

Case Series: Intraoperative Anaphylaxis and Preventive Measures in Hydatid Cyst Surgery

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Abstract

Background:

Hydatid disease caused by *Echinococcus granulosus* remains a considerable surgical and anesthetic challenge in endemic areas. One of the most feared intraoperative complications is anaphylaxis due to rupture or spillage of cyst contents, which can rapidly progress to Cardiovascular collapse and even death. Prompt recognition, timely intervention, and preventive strategies are essential for safe patient outcomes.

Case Series:

We report three cases of hepatic hydatid cyst surgery that illustrate the varying severity of intraoperative reactions. The first case involved a 50-year-old female with a large right lobe cyst who developed fulminant anaphylaxis characterized by severe hypotension, tachycardia, desaturation, and bronchospasm. She required aggressive resuscitation with adrenaline boluses, infusion, corticosteroids, inotropes, and elective postoperative ventilation before making a complete recovery. The second case involved a 60-year-old male with a multiloculated cyst of the right hepatic lobe who experienced transient intraoperative hypotension during cyst aspiration. His blood pressure responded promptly to intravenous fluids, phenylephrine, and hydrocortisone, and he remained stable with an uneventful postoperative course. The third case was a 45-year-old female with a unilocular cyst in the left lobe of the liver. She received preoperative albendazole, perioperative steroid and antihistamine prophylaxis, and surgery was performed with meticulous preventive measures, including controlled aspiration and scolicidal irrigation. No intraoperative reaction occurred, and recovery was smooth.

Conclusion:

This case series demonstrates the unpredictable spectrum of anaphylaxis in hepatic hydatid surgery. Preparedness with adrenaline and resuscitation protocols, combined with prophylaxis and careful surgical technique, remain the cornerstones of anesthetic management.

Keywords: Hydatid cyst, hepatic hydatidosis, intraoperative anaphylaxis, anesthesia, preventive strategies

INTRODUCTION

Hydatid disease, also known as echinococcosis, is a parasitic zoonosis caused predominantly by the larval form of *Echinococcus granulosus*. It continues to be a significant global health problem, especially in developing and endemic regions. The liver is the most common site of involvement, followed by the lungs, but virtually any organ can be affected. The disease often remains asymptomatic until cysts reach a considerable size or rupture, producing complications. Among the most feared intraoperative complications, anaphylaxis, poses a life-threatening challenge during surgical management of hydatid cysts [1].

The potential for intraoperative anaphylaxis has long been recognized in surgical literature. Sola et al. documented a case where intraoperative rupture of a hydatid cyst resulted in severe anaphylaxis, demonstrating how an otherwise straightforward operation can be rapidly

deteriorated into a critical event if cyst contents spilled into the systemic circulation [1]. The underlying mechanism involves the release of highly antigenic hydatid fluid, which contains scolices, hooklets, and various parasitic proteins. These antigens can trigger an immediate immunoglobulin E (IgE)-mediated hypersensitivity response, leading to histamine release, mast cell degranulation, and systemic circulatory collapse.

An early landmark observation was reported by Jakubowski and Bernard, who described an intraoperative case of anaphylactic shock occurring during hydatid surgery [2]. Their report highlighted that even when surgical and anesthetic protocols were carefully followed, cyst rupture could provoke sudden, severe, and unpredictable hypersensitivity reactions. The patient in their series developed abrupt hypotension and cardiovascular collapse, necessitating rapid resuscitation with fluids, vasopressors, and airway control [2]. This established that anesthesiologists should always anticipate such rare but catastrophic scenarios when managing hydatid cyst patients.

From an anesthesiologist's perspective, allergic and anaphylactic reactions represent some of the most serious intraoperative emergencies. A large European review by Mertes and Laxenaire showed that perioperative allergic reactions, although relatively rare, carry a high burden of morbidity and mortality [3]. Their analysis revealed that reactions could be triggered by a wide variety of agents, including antibiotics, neuromuscular blocking drugs, and latex.

Importantly, in surgeries for hydatid disease, the additional risk factor of hydatid fluid spillage complicates the perioperative landscape [3]. This creates a dual responsibility for anesthesiologists: not only must they guard against common anesthetic allergens, but they must also be vigilant for parasitic antigens introduced into circulation during cyst manipulation.

