Section: Urology

Case Series

MINIMALLY INVASIVE BILATERAL ADRENALECTOMY FOR REFRACTORY HYPERCORTISOLISM: A SINGLE-CENTRE CASE SERIES OF FIVE PATIENTS WITH NARRATIVE REVIEW

Manish Gupta[1], Girraj Prasad Sharma[2], Vinayak Onkarka Jindal[3]

[1]Professor & Unit Head, Department of Urology, Mahatma Gandhi Hospital & Medical College, Sitapura, Jaipur, Rajasthan, India
[2]Assistant Professor, Department of Urology, Mahatma Gandhi Hospital & Medical College, Sitapura, Jaipur, Rajasthan, India
[3]Final Year MBBS student, Mahatma Gandhi Hospital & Medical College, Sitapura, Jaipur, Rajasthan, India

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Corresponding Author:
Dr. Girraj Prasad Sharma,
Assistant Professor, Department of Urology, Mahatma Gandhi Hospital & Medical College, Sitapura, Jaipur, Rajasthan, India.
Email: girrajsharmajpr@gmail.com

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ABSTRACT

Background: Refractory hypercortisolism, whether driven by an occult or unresectable ectopic adrenocorticotropic hormone (ACTH) source, persistent Cushing's disease, or primary bilateral adrenal disease, carries substantial metabolic, infectious, and thromboembolic risk. Bilateral adrenalectomy (BLA) offers definitive endocrine control when medical therapy and targeted resection fail. We describe our institutional experience with minimally invasive BLA for refractory hypercortisolism across three distinct aetiologies.

Materials and Methods: This is a retrospective single-centre case series of five consecutive adult patients (median age 50 years; three women) who underwent laparoscopic or robotic-assisted BLA for refractory hypercortisolism. Perioperative management followed an institutional enhanced recovery after surgery (ERAS) protocol. Demographic, biochemical, radiological, operative, and follow-up data were extracted from the electronic record.

Results: Three patients had ectopic ACTH syndrome (EAS): two with primary somatostatin-receptor–avid bronchial lesions and one with an occult source that was subsequently unmasked as a bronchial carcinoid on surveillance imaging eight months post-adrenalectomy. One patient had primary bilateral macronodular adrenal hyperplasia and one had persistent Cushing's disease following endoscopic transsphenoidal adenomectomy. Four of five patients achieved immediate biochemical control and were discharged within 3–8 days; one patient with preoperative acute necrotising pancreatitis, symptomatic hypokalaemia, and massive hypercortisolaemia (ACTH >2,000 pg/mL) died of postoperative septic shock. No patient developed clinically evident Nelson-like ACTH escape during follow-up.

Conclusion: Minimally invasive BLA delivers rapid, definitive endocrine control across the spectrum of refractory hypercortisolism and integrates well with ERAS pathways. Preoperative physiological optimisation, aggressive infection control, and lifelong surveillance for an evolving primary neuroendocrine neoplasm are essential to translate the immediate metabolic benefit into durable clinical gain.

Keywords: bilateral adrenalectomy; ectopic ACTH syndrome; Cushing's syndrome; laparoscopic surgery; robotic surgery; hypercortisolism; neuroendocrine tumour.

INTRODUCTION

Endogenous Cushing's syndrome (CS) is a rare but potentially lethal state of chronic glucocorticoid excess with an estimated incidence of 0.7–2.4 per million person-years and a standardised mortality ratio of 1.5–4.8 when the syndrome is left uncontrolled.[1,2] Approximately 80–85% of

endogenous cases are adrenocorticotropic hormone (ACTH)-dependent; among these, pituitary adenomas (Cushing's disease, CD) account for roughly 70%, ectopic ACTH syndrome (EAS) for 10–20%, and ectopic corticotropin-releasing hormone secretion for <1%.[1–3]

