Case Series

Mitigating the Catecholamine Surges: A Case Series on Intraoperative Anaesthetic Management of Resistant Pheochromocytoma

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ABSTRACT

Background and Aims: Surgical excision of pheochromocytoma is associated with exaggerated catecholamine surges that may lead to life-threatening haemodynamic instability. Resistant pheochromocytoma, characterised by intraoperative hypertension despite adequate preoperative preparation, poses additional anaesthetic challenges. We present our institutional experience in managing such patient

Methods: We retrospectively analysed 20 patients who underwent adrenalectomy for pheochromocytoma at Sher-i-Kashmir Institute of Medical Sciences, Soura, Srinagar, from October 2023 to March 2025. Among them, 6 patients developed resistant hypertension intraoperatively. Details of anaesthetic technique, drug infusions, haemodynamic events, and postoperative outcomes were reviewed.

Results: All patients were preoperatively optimised with alpha- and beta-blockade. Intraoperatively, all 6 resistant cases required simultaneous infusions of nitroglycerine, esmolol, labetalol, lignocaine, and sodium nitroprusside (0.5–5 µg/kg/min) to control hypertension during tumour manipulation. Two patients developed severe hypotension after tumour ligation requiring aggressive fluid therapy, phenylephrine boluses/infusion, and vasopressin infusion. No patient developed arrhythmias, myocardial ischaemia, or cerebrovascular complications. All were shifted to ICU for elective ventilation and recovered uneventfully

Conclusion: Resistant pheochromocytoma presents formidable intraoperative challenges. Anticipation, vigilant monitoring, and proactive use of multidrug infusion protocols—including SNP—ensure haemodynamic stability and favourable outcomes.

Keywords: Pheochromocytoma, resistant hypertension, catecholamine surge, anaesthetic management, adrenalectomy, sodium nitroprusside

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INTRODUCTION

Pheochromocytomas are rare catecholamine-secreting tumours that typically present with paroxysmal hypertension, headache, and palpitations. Surgical excision is the definitive treatment but carries significant anaesthetic challenges due to catecholamine surges during tumour manipulation.[1,2]

With improved pharmacological preparation, perioperative mortality has reduced from nearly 40–45% in the pre-alpha blockade era to <3% in modern practice. However, resistant pheochromocytomas, where hypertensive crises persist despite adequate blockade, continue to test anaesthesiologists.[3,4]

The present case series highlights our institutional experience in managing such resistant cases. The objective was to describe intraoperative anaesthetic strategies to mitigate catecholamine surges and ensure haemodynamic stability, in keeping with established institutional and multidisciplinary protocols for the anaesthetic management of this tumour.[5]

MATERIAL AND METHODS

Study design

This retrospective observational case series was conducted in the Department of Anaesthesiology, Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Soura, Srinagar, from October 2023 to March 2025.

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Study population and case selection

Records of 20 patients who underwent adrenalectomy for pheochromocytoma were reviewed. Six patients who developed resistant intraoperative hypertension were included.

Definition

Resistant intraoperative hypertension was defined a priori as systolic blood pressure >200 mmHg occurring despite adequate volatile anaesthesia, opioid supplementation, and bolus antihypertensive therapy. No single, universally accepted definition of intraoperative haemodynamic instability exists in the pheochromocytoma literature. Some authors have used a lower threshold of systolic blood pressure >160 mmHg to define an unstable episode,[6] others have defined instability chiefly by the requirement for cardioactive or vasoactive drug administration irrespective of the absolute pressure recorded,[7] and more recent series have combined both the pressure threshold and the requirement for vasoactive support into a single composite definition.[8] We deliberately adopted a higher, more stringent cut-off of >200 mmHg to isolate a subgroup with unequivocally severe, treatment-refractory hypertension rather than the broader and more heterogeneous population of intraoperative hypertensive episodes captured by lower thresholds, so that the pharmacological strategies described here could be interpreted specifically in the context of genuinely resistant disease.

