgical follow-up, laboratory work, dietary and psychological counselling, and exercise prescription,^{9–10} but there is significant variability in medical management recommendations, which is likely secondary to patient variability. Unfortunately, reviews have demonstrated that patients often receive inadequate bariatric surgery preoperative and postoperative care.¹¹ While bariatric surgery may improve or eliminate obesity-related hypertension,^{12–14} patients with refractory hypertension will likely require gradual titration off of antihypertensive medications. Current data suggest that patients should be monitored weekly until their BP has stabilised, and patients may need to resume antihypertensive medications (often at adjusted doses).¹⁵ Our case suggests that patients with severe hypertension need

closer monitoring of their BP in the early and late postoperative course. As the popularity of bariatric surgery procedures increases secondary to burgeoning obesity rates in developed countries, bariatric surgeons, obesity medicine physicians and primary care providers should be aware of the need to closely monitor this unique patient population in the postoperative period. Providers should also be aware of the wide variation in response to bariatric surgery as it relates to weight response and resolution or improvement of obesity comorbidities such as hypertension.

PRES refers to a disorder of reversible subcortical vasogenic brain oedema in patients with acute neurological symptoms (eg, seizures, encephalopathy, headache and visual disturbances) in the setting of renal failure, BP fluctuations, cytotoxic drugs, autoimmune disorders and preeclampsia or eclampsia. We have provided a brief table with causes of PRES (box 1). Brain imaging usually reveals vasogenic oedema predominantly involving the bilateral parieto-occipital regions.¹⁶ Unfortunately, the pathogenesis of PRES is not well understood. There are three hypotheses that have been proposed to explain the entity: (1) derangements in the autoregulatory system in cerebral circulation lead to brain oedema, (2) hypertension activates the autoregulatory system leading to vasoconstriction of brain vessels and resultant hypoperfusion, ischaemia and fluid leakage and (3) the activation of the immune system and the consequent endothelial activation start a molecular cascade, which produces molecules that alter the normal homeostasis of the blood–brain barrier with subsequent fluid leakage and drainage.¹⁷ Despite the unclear pathophysiology, the diagnosis has important therapeutic and prognostic implications, as the reversibility of the clinical and radiological abnormalities is contingent on prompt control of BP and/or discontinuing the offending drug.¹⁸

To the best of our knowledge, this is the first case presentation of PRES after bariatric surgery secondary to rapid withdrawal of antihypertensive medications. Nakabou *et al*¹⁹ reported two cases of PRES in patients with chronic renal insufficiency who developed hypertensive encephalopathy after the sudden discontinuance of antihypertensive agents. Patients who undergo bariatric surgery often have complex medical histories, and it is important to tailor postoperative care to minimise morbidity while maximising high quality care.

Box 1 Abbreviated list of causes of posterior reversible encephalopathy syndrome

Hypertension/hypertensive encephalopathy
Infections/sepsis/septic shock
Acute/chronic renal disease
Preeclampsia/eclampsia
Autoimmune diseases
Immunosuppressive drugs
Nutritional derangements (eg, hypercalcaemia, hypomagnesaemia)
Other drugs (eg, erythropoietin, interferon α , intravenous immunoglobulin, caffeine)
Blood transfusions
Endocrine disorders (eg, pheochromocytoma, primary aldosteronism)

Patient's perspective

- I have had high blood pressure for years and was on several medications before weight loss surgery. Now, two years after surgery, I still take several blood pressure medications; given my own medical history, and my family history, I expect that I will need to continue to do so for the rest of my life.
- When I was being discharged from the hospital after the weight loss surgery, the medical team told me to just take one of the blood pressure medications I had been prescribed because they did not want my blood pressure to get too low. After leaving the hospital, I did see an associate of my primary care doctor [my doctor was on vacation] to have my blood pressure checked, and it was fine. I monitored it at home as well, and it was within the range the discharge medical team had recommended. However, three weeks later, I felt "off," tried to do a crossword puzzle [which I do often] but couldn't put letters in the right boxes; I tried to check my blood pressure but kept getting an error message, and I couldn't see the numbers on my cell phone. I vaguely remember arriving at the hospital, but don't remember having a CAT scan, MRI, or my clothing being replaced with a "Johnny".
- I am very thankful for having had the vertical sleeve procedure, as my quality of life, particularly my well-being, has improved immensely. I do hope that the lessons learned from my experience will help future weight loss surgery patients avoid similar experiences.