EAS arises from non-pituitary neuroendocrine neoplasms, most commonly small-cell lung carcinoma, bronchial and thymic carcinoids, medullary thyroid carcinoma, phaeochromocytoma, and pancreatic neuroendocrine tumours.[3,4] The primary tumour is not identifiable at presentation in 12–19% of cases, and localisation may require years of serial imaging, often assisted by somatostatin-receptor–targeted positron emission tomography (SSTR-PET/CT) using ^{68}Ga-DOTA peptides.[4,5] Regardless of the underlying aetiology, sustained cortisol excess drives a cascade of metabolic, cardiovascular, thromboembolic, psychiatric, and infectious complications that can overwhelm the patient within weeks.[4,6]

The optimal management of ACTH-dependent CS is resection of the primary lesion — transsphenoidal adenomectomy for CD and complete extirpation of the ectopic source for EAS.[3] When the source is occult, metastatic, or the disease persists after primary surgery, and when medical therapy with steroidogenesis inhibitors is inadequate or poorly tolerated, bilateral adrenalectomy (BLA) offers immediate and definitive endocrine control by eliminating the peripheral target of ACTH stimulation.[5–7] Modern laparoscopic and robotic-assisted approaches have replaced the historical open technique in most centres and are associated with shorter hospitalisation, reduced wound morbidity, and earlier recovery — advantages that are particularly valuable in an immunocompromised, catabolic patient population.[7–9]

The literature on BLA in this setting comprises predominantly single-centre series, and the case mix is heterogeneous.[3,7–10] We describe the perioperative course and medium-term outcomes of five consecutive patients who underwent minimally invasive BLA for refractory hypercortisolism at our institution, encompassing three distinct aetiologies — confirmed EAS, occult EAS, primary bilateral macronodular adrenal hyperplasia (PBMAH), and persistent CD — and place our observations within the contemporary literature.

MATERIALS AND METHODS

Study design and patients: This is a retrospective, single-centre, descriptive case series. Consecutive adult (≥18 years) patients who underwent minimally invasive (laparoscopic or robotic-assisted) BLA for refractory hypercortisolism at our institution between [start date] and [end date] were identified from the departmental operative registry. "Refractory" was defined a priori as (i) biochemically confirmed hypercortisolism with an inadequate response to, or intolerance of, medical therapy; and (ii) either an unresectable, occult, or postoperatively persistent primary lesion, or a primary bilateral adrenal disease not amenable to unilateral resection. Patients who underwent unilateral adrenalectomy or open BLA were excluded. Follow-up was censored at the last outpatient contact before manuscript preparation.

Preoperative work-up: All patients underwent standardised biochemical confirmation of hypercortisolism (24-hour urinary free cortisol, late-night salivary or serum cortisol, and low-dose dexamethasone suppression test), followed by determination of ACTH dependence, high-dose dexamethasone suppression testing (HDDST), and, where indicated, bilateral inferior petrosal sinus sampling (IPSS). Anatomical imaging comprised contrast-enhanced computed tomography (CT) of the chest, abdomen, and pelvis; magnetic resonance imaging (MRI) of the sella was obtained in all ACTH-dependent cases. Functional imaging with ^{68}Ga-DOTATOC or ^{68}Ga-DOTANOC PET/CT was used to interrogate for occult neuroendocrine sources. Medical stabilisation with steroidogenesis inhibitors (ketoconazole and/or metyrapone), potassium replacement, glycaemic control, thromboprophylaxis, and treatment of active infections was pursued before surgery.

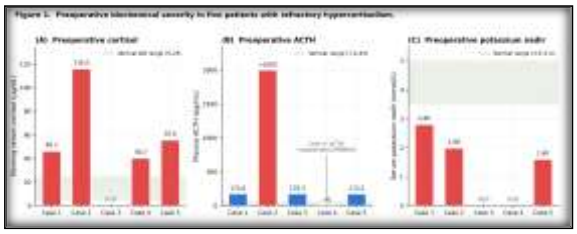
Surgical technique and perioperative management: BLA was performed in a single anaesthetic sitting in all patients. The lateral transperitoneal approach was used for laparoscopic cases; robotic-assisted BLA was performed using the da Vinci Xi platform with the patient repositioned between sides. Adrenal veins were controlled with polymer clips or an energy device, and specimens were extracted in retrieval bags. A single perinephric drain was placed on each side at the operating surgeon's discretion. Intraoperative hydrocortisone (100 mg intravenously) was administered at induction and continued as physiological replacement postoperatively. Patients were managed on an institutional ERAS pathway that included early enteral nutrition, opioid-sparing analgesia, ambulation on postoperative day (POD) 1, urinary catheter removal on POD 1, and drain removal on POD 2 when output was <50 mL/24 h and non-chylous. Discharge criteria comprised haemodynamic stability, tolerance of oral intake, adequate pain control on oral analgesia, and demonstrated competence with glucocorticoid and mineralocorticoid replacement.