Fisher and Baldo further emphasized the diagnostic and preventive challenges of anaphylaxis during anesthesia [4]. They noted that perioperative anaphylaxis is often misdiagnosed initially, as hypotension and tachycardia can also result from blood loss, anesthetic depth, or other intraoperative events. The absence of typical skin manifestations due to surgical draping compounds this diagnostic difficulty. In the context of hydatid surgery, the sudden onset of cardiovascular instability without another obvious explanation should prompt immediate suspicion of anaphylaxis [4]. Their work underscores the necessity of preparing adrenaline, corticosteroids, and antihistamines in advance, particularly when operating on large or multiloculated hepatic cysts where rupture is a foreseeable risk.

Surgical approach also influences the likelihood of cyst rupture and subsequent antigen release. Khoury et al. described a striking case of anaphylactic shock during laparoscopic management of a hepatic hydatid cyst [5]. Despite the minimally invasive approach, leakage of cyst fluid into the peritoneal cavity triggered a full systemic reaction. The pneumoperitoneum of laparoscopy may accelerate the systemic absorption of antigens, increasing the severity of anaphylaxis [5]. This demonstrates that while laparoscopic techniques reduce postoperative pain and recovery time, they do not eliminate the risk of life-threatening intraoperative hypersensitivity.

Spontaneous systemic anaphylaxis caused by hydatid disease has also been documented, even outside the operating room. Boyano et al. reported systemic anaphylaxis in hepatic hydatid disease, suggesting that even minor leakage of cyst fluid can sensitize the host immune system [6]. Such reports highlight the immunological activity of hydatid cysts and demonstrate that patients presenting for surgery may already have an altered immune status.

For anesthesiologists, this indicates that intraoperative reactions may be more severe in previously sensitized individuals [6].

Pulmonary hydatid disease adds another layer of complexity. Shameem et al. described a case of ruptured pulmonary hydatid cyst complicated by both anaphylactic shock and pneumothorax [7]. The combined effect of acute bronchospasm, airway compromise, and circulatory collapse illustrates the wide spectrum of clinical manifestations anesthesiologists must anticipate [7]. Although this present case series focuses on hepatic hydatid cysts, such reports show that echinococcosis has multisystemic consequences and that perioperative teams must be prepared for unpredictable airway

and circulatory crises. Notably, hydatid cyst rupture can occur spontaneously, leading to emergency presentations.

Tonnelet et al. described a case where spontaneous rupture of a cyst produced fulminant

anaphylactic shock [8]. Such emergencies are particularly dangerous, as patients may present to the operating room already unstable. In these circumstances, prompt airway management, high-flow oxygen, large-volume crystalloids, and immediate adrenaline administration are lifesaving [8]. This illustrates the continuum of risk across both elective and emergency surgical settings. The most extreme outcomes include sudden unexpected death from unrecognized hydatid cyst rupture. Malamou-Mitsi et al. reported a case of sudden death caused by an unrecognized cardiac hydatid cyst [9]. Although cardiac involvement is rare, it highlights that hydatid cysts can occur in atypical locations and that their rupture may cause catastrophic consequences through both anaphylaxis and direct organ damage [9]. This underlines the importance of thorough preoperative imaging, careful surgical planning, and anesthetic preparedness for rare but lethal complications.

Rationale for the Case Series

Despite decades of case reports and reviews, intraoperative anaphylaxis during hydatid cyst surgery remains a major perioperative hazard. The clinical spectrum ranges from mild hypersensitivity to profound cardiovascular collapse, with mortality risk if diagnosis and treatment are delayed. The lessons from published cases emphasize the importance of early recognition, aggressive management with adrenaline, and preventive measures including preoperative albendazole therapy, intraoperative prophylaxis with steroids and antihistamines, and meticulous surgical technique to avoid spillage.

The present case series adds to the literature by describing three contrasting intraoperative experiences with hepatic hydatid cyst surgery: one case with fulminant anaphylaxis, another with transient instability, and a third managed successfully without hypersensitivity due to preventive measures. By highlighting these different clinical trajectories, we aim to reinforce the importance of vigilance, preparedness, and preventive strategies for anesthesiologists and surgeons managing hydatid disease.

