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Acute Paralytic Post-Bariatric Surgery Axonal Polyneuropathy: Clinical Features and Outcome

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Obesity · Neuropathy · Bariatric surgery · Paralysis · Prognosis

Abstract
Introduction: The syndrome of post-bariatric surgery axonal polyneuropathy (PAP) may present with various sensory and motor symptoms including paralysis. We aim to describe the diagnostic features and outcome of treatment of the acute paralytic form of PAP (acute paralytic PAP [APPAP]) as it was not described in a separate cohort previously. ***Methods:*** We retrospectively reviewed medical charts and described the clinical, electrodiagnostic features, treatment, and outcome for patients who presented to our clinical neurophysiology unit with disabling weakness within 24 months post-bariatric surgery. The main outcome measure was the percent of patients who are able to walk independently at the last visit and comparing the group who resumed walking independently at 6 months to the group who did not, in regards to the use of intravenous immunoglobulin (IVIg). ***Results:*** Thirteen patients were included (10 women and 3 men) with a mean age of 29.8 years (SD 12.5). All presented with loss of ambulation resembling Guillain-Barre Syndrome. The median time of onset was 4 months (interquartile range [IQR] 3–6) post-surgery, and the median time to weakness nadir was 3 weeks (IQR 3–3.5) with an average weight loss of 38.6 kg (SD 17.09). All patients regained their ability to ambulate; however, the ability to walk independently was achieved in 66.7% of patients. The percent of patients who were able to ambulate independently at 6 months were 16% with IVIg versus 66.7% without IVIg. ***Conclusion:*** The syndrome of APPAP develops in the first-year post-bariatric surgery. The majority of patients regain independent ambulation.

Introduction

Bariatric surgeries are associated with several neurological complications including polyradiculoneuropathy, peripheral neuropathy, myelopathy, encephalopathy, and optic neuropathy [1, 2]. In a previous study, peripheral neuropathy was reported in 16% of patients who underwent bariatric surgery and was characterized into polyneuropathy (6.2%), mononeuropathy (9.0%), and radiculoplexus neuropathy (1.1%) [3]. Polyneuropathy typically presents with distal sensory symptoms with or

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without weakness. More than 15 cases of acute paralytic post-bariatric surgery axonal polyneuropathy (APPAP) have been described in several previous studies [2, 4–6]. These were small-scale reports ($n < 5$ patients) and lacked detailed clinical, electrodiagnostic (EDX) evaluation, and outcome data [2].

Obesity has become a huge burden worldwide in both developed and developing countries. In 2016, the World Health Organization reported a prevalence of obesity of 13% among the adult population (11% of men and 15% of women) [7]. In addition, it has been reported that bariatric surgery reduces the morbidity of obesity and has been shown to lower the 10-years relative risk of coronary artery disease by 40% [8]. Therefore, the demand for bariatric surgeries is likely to continue rising.

This study sought to retrospectively describe the clinical presentation, EDX findings, and outcome of different treatment in patients who underwent bariatric surgery and presented with disabling weakness related to post-bariatric surgery axonal polyneuropathy syndrome (PAP). We explored the possible association of different clinical variables with outcome. Few cases were reported in the literature as Guillain-Barre syndrome (GBS), and hence we aimed to investigate the rule of intravenous immunoglobulin (IVIg) on outcome [6, 9].

Materials and Methods

Study Design

This was a retrospective study of patients who presented with APPAP at King Abdulaziz University Hospital from January 2014 to May 2018. The study was approved by the institutional review board at King Abdulaziz University Hospital. Patients were identified by reviewing our clinical neurophysiology unit and neuromuscular clinic database. Subsequently, patients' charts were reviewed to extract data related to clinical presentation and outcome, and EDX studies were retrieved and reviewed. Inclusion criteria included (1) age of 18 years or more, (2) weakness within 24 months post bariatric surgery, and (3) proximal and distal weakness in lower limbs and or upper limbs, and (4) loss of ambulation. We excluded patients with coexisting neurological conditions known to cause proximal weakness such as myasthenia gravis, inflammatory myopathies, congenital myopathy, and myelopathy.

Study Variables

Clinical Presentation

In addition to demographic data, we collected data on the distribution of sensory and motor features, time of onset of weakness (defined as number of months from surgery date to onset of symptoms), time to reach weakness nadir (defined as number of weeks from onset of symptoms to the maximum degree of weakness), degree of weight loss at the time of presentation to our hospital, type of bariatric surgery, or procedure and type of therapy received (vitamin replacement and/or IVIg).