Data collection and outcomes: Extracted variables included demographics, presenting features, preoperative biochemistry (morning cortisol, ACTH, HDDST response, IPSS results, 24-hour urinary free cortisol, serum potassium), imaging findings, operative details (approach, operative time, estimated blood loss, conversions, adrenal weight, histopathology), postoperative complications graded by the Clavien–Dindo system, length of stay, and short- and medium-term endocrine and oncological follow-up. The primary outcome was biochemical

control of hypercortisolism, defined as morning serum cortisol ≤5 µg/dL on physiological replacement within the first postoperative month.
All directly identifying information (names, addresses, family occupations, hospital record numbers, and calendar dates that could enable re-identification) has been removed.

RESULTS

Cohort overview: Five consecutive patients (three women, two men; median age 50 years, range 37–64) were included. Three patients (Cases 1–3) had EAS, one (Case 4) had PBMAH, and one (Case 5) had persistent CD following transsphenoidal surgery. All patients underwent minimally invasive BLA — four by conventional laparoscopy and one by robotic assistance. Baseline characteristics, operative details, and outcomes are summarised in [Table 1]. The preoperative biochemical severity of the cohort is depicted in [Figure 1].



[Figure 1] Preoperative biochemical severity across the five patients. (A) Morning serum cortisol; (B) plasma ACTH (Case 2 was capped at the assay upper limit of >2,000 pg/mL; Case 4 was ACTH-independent with a suppressed ACTH); (C) serum potassium nadir. Shaded bands indicate the normal adult reference range for each analyte; red bars indicate values outside the reference band. "N/D" indicates that the exact value was not documented in the source clinical record.

Case 1 – Ectopic ACTH syndrome with an unresectable pulmonary source

A 50-year-old man presented with a six-month history of central weight gain, facial plethora, proximal myopathy, and severe symptomatic hypokalaemia (serum potassium 2.8 mmol/L). Morning serum cortisol was 46.18 µg/dL with a plasma ACTH of 174.6 pg/mL. Cortisol failed to suppress on HDDST, consistent with an ectopic source. Contrast-enhanced CT of the chest identified a non-calcified 1.2 cm nodule in the right lower lobe that was avid on ^{68}Ga-DOTATOC PET/CT. After preoperative optimisation with ketoconazole and potassium repletion, the patient underwent laparoscopic BLA. Operative time was uneventful and blood loss was minimal. Recovery followed the ERAS pathway; the patient was discharged on POD 5 on physiological hydrocortisone and fludrocortisone replacement. At six months, serum cortisol was suppressed and hypokalaemia had resolved. Surveillance of the pulmonary nodule is ongoing.

Case 2 – Fulminant EAS complicated by acute necrotising pancreatitis

A 45-year-old man was admitted in extremis with acute necrotising pancreatitis, symptomatic hypokalaemia (1.98 mmol/L), and new-onset diabetes. Massive hypercortisolaemia was confirmed (morning serum cortisol 116 µg/dL; plasma ACTH >2,000 pg/mL). ^{68}Ga-DOTATOC PET/CT identified a somatostatin-receptor–avid nodule in the right middle lobe. Percutaneous peripancreatic pigtail drainage was performed and the patient was optimised over one month with high-dose ketoconazole, insulin, potassium replacement, and broad-spectrum antimicrobial cover. He then underwent robotic-assisted BLA to expedite endocrine control. Despite technically uncomplicated surgery, he developed progressive septic shock in the early postoperative period and died on POD 9. The clinical course underscores the extreme perioperative fragility of patients presenting with fulminant EAS and concurrent visceral catastrophe.