Case Presentations Case 1

A 50-year-old woman was admitted following the incidental discovery of a hepatic hydatid cyst during a master health check-up. She had previously undergone excision of a hydatid cyst in 2005, which had been uneventful. In preparation for surgery, she received albendazole 400 mg twice daily for two weeks. On preoperative evaluation she was thin built, with no

comorbid illnesses. Laboratory investigations revealed hemoglobin of 12.4 g/dL, packed cell volume of 38%, and platelet count of 1.98×10^5 /cmm. Renal and hepatic function tests were within normal limits. Electrocardiogram and chest radiograph were normal. Contrast-enhanced computed tomography (CECT) of the abdomen demonstrated a multiloculated cyst measuring 15×11 cm in the right hepatic lobe, with a calcified wall.

An epidural catheter was inserted at the T10-T11 interspace before induction. Premedication included intravenous glycopyrrolate 0.2 mg, fentanyl 2 mcg/kg, and ranitidine 50 mg.

General anesthesia was induced with thiopentone 5 mg/kg and atracurium 0.5 mg/kg. The trachea was intubated with a cuffed 7.5 mm endotracheal tube. Anesthesia was maintained with sevoflurane at 0.5 to 1MAC in a nitrous oxide-oxygen mixture (2:1), supplemented with intermittent epidural boluses of 0.25% bupivacaine. Standard monitoring included non-invasive blood pressure, electrocardiography, pulse oximetry, and urine output.

Approximately 80 minutes after commencement of surgery, during cyst excision, the patient developed sudden hypotension (blood pressure 60/40 mmHg), tachycardia (132 beats per minute), hypoxemia (SpO₂ 88%), and pulmonary crepitations. Blood loss at the time was less than 200 ml and electrocardiographic monitoring showed no ischemic changes. Intraoperative anaphylaxis was suspected. Immediate resuscitative measures included rapid intravenous crystalloids, ephedrine 6mg bolus twice (12 mg), and hydrocortisone 200 mg, but without significant effect. Adrenaline 100 mcg intravenous boluses were administered twice, resulting in partial stabilization of blood pressure. A continuous adrenaline infusion at 0.5 mcg/kg/min was commenced, along with methylprednisolone 125 mg and an

aminophylline infusion at 5 mcg/kg/min for associated bronchospasm.

The patient was electively ventilated in the intensive care unit. She was gradually weaned off inotropes within six hours and subsequently extubated. She recovered uneventfully and was discharged to the surgical ward on the fourth postoperative day.

Case 2

A 60-year-old male patient, with a history of hypertension on regular medication and chronic alcohol consumption for 25 years, presented with right hypochondrial pain persisting for six months. Preoperative assessment revealed no significant cardiovascular or respiratory abnormalities. Airway examination indicated Mallampati grade III with poor dentition and multiple loose teeth. Laboratory evaluation was within normal limits, with hemoglobin 13.6 g/dL. Electrocardiogram showed sinus tachycardia and echocardiography demonstrated preserved left ventricular function with an ejection fraction of 62% and no structural defects. CECT of the abdomen revealed a large multiloculated cyst, 13 × 11 × 14 cm in size, involving segments V to VIII of the right hepatic lobe. The patient was classified as ASA grade II and was treated with albendazole 800 mg once daily for three weeks prior to surgery. Premedication consisted of intravenous glycopyrrolate 0.2 mg, midazolam 2 mg, and fentanyl 100 mcg. Induction was achieved with propofol 100 mg and succinylcholine 150 mg, followed by intubation using a cuffed 7.5 mm endotracheal tube with video laryngoscopy. Muscle relaxation was maintained with atracurium (30 mg loading dose, 5 mg top-ups). Prophylaxis against hypersensitivity included intravenous hydrocortisone 100 mg and chlorpheniramine 45.5 mg prior to incision. Invasive arterial pressure monitoring via the radial artery was instituted, along with electrocardiography, pulse oximetry, capnography, and urine output measurement. Anesthesia was maintained with sevoflurane 2% in a mixture of oxygen and nitrous oxide (1:1).

During aspiration of approximately 2–2.5 L of cyst fluid, the patient experienced a sudden fall in blood pressure to 80/50 mmHg, with a heart rate of 120/min. Oxygen saturation and end-tidal carbon dioxide remained stable. He was managed with rapid infusion of 500 ml crystalloids and a phenylephrine bolus of 50 mcg, which restored blood pressure to 100/60 mmHg. An additional 50 mg of hydrocortisone was administered. There was no desaturation, bronchospasm, or cutaneous evidence of anaphylaxis.