Preoperative preparation

In accordance with the 2014 Endocrine Society clinical practice guideline,[9] alpha blockade with phenoxybenzamine or prazosin was instituted for 10-14 days before beta blockade with propranolol or metoprolol was added to control reflex tachycardia; beta-blockade was never commenced first, since unopposed alpha-adrenergic vasoconstriction from isolated beta-blockade can itself precipitate a hypertensive crisis.[10] Adequacy of alpha blockade was judged clinically by resolution of paroxysmal symptoms, orthostatic blood pressure reduction, and absence of ST-T changes on the electrocardiogram, together with a liberal salt and fluid intake and intravenous fluid loading in the 24 hours before surgery to counter the chronic intravascular volume contraction caused by long-standing catecholamine excess.

Anaesthetic technique and monitoring

Standard ASA monitoring was supplemented with invasive arterial blood pressure and central venous pressure monitoring. Induction consisted of glycopyrrolate 5 µg/kg, fentanyl 1.5 µg/kg, lignocaine 1 mg/kg, propofol 2 mg/kg, and rocuronium 0.6 mg/kg. Maintenance used an oxygen-air mixture, sevoflurane or isoflurane, and opioids.

Management of resistant hypertension

Nitroglycerine 0.5–2 µg/kg/min, esmolol 50–300 µg/kg/min, labetalol 1–2 mg/min, and sodium nitroprusside 0.5–5 µg/kg/min were used as titratable infusions, selected for their short onset and offset of action to allow rapid up- and down-titration against a rapidly changing haemodynamic target. Lignocaine was administered as a bolus followed by infusion for its membrane-stabilising, sympatholytic adjunctive effect. Escalation followed a stepwise protocol: a vasodilator infusion was started first, esmolol or labetalol was added for persistent tachycardia, and sodium nitroprusside was reserved for pressures refractory to the above, reflecting evidence that larger tumour size and the type of alpha-blockade used independently predict the severity and multidrug nature of intraoperative hypertensive episodes.[11]

Post-ligation hypotension

Management consisted of intravenous fluids, phenylephrine boluses or infusion, and vasopressin 0.01–0.04 U/min when required. Vasopressin was favoured over escalating catecholamine vasopressors when hypotension proved refractory, since it acts on V1 receptors independent of the adrenoceptors that become downregulated after prolonged catecholamine excess, and has been reported to reverse similarly refractory post-resection hypotension when adrenergic agents alone were insufficient.[13]

Postoperative care and outcomes

All resistant cases were transferred to ICU for elective postoperative ventilation and haemodynamic observation. Outcomes reviewed included vasoactive drug requirements, post-ligation hypotension, arrhythmia, myocardial ischaemia, cerebrovascular complications, ICU disposition, and recovery.

Ethics and consent

Appropriate patient consent was obtained. Ethical

certificate was not required as per institutional protocol for this case series.

Table 1. Study population and case definition

Characteristic	Details
Total adrenalectomies	20
Resistant cases	6 (30%)
Definition	SBP >200 mmHg despite adequate anaesthesia, opioid supplementation and bolus antihypertensives
Design	Retrospective observational case series
Period	October 2023–March 2025

Table 2. Perioperative pharmacological management.

Drug / strategy	Dose or approach
Alpha blockade	Phenoxybenzamine or prazosin before beta blockade
Beta blockade	Propranolol or metoprolol
Nitroglycerine	0.5–2 µg/kg/min
Esmolol	50–300 µg/kg/min
Labetalol	1–2 mg/min
Lignocaine	Bolus followed by infusion
SNP	0.5–5 µg/kg/min
Phenylephrine	Bolus/infusion
Vasopressin	0.01–0.04 U/min

Table 3. Clinical outcomes in resistant cases.