Case 3 – Occult EAS unmasked on postoperative surveillance

A 64-year-old woman presented with rapid-onset Cushingoid features, refractory hypertension, and biochemically confirmed ACTH-dependent hypercortisolism. HDDST failed to suppress and MRI of the sella was normal; IPSS did not demonstrate a central-to-peripheral ACTH gradient. Cross-sectional and ^{68}Ga-DOTATOC imaging did not localise a primary lesion. Given progressive metabolic deterioration, laparoscopic BLA was performed. The postoperative course was uneventful and the patient transitioned to standard replacement therapy. Serial surveillance imaging at eight months revealed an enlarging, SSTR-avid 16 × 13.7 mm left hilar nodule abutting the lingular bronchus. Endobronchial ultrasound-guided biopsy confirmed a typical bronchial carcinoid, permitting definitive oncological management. This case illustrates the well-recognised phenomenon in which BLA both stabilises the patient and, by relieving glucocorticoid-mediated suppression of tumour growth, unmasks the primary lesion.

Case 4 – Primary bilateral macronodular adrenal hyperplasia

A 54-year-old woman presented with progressive proximal myopathy, central adiposity, purple striae, and secondary hypertension. Morning serum cortisol was 40.20 µg/dL with a suppressed plasma ACTH (<5 pg/mL), consistent with ACTH-independent CS. CT of the abdomen demonstrated bilateral nodular adrenal enlargement (right adrenal mass 10 × 4.5 × 2 cm), and functional imaging excluded an ectopic source. She underwent laparoscopic BLA. Histopathology confirmed PBMAH. The postoperative course was uneventful; she was discharged on POD 8 with adequate glucocorticoid and mineralocorticoid replacement and a suppressed morning cortisol of 0.20 µg/dL, and remains symptom-free at last follow-up. She is included in the present series as an illustrative comparator,

demonstrating that a similar minimally invasive strategy is safe and effective in ACTH-independent bilateral adrenal disease.

Case 5 – Persistent Cushing's disease after transsphenoidal adenomectomy

A 37-year-old woman presented with a four-to-five-month history of oligomenorrhoea progressing to amenorrhoea, progressive proximal muscle weakness, facial and truncal hyperpigmentation, hirsutism, acne, and a non-healing left cubital fossa ulcer. She had new-onset diabetes and secondary hypertension. Biochemistry revealed severe recurrent hypokalaemia (nadir 1.6 mmol/L), basal serum cortisol repeatedly >80 µg/dL, late-night cortisol >63 µg/dL, 24-hour urinary free cortisol of 23,458 µg/24 h, and plasma ACTH up to 174.5 pg/mL. HDDST failed to suppress cortisol. Initial pituitary MRI was reported as normal; CT and PET/CT of the chest and abdomen identified a fibrocavitatory lesion and a solid right upper lobe nodule together with bulky bilateral adrenal glands, initially raising suspicion of an ectopic source. Ultrasound-guided pleural biopsy showed only inflammatory tissue.

IPSS was performed twice and demonstrated a clear central ACTH gradient. Repeat contrast-enhanced pituitary MRI identified a 7.5 × 7.5 × 6.4 mm heterogeneously hypo-enhancing right-sided microadenoma. Endoscopic transnasal transsphenoidal adenomectomy was performed, with histopathology confirming a corticotroph adenoma without atypia. Postoperatively, bronchoalveolar lavage identified acid-fast bacilli and a non-tuberculous mycobacterial pulmonary infection, and she developed hospital-acquired pneumonia. She was treated with combination antimycobacterial therapy. She subsequently developed osteonecrosis of the proximal tibia with superadded chronic osteomyelitis and pyomyositis requiring arthroscopic debridement, together with extensive right lower-limb deep vein thrombosis managed with therapeutic anticoagulation.

Two months after transsphenoidal surgery she was readmitted with recrudescent moon facies, weight gain, and poorly controlled hyperglycaemia. Reassessment revealed persistent disease with morning cortisol 50 µg/dL and a post-overnight-dexamethasone cortisol of 55.6 µg/dL. Given her life-threatening infectious burden, the poor medium-term efficacy of repeat transsphenoidal surgery in this scenario, and the need to rapidly abolish glucocorticoid-mediated immunosuppression, a joint endocrine–surgical decision was made to proceed to definitive laparoscopic BLA rather than to escalate medical therapy or consider pituitary radiotherapy. Surgery was uneventful; the urinary catheter was removed on POD 1, drains on POD 2, and the patient was discharged haemodynamically stable on POD 3 on hydrocortisone and fludrocortisone replacement. At medium-term follow-up she remains biochemically controlled with resolution of the infectious complications.