The cyst wall was marsupialized and subhepatic drains were placed. The patient was extubated smoothly at the end of surgery and transferred to the intensive care unit for monitoring. His postoperative course was uneventful.

Case 3

A 45-year-old woman presented with vague upper abdominal discomfort and early satiety.

She had no comorbid illnesses. Ultrasonography of the abdomen revealed a solitary unilocular cyst, measuring 9 × 8 cm, located in the left lobe of the liver. Laboratory investigations, including liver and renal function tests, were within normal limits.

Hemoglobin was 11.2 g/dL. She was categorized as ASA grade I and had received albendazole 10 mg/kg/day for four weeks preoperatively.

Premedication included intravenous glycopyrrolate 0.2 mg, midazolam 1 mg, and fentanyl 2 mcg/kg. Induction was carried out with propofol 2 mg/kg and rocuronium 0.6 mg/kg. The trachea was intubated with a 7.0 mm cuffed endotracheal tube without difficulty. Prophylactic medications administered before incision included hydrocortisone 100 mg, chlorpheniramine 25 mg, and ranitidine 50 mg intravenously. An epidural catheter was placed at the T9–T10 level, and a bolus of 0.25% bupivacaine 6 ml was given for intraoperative analgesia.

Monitoring included invasive blood pressure, electrocardiography, pulse oximetry, capnography, and urine output. Anesthesia was maintained with Sevoflurane at 1-2% in oxygen and nitrous oxide (40:60).

Preventive strategies were emphasized to minimize the risk of spillage. The operative field was surrounded with pads soaked in 20% hypertonic saline, and controlled aspiration of cyst contents was performed using a closed suction system. The cavity was irrigated with 10% povidone-iodine, and careful dissection was undertaken to avoid rupture or dissemination.

Throughout the intraoperative period, hemodynamics remained stable, with blood pressure ranging between 110–130/70–80 mmHg and heart rate between 70–90/min. There was no desaturation, bronchospasm, or allergic manifestation. Estimated blood loss was approximately 150 ml. At the conclusion of surgery, muscle relaxation was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg. The patient was extubated uneventfully. Postoperative analgesia was maintained with an epidural infusion of 0.125% bupivacaine at 5 ml/hr. She recovered well and was discharged on the fourth postoperative day without complications.

Table 1. Comparison of Three Cases of Hydatid Cyst Surgery and Intraoperative Anaphylaxis

Parameter	Case 1	Case 2	Case 3
Age / Sex	50-year-old female	60-year-old male	45-year-old female
Comorbidities	None	Hypertension, chronic alcoholism	None
Preoperative Therapy	Albendazole 400 mg BID × 15 days	Albendazole 800 mg OD × 3 weeks	Albendazole 10 mg/kg/day × 4 weeks
Imaging Findings	Multiloculated cyst, 15 × 11 cm, right lobe, calcified wall (CECT)	Multiloculated cyst, 13 × 11 × 14 cm, segments V–VIII, right lobe (CECT)	Solitary unilocular cyst, 9 × 8 cm, left lobe (USG)
ASA Classification	I	II	I
Airway / Pre-op Findings	Normal airway	Mallampati III, poor dentition	Normal airway
Premedication	Glycopyrrolate 0.2 mg, Fentanyl 2 mcg/kg, Ranitidine 50 mg	Glycopyrrolate 0.2 mg, Midazolam 2 mg, Fentanyl 100 mcg	Glycopyrrolate 0.2 mg, Midazolam 1 mg, Fentanyl 2 mcg/kg