Laboratory Data

We collected data on levels of albumin (as a nutritional status indicator), potassium (as an indicator of vomiting severity), vitamin B12, thiamine, vitamin D, and copper and data on cerebrospinal fluid and MRI findings.

EDX Data

We extracted the following variables from sensory and motor nerve conduction studies: compound muscle action potential (CMAP) amplitude, CMAP distal latency, sensory nerve action potential (SNAP) amplitude, SNAP peak latency, and conduction velocity. Regarding electromyography, we extracted data on affected muscles (proximal, distal or both) and data on abnormalities (active and/or chronic denervation).

Prognosis

To determine prognosis, we used the following variables: (1) ability to regain walking with or without assistance and (2) time (in months) to regain walking from onset. The effect of IVIg on outcome was explored as one of the outcome predictors.

Exploring Predictors of Outcome

Patients were characterized into 2 groups, those who regained independent walking at 6 months from onset of the disease (good prognosis) and those who did not regain independent walking (bad prognosis). The 2 groups were compared on the following variables: age, sex, weight loss at the time of disease onset, time of onset of weakness, time to reach weakness nadir, presence of upper limb weakness, vitamin B12 level, vitamin D level, CMAP, and SNAP amplitudes.

Statistical Analysis

Descriptive statistics were used for demographic variables as appropriate. Data on the clinical presentation, laboratory tests, EDX studies, and outcomes were presented as median, interquartile range (IQR), and proportion, as appropriate. In order to explore variables for their potential association with a good outcome, we compared the 2 groups (good and bad prognosis) using the Mann-Whitney test for the median and Fisher's exact test for the proportion.

Results

Clinical Presentations

A total of 13 patients were included, 10 women (77.0%) and 3 men (34%), with a median age of 26 years (IQR 23–28). Eleven patients (84.6 %) had sleeve surgery, 1 (7.7%) had balloon, and 1 (7.7%) had bypass surgery. Weakness was preceded by vomiting in 12/13 patients (92.3%), and the average weight loss was 38.6 (SD 17.09) kg. All patients presented with acute to subacute bilateral ascending weakness with a median time of onset from

Table 1. Clinical characteristics of patients

Clinical feature, median (IQR)	
Age, years	26 (23–28)
Gender, female, *n* (%)	10 (77.0)
DM, *n* (%)	2 (15.4)
HTN, *n* (%)	1 (7.7)
Weight loss, kg	30 (28–60)
Onset post-surgery, months	4 (3–6)
Nadir of weakness, weeks	3 (3–3.5)
UL weakness, *n* (%)	7 (53.8)
Albumin, g/L	30.9 (28–34)
Vitamin B12 level, pmol/L	993 (456–1,186)
Vitamin D level, nmol/L	46.12 (24–80)
Received IVIg, %	41.67
EDX, median (IQR)	
Median CMAP, mV	9 (6.5–9.37)
Ulnar CMAP, mV	8 (6.3–10.9)
Tibial CMAP, mV	4.9 (0.05–8.22)
Common peroneal CMAP, mV	1.58 (0–3.88)
Median SNAP, μV	32.4 (5.8–44.4)
Ulnar SNAP, μV	30 (8.1–45.8)
Sural SNAP, μV	4.45 (0–11.9)

IQR, interquartile range; UL, upper limb; IVIg, intravenous immunoglobulin; CMAP, compound muscle action potential; SNAP, sensory nerve action potential.

surgery of 4 months (IQR 3–6) and time from onset to weakness nadir of 3 weeks (IQR 3–3.5; Table 1).

Neurological examination revealed symmetrical and proximal predominant weakness in 6 patients (46.2%) and distal predominant weakness in 7 patients (53.8%). Seven patients (53.8%) had upper limb weakness (proximal predominant in 2 patients, distal predominant in 3 patients, and equal proximal and distal weakness in 2 patients), 2 patients (15.4%) had bilateral facial weakness, 1 patient (7.7%) had weak neck flexion (patient number [No.] 13), 2 patients (15.4%; Nos. 1, 2) had respiratory weakness, 11 patients (84.6%) had sensory impairment in a glove-stocking distribution, and all patients had normal cerebellar examination. All patients were prescribed multivitamins after bariatric surgery, but only 4 reported a good adherence despite having episodes of severe vomiting that they attributed to vitamins (Table 2).

