A Case of Endoscopic Sleeve Gastroplasty in Bardet–Biedl Syndrome After Solid Organ Transplantation

Yael Sofer, MD[1,4] · Mati Shnell, MD[2,4] · Sharon Levy, MD[2,4] · Michal Raz, MD[3,4] · Limor Ben Haim, MSc[1,4] · Roy Eldor, MD, PhD[1,4] · Sigal Fishman, MD[2,4]

[1]Institute for Endocrinology, Metabolism and Hypertension · [2]Institute for Diseases of the Digestive Tract and Liver · [3]Nephrology — Tel Aviv Sourasky Medical Center (Ichilov), Tel Aviv, Israel

[4]Gray Faculty of Medical and Health Sciences, Tel Aviv University, Israel

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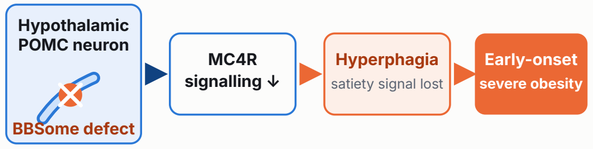
BACKGROUND

Bardet–Biedl syndrome (BBS) is a rare autosomal-recessive ciliopathy characterised by severe early-onset obesity, hyperphagia, retinal dystrophy, metabolic dysfunction and multisystem disease.

Dysfunction of the immotile primary cilium in the hypothalamic melanocortin-4 receptor (MC4R) pathway impairs satiety signalling and drives relentless food-seeking behaviour.[1,2]

Data on endoscopic bariatric therapies in BBS are lacking, and the evidence base in solid organ transplant recipients remains limited to isolated reports.[7,8]

BBS — MECHANISM & PHENOTYPE



MULTISYSTEM CILIOPATHY

- Rod–cone dystrophy
- Renal structural disease
- Hepatic steatosis → MASH
- Type 2 diabetes, dyslipidaemia
- Polydactyly, hypogonadism
- Learning difficulties

Ciliary BBSome dysfunction → impaired MC4R trafficking and signalling → hyperphagia → severe early-onset obesity, superimposed on multisystem ciliopathy.

THE THERAPEUTIC GAP

- Lifestyle intervention alone is rarely sufficient in hyperphagic syndromic obesity.
- MC4R agonist therapy (setmelanotide) is approved but not universally available, tolerated or funded.[3]
- Bariatric surgery carries amplified risk in transplant recipients with cardiopulmonary disease.
- **An organ-sparing, incisionless option was required.**

CASE PRESENTATION

PATIENT

48-year-old man with genetically and clinically confirmed Bardet–Biedl syndrome and severe obesity (baseline weight ~116 kg), previously treated with basal–bolus insulin and semaglutide, with persistent obesity despite lifestyle intervention.

Liver transplant (MASH) · **Kidney transplant** · Type 2 diabetes · CKD · Macroalbuminuria · Hypertension · Dyslipidaemia · Hypertrophic cardiomyopathy · Mod–severe aortic stenosis · COPD · Retinitis pigmentosa

CLINICAL COURSE

- **Prior** — Liver transplantation for MASH; kidney transplantation, maintenance tacrolimus
- **Pre-procedure** — Severe obesity (116 kg) despite lifestyle intervention, basal–bolus insulin and prior semaglutide
- **Multidisciplinary review** — Bariatric surgery declined: HCM, moderate–severe aortic stenosis, COPD, CKD, dual allograft
- **June 2025 — Endoscopic sleeve gastroplasty**
  Organ-sparing, incisionless; no major procedural complication
- **3 months** — −30 kg; marked appetite reduction, improved portion control; insulin requirement reduced
- **~6 months** — Weight loss durable; graft function stable; tacrolimus within therapeutic range

WHY ENDOSCOPIC SLEEVE GASTROPLASTY?

- Incisionless, organ-sparing and reversible — no intestinal diversion.
- Preserves enteral absorption of calcineurin-inhibitor immunosuppression.
- Avoids general surgical and haemodynamic risk in severe aortic stenosis and HCM.
- Established efficacy in non-syndromic obesity: ~15–16% total body weight loss sustained to 5 years.[4–6]

RESULTS AT 3 MONTHS

−30 kg WEIGHT LOSS

25.9% TOTAL BODY WEIGHT LOSS



WEIGHT TRAJECTORY



ALLOGRAFT & PROCEDURAL SAFETY

- **No major procedural complications.**
- No evidence of hepatic or renal allograft rejection.
- Tacrolimus levels remained stable within the therapeutic range.
- Insulin requirements reduced; improved functional capacity and exercise tolerance.
- Weight loss durable at ~6 months of follow-up.