Endocrine and perioperative outcomes

Immediate biochemical control was achieved in every operated patient. The trajectory of morning serum cortisol before and after BLA is shown in Figure 2. Length of hospital stay is summarised in Figure 3. Median length of stay among survivors was 5.5 days (range 3–8); Case 2 died of septic shock on POD 9.

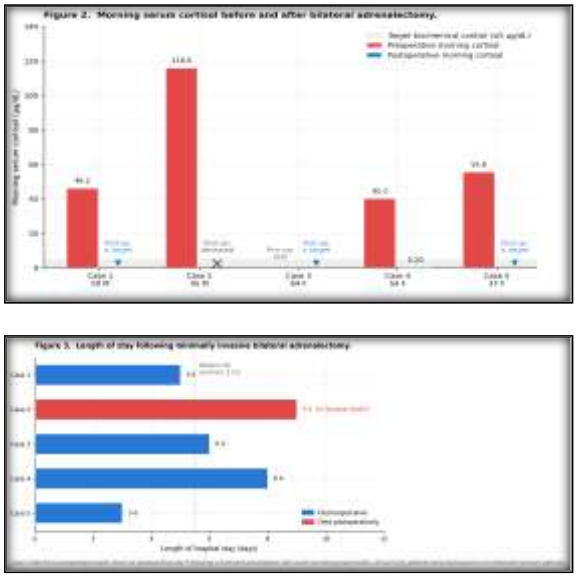
Table 1: Summary of five patients undergoing minimally invasive bilateral adrenalectomy for refractory hypercortisolism

Case	Age (y) / Sex	Aetiology	Key preoperative biochemistry	Operative approach & LOS	Outcome
1	50 / M	EAS – right lower lobe pulmonary nodule	Cortisol 46.18 µg/dL; ACTH 174.6 pg/mL; K^+ 2.8 mmol/L; HDDST non-suppressed	Laparoscopic BLA; LOS 5 d	Biochemical control; alive at last follow-up
2	45 / M	EAS – right middle lobe SSTR-avid nodule	Cortisol 116 µg/dL; ACTH >2,000 pg/mL; K^+ 1.98 mmol/L; concurrent acute necrotising pancreatitis	Robotic-assisted BLA; LOS n/a (in-hospital death)	Postoperative septic shock; death POD 9
3	64 / F	Occult EAS; bronchial carcinoid unmasked at 8 months	ACTH-dependent hypercortisolaemia; HDDST non-suppressed; IPSS non-central	Laparoscopic BLA; LOS 6 d	Biochemical control; primary tumour localised and treated
4	54 / F	PBMAH (ACTH-independent)	Morning cortisol 40.20 µg/dL; ACTH suppressed; bilateral macronodular adrenals	Laparoscopic BLA; LOS 8 d	Biochemical control (cortisol 0.20 µg/dL); asymptomatic
5	37 / F	Persistent CD after transsphenoidal adenomectomy	Post-op cortisol 55.6 µg/dL; ACTH 172.5 pg/mL; IPSS central; osteomyelitis; DVT	Laparoscopic BLA; LOS 3 d	Biochemical control; infections resolving

ACTH, adrenocorticotropic hormone; BLA, bilateral adrenalectomy; CD, Cushing's disease; DVT, deep vein thrombosis; EAS, ectopic ACTH syndrome; HDDST, high-dose dexamethasone suppression test; IPSS, inferior petrosal sinus sampling; K^+, serum potassium; LOS, length of stay; PBMAH, primary bilateral macronodular adrenal hyperplasia; POD, postoperative day; SSTR, somatostatin receptor.