Laboratory Analysis

Five patients (38.4%) had hypokalemia, with the median K level of 3.7 (IQR 3–4.1); 12 patients (92.3%) had a low albumin level with the median albumin level of 30.9 (IQR 28–34); 4/4 patients had low vitamin B1 level; all patients had a normal or high vitamin B12 level (on supplements) with a median level of 993 IQR (456–1,186); 7/11 patients (63.6%) had a low vitamin D level (median 46.12, IQR 24–80). Regarding heavy metals, zinc was normal in 3/3 patients, selenium was normal in 2/2 patients, and copper was normal in 5/5 patients. Cerebrospinal fluid was normal in most patients 6/7 (86%), and only 1/7 patient (14%) had albuminocytologic dissociation (No. 3). Spine MRI was normal in 11/11 patients. (online suppl. Table 1; for all online suppl. material, see www.karger.com/doi/10.1159/000503286).

Electrophysiological Examination

EDX studies were abnormal in all patients as shown in Table 1 (also, online suppl. Table 2: EDX findings per patient). Tibial CMAP amplitudes were decreased in 5/12 patients (41.7%); fibular CMAP amplitudes were decreased in 8/12 patients (66.6%); median and ulnar CMAP amplitudes were reduced in 1/12 patient (8.33%); sural SNAP amplitudes were decreased in 5/12 patients (41.67%); superficial fibular SNAP amplitudes were decreased in 3/7 patients (42.8%); median SNAP amplitudes were decreased in 4/12 patients (33.3%), and ulnar SNAP amplitudes were decreased in 3/12 patients (25%). Conduction velocities and distal latencies were either normal or mildly abnormal (within the expected range for the degree of axonal loss). Electromyography showed active denervation in the proximal and distal lower limb muscles in 10/11 patients (90.9%) and in proximal upper limb muscles in 5/11 patients (45.4%). Overall, EDX studies were consistent with axonal polyneuropathy.

Therapy and Outcome

All the patients received multivitamins including vitamin B1, B6, and B12 in variable doses, while only 6 patients (Nos. 1, 2, 3, 6, 8, and 13) received IVIg as empirical treatment for a suspected autoimmune neuropathy. Two patients (Nos. 1, 2) were admitted to the intensive care unit. Clinical follow-up was available for 12 patients; 8/12 patients (66.7%) regained their ability to walk with a median time from onset of 3 months (IQR 2.5–7), and the remaining 4 patients improved from baseline and resumed ambulation but needed support to walk.

Sural SNAP amplitudes were significantly higher in patients who regained independent walking at 6 months than in those who did not regain independent walking (Table 3). Other variables were not statistically different between the 2 groups. The use of IVIg was not associated with increased chance to walk independently (16% with IVIg vs. 66.7% without IVIg, $p = 0.242$). Other variables

Table 2. Clinical characteristics and outcome per patient

Patient No.	1	2	3	4	5	6	7	8	9	10	11	12	13
Age, years	21	26	22	65	19	28	23	41	27	40	26	23	26
Gender	Female	Female	Female	Male	Female	Male	Female	Female	Female	Female	Female	Male	Female
Comorbidity	Free	Free	Free	DM, HTN	Free	Free	Free	Free	DM	Free	Free	Free	Free
Surgery	Sleeve	Sleeve	Sleeve	Sleeve	Sleeve	Sleeve	Sleeve	Balloon	Sleeve	Bypass	Sleeve	Sleeve	Sleeve
Onset post OP, months	3	4	2	8	3	3	4	3	7	5	9	6	2
Nadir, weeks	3	5	4	3	4	3w	3	2	3	ND	3	2	3
Weight loss, kg	30	20	NA	60	40	47	15	28	NA	30	60	65	30
BMI	42	35	43	46	51	35	47	45	NA	37	NA	NA	38
Vomiting	Present	Present	Present	Present	Absent	Present	Present	Present	Present	Present	Present	Present	Present
Preceding infection	+	_	_	_	_	+	_	_	_	_	+	_	+
Facial weakness	Intact	Weak	Intact	Intact	Intact	Weak	Intact	Intact	Intact	Intact	Intact	Intact	Intact
UL power (MRC)													
Prox/dist	Intact	2/0	Intact	4/5	5/4	4+/4–	Intact	4	Intact	Intact	4	4+/5	Intact
LL power													
Prox/dist	4–/1	2/0	3/3	3/4	2/0	2/4	4/5	1/5	4/4	3/3	4/2	4–/4	4/4
Sensory													
Pinprick	Impaired	Impaired	Impaired	Impaired	Impaired	Impaired	Intact	Impaired	Impaired	Impaired	Impaired	Impaired	Impaired
Dorsal column	Impaired	Impaired	Intact	Intact	Impaired	Impaired	Intact	Impaired	Impaired	Impaired	Impaired	Intact	Intact
Reflex UL/LL	+2/0	Absent	+2/0	Absent	Absent	+2/0	+2/0	NA	NA	+2/+2	Absent	NA	+2/0
Outcome													
Ability to walk	8 months	18 months	Cane within 3 months	Lost F/u	Support within 3 months	Walker. Within 3 months	3 months	6 months	3 months	2 months	3months	Cane within 3 months	1 months

NA, not available; UL, upper limb; LL, lower limb; MRC, Medical Research Council; prox, proximal; dist, distal.