LIMITATIONS

- Single case; no causal inference and no comparator.
- Residual effect of prior GLP-1 receptor agonist therapy cannot be formally excluded, although weight had not responded to it.
- Short follow-up (~6 months); durability beyond this is unknown.
- Single-centre measurement; albuminuria is subject to intra-individual variability.

DISCUSSION

In hyperphagic syndromic obesity, the therapeutic target is **ingestive behaviour**. ESG reduces gastric reservoir capacity and slows emptying, producing early satiation — a mechanism that operates **downstream of the defective hypothalamic MC4R pathway** and is therefore not dependent on intact ciliary signalling.

The magnitude of weight loss observed here (25.9% TBWL at 3 months) exceeds reported ESG outcomes in non-syndromic obesity.[4–6] Semaglutide had been introduced **before** ESG without achieving sustained weight reduction, so the post-procedural loss is unlikely to represent a residual GLP-1 receptor agonist effect.

Metabolic and renal parameters moved in parallel with weight, and critically, **graft function and immunosuppressant exposure were unaffected** — a central concern for any bariatric intervention after transplantation.[7–9]

CONCLUSIONS

- ESG was **feasible, well tolerated and effective** in an adult with BBS and dual solid organ transplantation at prohibitive surgical risk.
- Clinically meaningful weight loss was accompanied by improved glycaemia, albuminuria and functional capacity, **without allograft compromise**.
- To our knowledge this is **among the first reported cases of ESG in Bardet–Biedl syndrome**.
- Endoscopic bariatric therapy merits prospective evaluation in syndromic obesity and in medically complex transplant recipients.

REFERENCES

1. **Beales PL, et al.** Hyperphagia in Bardet–Biedl syndrome: pathophysiology, burden, and management. *Obes Rev*. 2025. doi:10.1111/obr.13915
2. **Tomlinson JW.** Bardet–Biedl syndrome: a focus on genetics, mechanisms and metabolic dysfunction. *Diabetes Obes Metab*. 2024. doi:10.1111/dom.15480
3. **Haqq AM, et al.** Efficacy and safety of setmelanotide in Bardet–Biedl and Alström syndrome: randomised phase 3 trial. *Lancet Diabetes Endocrinol*. 2022. doi:10.1016/S2213-8587(22)00277-7
4. **Abu Dayyeh BK, et al.** Endoscopic sleeve gastroplasty for treatment of class 1 and 2 obesity (MERIT): randomised trial. *Lancet*. 2022. doi:10.1016/S0140-6736(22)01280-6
5. **Sharaiha RZ, et al.** Five-year outcomes of endoscopic sleeve gastroplasty for the treatment of obesity. *Clin Gastroenterol Hepatol*. 2021. doi:10.1016/j.cgh.2020.09.055
6. **Fehervari M, et al.** Medium-term weight loss and remission of comorbidities following endoscopic sleeve gastroplasty: systematic review and meta-analysis. *Obes Surg*. 2023. doi:10.1007/s11695-023-06778-x
7. **Richter BI, et al.** Safety and efficacy of endoscopic sleeve gastroplasty after liver transplantation. *iGIE*. 2025. doi:10.1016/j.igie.2025.03.013
8. **Abufarhaneh E, et al.** Endoscopic sleeve gastroplasty after solid organ transplantation: first reported case worldwide in a kidney transplant recipient. *ACG Case Rep J*. 2026. doi:10.14309/crj.0000000000002170
9. **Ghanem O, et al.** Obesity, organ failure, and transplantation: role of metabolic and bariatric surgery in transplant candidates and recipients. *Am J Transplant*. 2024. doi:10.1016/j.ajt.2024.04.013

Yael Sofer, MD
Institute for Endocrinology, Metabolism and Hypertension
Tel Aviv Sourasky Medical Center · Gray Faculty of Medical and Health Sciences, Tel Aviv University
Scan for author profile · tasmc.org.il

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