[Figure 2] Morning serum cortisol before and after bilateral adrenalectomy. Discrete preoperative cortisol values are shown for four patients; Case 3 (labelled N/D) had biochemically confirmed hypercortisolism by other assays (24-hour urinary free cortisol and late-night cortisol) but no discrete

morning cortisol was recorded. Only Case 4 has a documented discrete postoperative value (0.20 µg/dL); for the remaining survivors, biochemical control (≤5 µg/dL on replacement) is indicated by a downward triangle. The shaded band marks the physiological replacement target after BLA.



[Figure 3] Length of hospital stay for each patient in the series. Median length of stay among the four surviving patients was 5.5 days. Case 2 died of postoperative septic shock on postoperative day 9 following a fulminant preoperative presentation with acute necrotising pancreatitis.

DISCUSSION

This series of five patients illustrates the contemporary role of minimally invasive BLA across the spectrum of refractory hypercortisolism. Immediate biochemical control was achieved in every operated patient, with a median length of stay of five days and no incidence of open conversion. The single mortality occurred in a patient with fulminant EAS complicated by acute necrotising pancreatitis and multi-organ physiological derangement — a clinical constellation that has previously been associated with perioperative mortality rates of 15–25%.[3,4,7] Our findings align with the reported contemporary experience of laparoscopic BLA in this population and reinforce three lessons: the need for individualised preoperative optimisation, the strategic role of BLA as a physiological "endocrine reset," and the necessity of lifelong tumour surveillance. A pragmatic decision framework that captures these considerations, consistent with contemporary guidelines and the patterns seen in our series, is presented in [Figure 4].

The pathophysiology of refractory hypercortisolism explains much of its perioperative risk profile. Sustained glucocorticoid excess induces severe insulin resistance and secondary diabetes, upregulates the renin–angiotensin–aldosterone axis, and saturates renal 11β-hydroxysteroid dehydrogenase type 2, allowing cortisol to illicitly activate the mineralocorticoid receptor and driving profound hypokalaemia.[2,4,6] The catabolic state produces proximal myopathy and osteopenia, while relative immunosuppression predisposes to opportunistic infections and disseminated venous thromboembolism.[2,6] Every element of this cascade was represented in our cohort: symptomatic hypokalaemia in Cases 1, 2 and 5; acute necrotising pancreatitis, plausibly triggered by hypertriglyceridaemia and cortisol-mediated microvascular injury, in Case 2; non-tuberculous mycobacterial pulmonary infection, chronic osteomyelitis, and extensive lower-limb deep vein thrombosis in Case 5.

The diagnostic pathway in ACTH-dependent CS is well established but remains challenging in practice.[3,4] In our cohort, HDDST and IPSS were pivotal in differentiating central from ectopic disease. Case 3 illustrates the residual uncertainty: despite negative HDDST, non-central IPSS, and initially non-localising anatomical and functional imaging, the primary lesion — a bronchial carcinoid — became apparent only after the suppressive effect of hypercortisolism on tumour growth was relieved by BLA. This phenomenon has been observed with a median time-to-localisation of 8–24 months after BLA,[4,10] reinforcing the recommendation that these patients undergo protocolised, high-resolution cross-sectional and SSTR-PET imaging at 6–12 monthly intervals indefinitely.

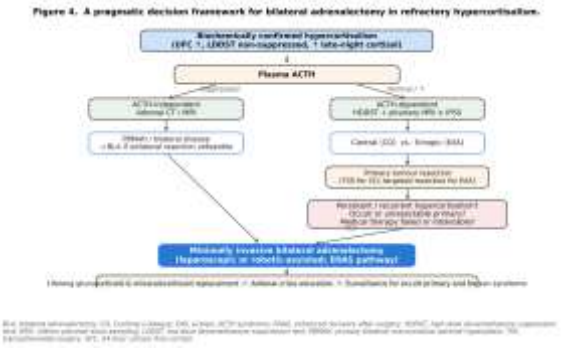
When primary tumour resection is not feasible, medical therapy with steroidogenesis inhibitors (ketoconazole, metyrapone, osilodrostat, mitotane) or with the glucocorticoid-receptor antagonist mifepristone provides valuable temporising control, but each is limited by hepatotoxicity, adrenal insufficiency, corticotroph escape, or incomplete symptomatic response.[3,5] In this context, BLA offers immediate and irrevocable elimination of the peripheral hormonal target. Meta-analytic data from contemporary series report biochemical remission in >95% of patients and perioperative mortality of 3–5% in modern minimally invasive practice, with mortality concentrated among fulminant EAS presentations,[7–10] — a pattern reflected in our single mortality.