Induction	Thiopentone 5 mg/kg, Atracurium 0.5 mg/kg	Propofol 100 mg, Succinylcholine 150 mg	Propofol 2 mg/kg, Rocuronium 0.6 mg/kg
Airway Management	7.5 mm cuffed ETT	7.5 mm cuffed ETT (video laryngoscope)	7.0 mm cuffed ETT
Maintenance	Sevoflurane 1% in N ₂ O:O ₂ (2:1), Epidural bupivacaine	Sevoflurane 2% in O ₂ :N ₂ O (50:50)	Sevoflurane 1-2% in O ₂ :N ₂ O (40:60), Epidural bupivacaine
Monitoring	NIBP, ECG, SpO ₂ , urine output	ECG, SpO ₂ , EtCO ₂ , invasive BP, urine output	ECG, SpO ₂ , EtCO ₂ , invasive BP, urine output
Prophylaxis	None pre-incision	Hydrocortisone 100 mg IV, Chlorpheniramine 25 mg IV	Hydrocortisone 100 mg IV, Chlorpheniramine 25 mg IV, Ranitidine 50 mg IV
Intraoperative Event	Fulminant anaphylaxis: hypotension (60/40 mmHg), tachycardia (122/min), desaturation (88%), bronchospasm	Transient hypotension (80/50 mmHg), stable SpO ₂ and EtCO ₂ , no bronchospasm	Uneventful; hemodynamics stable (110-130/70-80 mmHg), no desaturation or reaction

Management	IV crystalloids, Ephedrine, Hydrocortisone, Adrenaline boluses infusion, Methylprednisolone, Aminophylline	IV crystalloids, Phenylephrine 50 mcg, & Hydrocortisone 50 mg	Preventive measures: Prophylactic Hydrocortisone 100mg iv, hypertonic saline packs, closed suction aspiration, povidone-iodine irrigation, meticulous dissection
Outcome	Electively ventilated, extubated after 6 hrs, full recovery, discharged POD 4	Extubated smoothly, ICU monitoring, uneventful recovery	Extubated uneventfully, epidural analgesia, discharged POD 4

DISCUSSION

Hydatid disease continues to be an important clinical entity in endemic regions, with surgical excision remaining the treatment of choice. However, one of the most serious intraoperative complications is anaphylaxis due to spillage of cyst fluid. The present series of three cases demonstrates the variable presentation of anaphylaxis in hepatic hydatid surgery: one patient developed fulminant anaphylaxis with cardiovascular collapse, another experienced transient hypotension without respiratory compromise, while the third underwent uneventful surgery when preventive measures were rigorously implemented. The differences observed among our patients reflect both the unpredictable nature of anaphylaxis and the effectiveness of anticipatory management.

Pathophysiology and Risk Factors

In our first case, sudden onset of hypotension, tachycardia, desaturation, and bronchospasm occurred immediately after cyst manipulation, strongly suggesting IgE-mediated anaphylaxis.

Cotran et al. have described in detail the pathologic mechanisms underlying parasitic infections, emphasizing that rupture of hydatid cysts releases highly antigenic material capable of provoking type I hypersensitivity reactions [10]. This pathophysiologic explanation correlates well with the events observed in our case, where cyst fluid likely entered systemic circulation, triggering a cascade of mediator release and systemic collapse.

Surgical Management and Outcomes

Our second case, in which the patient developed only transient hypotension during aspiration of cyst fluid, illustrates that not all patients experience full-blown anaphylaxis. Gonzalez et al. studied outcomes of surgical treatment for hepatic hydatidosis and highlighted that while anaphylaxis is a feared complication, it does not occur uniformly and may vary in severity from transient hemodynamic instability to cardiovascular arrest [11]. Our findings align with this

variability, as two patients demonstrated different degrees of reaction despite both undergoing large cyst aspirations.

Magistrelli et al. reported that the success of surgical treatment depends not only on the type of operation but also on careful handling of the cyst to avoid rupture and dissemination [12]. In our third case, meticulous preventive strategies—such as packing the operative field with hypertonic saline-soaked pads, controlled aspiration, and irrigation with povidone-iodine—ensured that the patient had no adverse intraoperative event. This confirms the value of preventive techniques described in earlier literature, as deliberate efforts to reduce antigen spillage correlate directly with better anesthetic outcomes.

Case Comparisons with Literature

The catastrophic event observed in Case 1 resembles that described by Taşpınar et al., who reported intraoperative anaphylaxis during hepatic hydatid surgery where the patient developed sudden cardiovascular collapse requiring aggressive resuscitation [13]. Both cases highlight the unpredictable onset and the necessity for immediate administration of adrenaline. In contrast, our second case required only phenylephrine and fluids, which is more consistent with the milder presentation noted by Khanna et al., who documented intraoperative anaphylaxis with hypotension but without bronchospasm, leading to rapid recovery [14].