Table 3. Comparing variables based on outcome

Variable	Walk independently at 6 months	Walk with assistance at 6 months	*p* value
Age, years, median (IQR)	26.5 (26–40)	22.5 (21–26)	0.063
Gender, female, *n* (%)	6 (100)	6 (66.7)	0.455
Weight loss, kg, median (IQR)	30 (28–30)	40 (30-47)	0.341
Onset post-surgery, months, median (IQR)	4.5 (3–7)	3 (3–4)	0.287
Nadir of weakness, weeks, median (IQR)	3 (3–3)	3.5 (3–4)	0.193
UL weakness, %	33.33	66.67	0.567
Albumin, g/L, median (IQR)	32.5 (30–40)	30.5 (28–34)	0.46
Vitamin B12 level, pmol/L, median (IQR)	875 (412–1,068)	1089.5 (834–1,476)	0.337
Vitamin D level, nmol/L, median (IQR)	69.5 (30–91.9)	34.61 (24–69.58)	0.605
IVIg, %	16.67	66.67	0.242
EDX, median (IQR)			
Median CMAP, mV	9.1 (8.6–10)	9.05 (6.5–9.3)	0.39
Ulnar CMAP, mV	8.635 (6.8–10.5)	8.05 (6.3–10.9)	0.91
Tibial CMAP, mV	7.24 (5.1–9.2)	3.65 (0.1–6.1)	0.314
Common peroneal CMAP, mV	2.95 (2.3–4.8)	0.3 (0–1.3)	0.065
Median SNAP, μV	41.7 (32.5–46.2)	19.1 (4.7–33.3)	0.2
Ulnar SNAP, μV	50.5 (24–83)	22.75 (6.6–42)	0.2
Sural SNAP, μV	10 (7–19.7)	0.65 (0–3.7)	0.027

IQR, interquartile range; UL, upper limb; IVIg, intravenous immunoglobulin; EDX, electrodiagnostic; CMAP, compound muscle action potential; SNAP, sensory nerve action potential.

had no statistical association with the outcome, but there was possibly a trend of delayed walking regain in those who were younger, as shown in Table 3.

Discussion

Post-bariatric surgery neuropathy encompasses a spectrum of clinical presentations varying from the more common chronic sensory (painful) axonal predominant polyneuropathy and to a less common presentation with APPAP, which manifests as a disabling proximal and distal weakness resembling GBS [1, 3, 9, 10]. Our cohort of 13 patients characterized the APPAP syndrome in isolation from other types of post-bariatric surgery neuropathies and described its outcome in relation to therapy which is not reported in the literature before. All our APPAP cases had disabling weakness that progressed to loss of ambulation. The weakness grade was <4 on the MRC in most of APPAP cases (77% of patients). The nadir weakness was 4 weeks or less in 92.3% of APPAP cases. Active denervation in proximal muscles was seen in 76.9% of APPAP cases.

In our study, the median time of symptom onset after bariatric surgery was 4 months (range 3–6 months). This is consistent with the reports by Juhasz-Pocsine et al. [2] and Yasawy and Hassan [4], whereby all but 1 patient presented in the first 4 months. Unlike APPAP, PAP without loss of ambulation usually presents after the first-year post-bariatric surgery and more often many years later [2]. In our series, the first clinical feature developed was paresthesia (all patients), which was mostly painful and required neuropathic pain therapy in 66.6% of patients. This was followed by lower limb ascending weakness in all cases. Similar to our experience, a previous study reported the onset of paresthesia followed by weakness [2]. However, distinctive findings in some of our patients were facial and neck flexor weakness in 1 patient and respiratory muscle weakness in 2 patients. The nadir of weakness in our cohort was 4 weeks in all cases except one, and this is consistent with a study by Juhasz-pocsine et al. [2] who reported 5 cases developing APPAP, all of them became bedridden within a week.