Learning points

- Patients who undergo bariatric surgery require careful perioperative medical management for obesity-related comorbidities.
- Rapid withdrawal of blood pressure medications in the immediate postoperative course may lead to posterior reversible encephalopathy syndrome.
- Patient requiring multiple antihypertensives prior to bariatric surgery are unlikely to completely resolve their hypertension after surgery, but they may experience some improvement.

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Competing interests None declared.

Patient consent Obtained.

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REFERENCES

- 1 Athyros VG, Tziomalos K, Karagiannis A, *et al*. Cardiovascular benefits of bariatric surgery in morbidly obese patients. *Obes Rev* 2011;12:515–24.
- 2 Courcoulas AP, Christian NJ, Belle SH, *et al*. Weight change and health outcomes at 3 years after bariatric surgery among individuals with severe obesity. *JAMA* 2013;310:2416–25.
- 3 Crémieux PY, Ledoux S, Clerici C, *et al*. The impact of bariatric surgery on comorbidities and medication use among obese patients. *Obes Surg* 2010;20:861–70.
- 4 Kaul A, Sharma J. Impact of bariatric surgery on comorbidities. *Surg Clin North Am* 2011;91:1295–312.

- 5 Våge V, Sande VA, Mellgren G, *et al.* Changes in obesity-related diseases and biochemical variables after laparoscopic sleeve gastrectomy: a two-year follow-up study. *BMC Surg* 2014;14:8.
- 6 Vest AR, Heneghan HM, Agarwal S, *et al.* Bariatric surgery and cardiovascular outcomes: a systematic review. *Heart* 2012;98:1763–77.
- 7 Benaiges D, Goday A, Ramon JM, *et al.* Laparoscopic sleeve gastrectomy and laparoscopic gastric bypass are equally effective for reduction of cardiovascular risk in severely obese patients at one year of follow-up. *Surg Obes Relat Dis* 2011;7:575–80.
- 8 Zhang N, Maffei A, Cerabona T, *et al.* Reduction in obesity-related comorbidities: is gastric bypass better than sleeve gastrectomy? *Surg Endosc* 2013;27:1273–80.
- 9 McMahon MM, Sarr MG, Clark MM, *et al.* Clinical management after bariatric surgery: value of a multidisciplinary approach. *Mayo Clin Proc* 2006;81(10 Suppl): S34–45.
- 10 Tariq N, Chand B. Presurgical evaluation and postoperative care for the bariatric patient. *Gastrointest Endosc Clin N Am* 2011;21:229–40.
- 11 Kmietowicz Z. Obese patients get inadequate care before and after bariatric surgery, finds review. *BMJ* 2012;345:e6890.
- 12 Frezza EE, Wei C, Wachtel MS. Is surgery the next answer to treat obesity-related hypertension? *J Clin Hypertens (Greenwich)* 2009;11:284–8.
- 13 Sarkhosh K, Birch DW, Shi X, *et al.* The impact of sleeve gastrectomy on hypertension: a systematic review. *Obes Surg* 2012;22:832–7.
- 14 Wilhelm SM, Young J, Kale-Pradhan PB. Effect of bariatric surgery on hypertension: a meta-analysis. *Ann Pharmacother* 2014;48:674–82.
- 15 Elrazek AE, Elbanna AE, Bilasy SE. Medical management of patients after bariatric surgery: principles and guidelines. *World J Gastrointest Surg* 2014;6:220–8.
- 16 Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol* 2015;14:914–25.
- 17 Marra A, Vargas M, Striano P, *et al.* Posterior reversible encephalopathy syndrome: the endothelial hypotheses. *Med Hypotheses* 2014;82:619–22.
- 18 Lamy C, Oppenheim C, Mas JL. Posterior reversible encephalopathy syndrome. *Handb Clin Neurol* 2014;121:1687–701.
- 19 Nakabou M, Kai T, Maeshima T, *et al.* Hypertensive encephalopathy in patients with chronic renal failure caused by stopping antihypertensive agents: a report of two cases. *Clin Exp Nephrol* 2010;14:256–62.

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