Harper et al. analyzed suspected anaphylactic reactions in the perioperative period and emphasized that recognition is challenging because hypotension may be attributed to blood loss, anesthetic depth, or myocardial ischemia [15]. In our first case, the diagnosis of anaphylaxis was reached only after excluding other causes, since blood loss was minimal and ECG showed no ischemic changes. This difficulty in recognition reinforces the point made by Harper et al. that vigilance and high suspicion are essential in high-risk surgeries.

Chhabra et al. reported intraoperative anaphylaxis during pulmonary hydatid cyst excision,

demonstrating that the condition is not restricted to hepatic involvement and can present with a wide range of severities [16]. Their patient developed significant bronchospasm and hemodynamic instability, comparable to the severe reaction seen in our first case. Conversely, our second case paralleled the milder end of this spectrum, where hemodynamic changes were isolated and quickly reversible.

Inal et al. described an intraoperative allergic reaction during hydatid surgery where the patient responded favorably to steroids and supportive management [17]. This finding resonates with our second case, where supplementary hydrocortisone contributed to stabilization after hypotension, and highlights that not all reactions necessitate high-dose adrenaline if identified and treated promptly.

Preventive Measures and Prophylaxis

Our third case underscores the importance of preventive measures. By administering

preoperative albendazole, perioperative hydrocortisone, antihistamines, and by employing careful operative techniques, no adverse reaction occurred. Sabouni et al. documented that multiple organ hydatid involvement carries a higher risk of complications, including hypersensitivity, thereby stressing the importance of prophylaxis and comprehensive preoperative planning [18]. Our findings corroborate this, as rigorous prophylaxis appeared to play a pivotal role in preventing complications in Case 3.

Gomez et al. reviewed the treatment of hepatic hydatid cysts and concluded that advances in surgical and anesthetic techniques, combined with preventive pharmacological measures, have reduced the incidence of catastrophic intraoperative events [19]. Our third case exemplifies this trend: by implementing preoperative medical therapy and intraoperative scolicalid precautions and thereby avoided anaphylaxis altogether. This supports the recommendation that prevention remains the cornerstone of management in high-risk hydatid surgeries. Bengghir et al. analyzed cases of anaphylactic shock during hydatid cyst surgery and reported that prompt recognition and aggressive management with adrenaline are lifesaving [20].

Their conclusions resonate with the outcome of our first case, where adrenaline infusion was critical to

recovery. Importantly, the variable presentations across our three cases are consistent with their observation that no two patients present identically, making preparedness essential in all cases of hydatid surgery.

LIMITATIONS

Our case series is limited by the small number of patients, which restricts generalizability. However, the value lies in illustrating the full clinical spectrum of intraoperative reactions, from life-threatening anaphylaxis to uneventful surgery. Larger prospective studies are needed to quantify the incidence of anaphylaxis in hydatid cyst surgery and to evaluate the effectiveness of specific preventive strategies.

CONCLUSION

Hydatid disease of the liver continues to pose significant anesthetic challenges, particularly due to the risk of intraoperative anaphylaxis following rupture or spillage of cyst contents. This case series demonstrates the varied clinical spectrum of such reactions. In the first case, fulminant anaphylaxis with cardiovascular collapse required aggressive resuscitation with adrenaline, inotropes, and ventilatory support. The second case showed only transient hypotension, which responded to fluids and vasopressors without progression to full anaphylaxis. The third case highlighted the effectiveness of prophylaxis and preventive measures, where careful preoperative optimization, intraoperative administration of corticosteroids and antihistamines, and meticulous surgical technique ensured a complication-free course.

These contrasting outcomes emphasize that intraoperative anaphylaxis in hydatid surgery is

unpredictable and may range from mild instability to life-threatening circulatory shock. Early recognition, prompt administration of adrenaline, and supportive therapy remain the cornerstones of management. Equally important are preventive strategies such as

preoperative albendazole, perioperative prophylaxis, and intraoperative precautions to minimize cyst spillage.

In conclusion, anesthesiologists managing hydatid cyst surgery must maintain a high index of suspicion, ensure readiness for immediate intervention, and collaborate closely with surgeons to optimize patient safety. Vigilance and preparedness are critical to achieving favorable outcomes in this high-risk setting.

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