The choice between conventional laparoscopy and robotic assistance is largely institutional. Both approaches, whether transperitoneal or retroperitoneoscopic, consistently outperform open BLA on length of stay, wound morbidity, and postoperative pulmonary complications, and both integrate seamlessly with ERAS pathways.[7–9] The rapid convalescence achieved in Cases 4 and 5 — discharge on PODs 8 and 3 respectively — is consistent with contemporary benchmarks and is of particular importance in an immunocompromised cohort, in whom the risk of nosocomial infection accumulates with each additional inpatient day.[6,7,9]

Robotic assistance in Case 2 was chosen to optimise dissection in a hostile retroperitoneum inflamed by recent pancreatitis; the fatal outcome nonetheless reflects the underlying physiological reserve rather than the operative approach.

Persistent CD after transsphenoidal surgery, illustrated by Case 5, presents its own set of decisions. Salvage options include repeat transsphenoidal exploration, fractionated or stereotactic pituitary radiotherapy, long-term medical therapy, and BLA.[3,5,10] In our patient, the combination of massive persistent hypercortisolism, active non-tuberculous mycobacterial infection, chronic osteomyelitis requiring debridement, and deep vein thrombosis on therapeutic anticoagulation made rapid, definitive endocrine control the overriding priority. BLA achieved this within days and permitted resolution of the infectious burden. The recognised risk of Nelson's syndrome following BLA in CD (historically reported in up to 20–40%, and lower in the era of routine post-BLA pituitary surveillance and prophylactic radiotherapy),[3] mandates lifelong sellar imaging and ACTH monitoring, which are ongoing in this patient.

The ACTH-independent PBMAH represented by Case 4 sits outside the classical EAS/CD dichotomy but shares two features with the ACTH-dependent cases: bilateral disease and a requirement for bilateral adrenal removal to achieve cure. Its inclusion in this series demonstrates that the same minimally invasive strategy and ERAS pathway apply equally well when the driver is intra-adrenal rather than pituitary or ectopic. In selected patients with PBMAH, cortisol-sparing subtotal or unilateral adrenalectomy is an alternative that avoids lifelong replacement, but the marked bilateral gland enlargement and severe hypercortisolism in this patient favoured a definitive bilateral approach.



[Figure 4] A pragmatic decision framework for the use of bilateral adrenalectomy in refractory hypercortisolism, synthesised from contemporary guidelines and the patterns illustrated in the present series. Bilateral adrenalectomy is triggered from either arm when primary tumour control is not feasible or the disease persists despite optimised primary and medical therapy.

Several limitations warrant mention. This is a small retrospective series from a single centre, precluding formal comparative analysis and introducing selection bias. Follow-up duration is heterogeneous, so we cannot report on long-term rates of Nelson's syndrome, adrenal crisis, mortality, or quality of life. Assessment of the operative approach is confounded by case selection, and we have not reported detailed ERAS-compliance metrics or patient-reported outcomes. Notwithstanding these limitations, the series adds granular clinical detail to a literature dominated by aggregated cohorts and illustrates decision-making at the interface of endocrinology and minimally invasive surgery.

CONCLUSION

Minimally invasive BLA delivers immediate and durable biochemical control across the spectrum of refractory hypercortisolism, whether the underlying driver is ectopic, pituitary, or primary adrenal. When embedded within an ERAS pathway, laparoscopic and robotic-assisted approaches enable rapid convalescence and mitigate the accumulating nosocomial risk that threatens this immunocompromised cohort. Preoperative physiological optimisation, aggressive treatment of infection and thromboembolism, meticulous perioperative glucocorticoid replacement, and lifelong surveillance for an evolving primary neuroendocrine lesion together determine whether the immediate metabolic gain translates into durable clinical benefit. Adequately powered multicentre studies are needed to refine patient selection, benchmark long-term outcomes, and clarify the role of robotic assistance in the highest-risk presentations.

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