Outcome	n/N
Multidrug antihypertensive therapy	6/6
Severe post-ligation hypotension	2/6
Arrhythmia	0/6
Myocardial ischaemia	0/6
Cerebrovascular complications	0/6
ICU transfer with elective ventilation	6/6
Uneventful recovery	6/6

RESULTS

A total of 20 patients underwent adrenalectomy for pheochromocytoma during the study period. Six patients (30%) developed resistant intraoperative hypertension according to the predefined criterion of systolic blood pressure >200 mmHg despite adequate volatile anaesthesia, opioid supplementation, and bolus antihypertensive therapy. All six patients had received preoperative alpha-adrenergic blockade followed by beta-adrenergic blockade, together with salt and fluid loading, before surgery. Standardised induction and maintenance techniques were used, with invasive arterial and central venous pressure monitoring. During tumour manipulation, resistant hypertensive episodes required a multimodal pharmacological approach. All six patients required simultaneous infusions of nitroglycerine, esmolol, labetalol, lignocaine, and sodium nitroprusside (SNP). Nitroglycerine was administered at 0.5–2 µg/kg/min, esmolol at 50–300 µg/kg/min, labetalol at 1–2 mg/min, and SNP at 0.5–5 µg/kg/min. Lignocaine was administered as a bolus followed by infusion. The combination was used to achieve rapid control of marked blood pressure elevations during periods of tumour handling. Following tumour ligation and removal, two of the six patients developed severe hypotension. These episodes were managed with intravenous fluid resuscitation, phenylephrine boluses or infusion, and vasopressin infusion at 0.01–0.04 U/min. Both patients responded to this strategy without documented persistent haemodynamic compromise. No patient in the resistant group developed intraoperative arrhythmia, myocardial ischaemia, or cerebrovascular complications. Following surgery, all six patients were transferred to the intensive care unit for elective postoperative ventilation and haemodynamic observation. All patients recovered uneventfully. No postoperative major complication or mortality was reported in the case series. The resistant hypertensive episodes occurred particularly during tumour manipulation and required repeated titration according to the immediate haemodynamic response. Continuous invasive blood pressure monitoring was maintained throughout these periods. The transition after tumour ligation was managed as a separate haemodynamic phase, with early anticipation of vasodilation and prompt escalation of circulatory support when hypotension occurred. Despite these marked haemodynamic fluctuations, all patients completed the planned surgical procedure. No patient required reoperation, prolonged postoperative haemodynamic support, or experienced perioperative death. The requirement for concurrent vasodilator, beta-adrenergic, and adjunctive therapy indicates the severity of the hypertensive response in this selected subgroup. Following stabilisation, all six patients were transferred to the intensive care unit for elective ventilation and close postoperative observation. Recovery was uneventful in every patient.

DISCUSSION

Pheochromocytoma remains a demanding condition for the anaesthesiologist because manipulation of a catecholamine-secreting tumour can produce abrupt and