The amount of weight loss and short latency to weight loss may contribute to the development of APPAP, possibly through micronutrition deficiency and thiamine in particular [11]. Rapid weight loss is frequently associated with vomiting that increases the risk of micronutrients deficiency (specifically thiamine which has a short half-life of 18 days) [12]. The average weight loss in our cohort (38.6 kg) was slightly less than that reported with other APPAP cases (43.8 and 43.0 kg) [2, 3]. This amount of weight loss occurred over 4 months in our cohort as well as in prior reported series [2]. In contrast, Thaisetthawatkul et al. [3] found that the patients who did not develop neuropathy had a significantly lower degree of weight loss (33 kg), which occurred over a longer period of time (19 months) than in those who developed neuropathy (43 kg) over 8 months. The patients in our cohort who were able to walk independently at 6 months had a median weight loss of 30 kg (IQR 28–30) versus 40 kg weight loss (IQR 30–47) in those who did not regain independent walking. These numbers were not statistically significant but may provide another hint that greater and more rapid weight loss may be associated with developing APPAP in a dose-effect pattern.

Despite our cohort did not link APPAP with any nutrient in particular, however, in the context of some of the previous reports, APPAP cases seem to be commonly suffering from a low thiamine level and likely APPAP represents a form of dry beriberi [13]. In our cohort, APPAP was preceded by vomiting in 92% of the cases, and thiamine level was low in all 4 patients in whom it was measured. Koike et al. [14] described a series of 17 patients who developed motor predominant acute polyradiculoneuropathy postgastrectomy and all had a low thiamin level. In another cohort of patients with thiamin deficiency neuropathy, 84% had motor predominant neuropathy, and in 56% of patients it progressed acutely within 1 month [15]. A report of gastric surgery for morbid obesity described a case who developed thiamine deficiency and presented with severe weakness and loss of ambulation [16]. Another report of 5 cases of thiamine deficiency after gastric bypass or balloon procedures demonstrated cases presenting with weakness in legs with or without arm weakness and impaired ambulation to the point of requiring a wheelchair or crutches [5]. The acute nutritional axonal neuropathy cohort from a variety of causes showed a low thiamine level in all 4 patients in whom the level was measured before supplementation; however, those patients were able to ambulate with a walker or a cane unlike the other cohorts [10]. Thiamine deficiency or dry beriberi could be the likely associated with the syndrome of polyneuropathy as thiamine can be depleted in less than a month duration unlike other vitamins [10, 12, 17]. Also thiamine utilization could be impaired in malnourished patients [18].

The outcome of the APPAP is generally good. In our cohort, 66.7% of patients regained their ability to walk independently mostly within 4 months, but it may take

up to 18 months. All patients regained their ability to ambulate; however, 4/12 patients (33.3%) needed support. In regard to the outcome of the previously described cases of APPAP, all 6 cases reported by Juhaszpocsine et al. [2] regained ambulation but residual sensory ataxia persisted in most of them. Koike et al. [14] reported that 60% of patients regained their ability to ambulate within 3 months and 80% within 6 months after thiamine administration. All 3 patients reported by Yasawy et al. [4] regained their ability to walk within 4 months from the onset. Our study suggests that preserved sural nerve potential is a possible predictor of good outcome. Immunotherapy has been thought to improve the outcome in post-surgical inflammatory neuropathy [19]. IVIg treatment was tried in 6 patients in our APPAP cohort; however, it did not alter the chances to regain ambulation at 6 months as shown in Table 3.

There are several limitations to our study: first, the inherited bias associated with retrospective studies and the single center nature of our study. Second, the small sample size may affect the strength of the conclusions that can be drawn from our data. Third, not all investigations are done in our study as well as the absence of nerve biopsy [20]. The fourth limitation is the lack of a comprehensive outcome measure, as many patients might suffer from gait imbalance despite being able to walk again [21]. Despite these limitations, our data are in concordance with prior reports, and it adds another layer of confirmation to the clinical and prognostic information regarding this syndrome.

In conclusion, our study shows that APPAP mimics GBS in term of acute onset and time to nadir weakness. Cases with this syndrome usually present within 6 months after bariatric surgery. The preserved sural nerve potential could possibly be a predictor of cases with better outcome and needs to be investigated in future studies. Overall, this syndrome has a good prognosis as most patients regain their ability to walk.

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Statement of Ethics

The study has received an approval from the institutional review board at King Abdulaziz University, Medical School.

Disclosure Statement

The authors have no conflicts of interest to declare.

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Author Contributions

O.A. and H.A.A.: study design, data acquisition, and writing the manuscript. A.A.: study design, data acquisition, and reviewing the manuscript. M.H.A.: study design, data interpretation, and reviewing the manuscript. A.R.A.: study design, data acquisition and interpretation, statistical analysis, and writing the manuscript.

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