potentially severe haemodynamic changes. Although contemporary preoperative alpha-adrenergic blockade followed by beta blockade has substantially improved perioperative outcomes, adequate preparation does not completely eliminate intraoperative hypertensive crises. In the present case series, 6 of 20 patients developed resistant intraoperative hypertension despite preoperative optimisation and appropriate anaesthetic depth. This finding emphasises that preparation should extend beyond routine blockade to anticipation of catecholamine surges and immediate availability of titratable vasoactive drugs. The principal observation in our series was that resistant hypertension could not be controlled reliably with a single agent. All six patients required a combination of nitroglycerine, esmolol, labetalol, lignocaine, and sodium nitroprusside during tumour manipulation. The use of drugs with complementary mechanisms provided the flexibility required to address both the magnitude and the rapidly changing pattern of the hypertensive response. Beta-adrenergic blockade helped attenuate tachycardic and adrenergically mediated responses, whereas vasodilator therapy provided more immediate reduction of systemic vascular resistance. Lignocaine was used as an additional adjunct to blunt sympathetic responses. Sodium nitroprusside was particularly useful when rapid control of severe hypertension was required. Its short onset and offset of action permit rapid titration during periods of intense tumour manipulation. In our patients, SNP was used as part of a multidrug strategy rather than as isolated therapy. This approach is clinically relevant because catecholamine release during pheochromocytoma surgery can be abrupt, making prolonged or poorly titratable antihypertensive therapy less desirable. Our observations are also consistent with previous reports describing the importance of meticulous haemodynamic control and individualized combinations of vasoactive agents during pheochromocytoma surgery, and with two decades of accumulated evidence that no single vasoactive agent reliably controls the full spectrum of intraoperative haemodynamic swings seen in these patients.[12] Another important finding was the occurrence of severe hypotension immediately after tumour ligation in two patients. This transition from marked vasoconstriction to profound vasodilation is a well-recognised challenge after removal of the catecholamine source. Chronic catecholamine exposure may also leave patients relatively depleted of intravascular volume and less responsive to conventional catecholamine vasopressors. In our series, hypotension was managed with fluid resuscitation, phenylephrine, and vasopressin, with successful haemodynamic recovery. The response observed with vasopressin supports its role as an important rescue or adjunctive vasopressor when hypotension is difficult to control after tumour removal. Prompt correction of post-resection hypotension is not merely a matter of haemodynamic comfort: sustained reductions in mean arterial pressure after tumour resection have been independently associated with postoperative acute kidney injury, which reinforces our practice of early, proactive vasopressor escalation rather than a permissive approach to post-ligation hypotension.[14] Nevertheless, the choice and sequence of vasoactive therapy should be individualised according to the clinical response and institutional protocols. The absence of arrhythmia, myocardial ischaemia, or cerebrovascular complications in our resistant cases is encouraging, although it should not be interpreted as evidence that resistant hypertension is benign. Severe intraoperative hypertension may increase the risk of myocardial injury, arrhythmia, cerebrovascular events, and other end-organ complications. The favourable outcome in our series may reflect the combined effects of preoperative optimisation, invasive monitoring, early recognition of haemodynamic surges, rapid availability of multiple infusion agents, and close postoperative observation. Our findings also highlight the importance of preparation before induction. Arterial and central venous monitoring were established to facilitate continuous assessment and rapid treatment. Vasoactive infusions should be prepared and immediately available before tumour manipulation rather than introduced only after severe hypertension has developed. Communication between the anaesthesia and surgical teams is equally important, particularly before manipulation, venous ligation, and tumour removal, because these stages may produce predictable but rapid haemodynamic transitions. The present study has limitations. It is a retrospective case series from a single institution and includes only six resistant cases. There was no control group, and the study was not designed to compare the efficacy of individual vasoactive drugs. Detailed quantitative haemodynamic trends and drug-response relationships were not available for formal statistical

analysis. Therefore, the findings should be interpreted as an institutional experience that may help inform perioperative planning rather than as evidence establishing superiority of one pharmacological regimen. Despite these limitations, the series demonstrates a practical approach to a particularly challenging subset of pheochromocytoma patients. Resistant intraoperative hypertension requires anticipation, invasive monitoring, adequate anaesthetic depth, and a prepared multimodal infusion strategy. Equally important is readiness for abrupt post-ligation hypotension, including early fluid therapy and availability of vasopressors such as phenylephrine and vasopressin. A structured perioperative protocol and elective postoperative intensive care monitoring may contribute to safe outcomes in patients undergoing adrenalectomy for resistant pheochromocytoma. Future prospective studies involving larger cohorts could evaluate the incidence and predictors of resistant intraoperative hypertension and compare different pharmacological strategies using predefined haemodynamic endpoints. Standardised recording of blood pressure excursions, cumulative drug doses, duration of infusion, estimated blood loss, and postoperative vasopressor requirements would allow more meaningful comparisons between protocols. Until such evidence is available, management should remain individualised and based on careful preparation, rapid titration of short-acting agents, and anticipation of both hypertensive and hypotensive phases. The favourable postoperative course in this series further supports planned ICU admission for resistant cases.

CONCLUSION

Resistant pheochromocytoma poses unique intraoperative anaesthetic challenges. Successful management requires:

- Preoperative optimisation with alpha and beta blockad
- Invasive monitoring and vigilance
- Proactive use of multiple antihypertensive infusions including SNP
- Preparedness for post-ligation hypotension with vasopressors including vasopressin
- Elective postoperative ICU monitoring

A structured protocol ensures haemodynamic stability and safe surgical outcomes.

DECLARATION

Patient consent: Appropriate consent obtained.

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Conflicts of interest: None declared.

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