



ACUTE INTRAOPERATIVE VENTRICULAR TACHYCARDIA INDUCED BY INDOCYANINE GREEN (ICG) NEBULISATION DURING TOTAL ESOPHAGECTOMY: A CASE REPORT

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ABSTRACT

Background: Indocyanine green (ICG) is an emerging intraoperative fluorescence agent increasingly used for anatomical mapping and tissue visualization. Its administration through nebulisation or inhalation for tracheobronchial and lung segmentation mapping is expanding. However, life-threatening cardiovascular complications associated with ICG remain extremely rare.

Case Description: A 64-year-old woman undergoing total esophagectomy developed ventricular tachycardia and cardiovascular collapse approximately 30 seconds after initiation of nebulised ICG. Pre-event blood pressure was 136/82 mmHg, heart rate 92/min, SpO₂ 100%, and end-tidal CO₂ 30–32 mmHg during mechanical ventilation. Resuscitation included cardioversion, defibrillation, and advanced life support. Post-event serum tryptase was 35.5 ng/mL at 2 hours and 5 ng/mL at 24 hours, supporting acute mast-cell activation. Troponin I and CK-MB were negative, transthoracic echocardiography showed LVEF 55%, and coronary angiography revealed no significant coronary abnormalities. The procedure was abandoned; the patient was extubated and transferred to the SICU.

Conclusion: Ventricular tachycardia occurred in close temporal association with nebulised ICG, with a significant acute tryptase rise supporting a perioperative hypersensitivity reaction. However, the responsible trigger and mechanism cannot be established conclusively. Vigilant monitoring and immediate resuscitation capability are essential.

KEYWORDS: Indocyanine Green, Nebulisation, Ventricular Tachycardia, Esophagectomy, Intraoperative Arrhythmia, Fluorescence-guided Surgery.

Introduction

Indocyanine green (ICG) is a water-soluble tricarbo-cyanine fluorescent dye increasingly used in fluorescence-guided surgery. Its near-infrared fluorescence properties permit

real-time visualization of tissue perfusion, vascular and lymphatic structures, and anatomical planes during surgical procedures[1]. Alander et al. (2012) described the expanding applications of ICG fluorescence imaging across

different surgical specialties, highlighting its utility as an intraoperative imaging agent[1]. Although ICG is generally considered safe, uncommon hypersensitivity and serious cardiovascular reactions have been reported following intravenous administration[2].

Hope-Ross et al. (1994) evaluated adverse reactions associated with ICG angiography and reported that severe reactions were uncommon but potentially life-threatening[2]. Importantly, Chu et al. (2017) described severe hypotension, increased airway pressure, and ventricular tachycardia shortly after intravenous administration of ICG during robotic partial nephrectomy, demonstrating that severe cardiovascular manifestations may occur following ICG exposure[3].

More recently, nebulised ICG has emerged as an innovative technique for tracheobronchial visualization during esophageal surgery. Thammineedi et al. (2024) evaluated ICG tracheobronchial fluorescence following nebulisation in 37 patients undergoing minimally invasive esophagectomy and reported effective fluorescence without intraoperative allergic reactions attributable to ICG[4]. Rajan et al. (2026) similarly demonstrated successful tracheobronchial fluorescence following awake nebulised ICG in 30 patients, with no ICG allergy observed[5]. Despite these encouraging findings, clinical experience with the nebulised route remains limited.

We report a case of acute malignant ventricular tachycardia occurring within 30 seconds of initiating ICG nebulisation during total esophagectomy, highlighting a potentially serious cardiovascular complication associated with this emerging route of administration.

Case Presentation

A 64-year-old woman, weighing 53 kg and measuring 153 cm in height, with a diagnosis of carcinoma of the esophagus was scheduled for total esophagectomy. Her medical history was significant for mild chronic obstructive pulmonary disease (COPD). Preoperative electrocardiography demonstrated normal sinus rhythm. Two-dimensional echocardiography revealed a left ventricular ejection fraction of 55% with no regional wall-motion abnormalities. Contrast-enhanced computed tomography of the chest demonstrated hyperinflated lung fields with mild, stable COPD changes. Routine hematological and biochemical investigations, including serum electrolytes, renal function, hepatic function, and metabolic parameters, were within normal limits. Following respiratory and endocrine consultations and appropriate optimization, the patient was considered fit for general anesthesia.

Anaesthetic and Intraoperative Management

Standard American Society of Anesthesiologists monitoring, including non-invasive blood pressure, pulse oximetry, electrocardiography, and temperature monitoring, was

instituted. A central venous catheter was secured through the right internal jugular vein. A thoracic epidural catheter was placed at the T8–T9 interspace for postoperative analgesia.

Following induction of general anesthesia, a double-lumen endotracheal tube was placed. One-lung ventilation was initiated, and appropriate tube position was confirmed using fiberoptic bronchoscopy. The patient was subsequently positioned prone for the thoracic phase of the procedure, following which one-lung ventilation was reconfirmed. Laparoscopic/thoracoscopic surgery commenced and proceeded uneventfully for approximately two hours. Immediately before the event, recorded blood pressure was 136/82 mmHg, pulse rate 92/min, peripheral oxygen saturation 100%, and end-tidal carbon dioxide 30–32 mmHg. Ventilation was volume-controlled, with a tidal volume of 300–400 mL, respiratory rate 14–15/min, FiO₂ 1.0, and positive end-expiratory pressure 5 cmH₂O.

Acute Intraoperative Ventricular Tachycardia

Approximately two hours after commencement of surgery, at the surgeon's request for improved anatomical visualization, ICG nebulisation through the airway was initiated. The precise dose, concentration, dilution volume, nebuliser type, and delivery duration were not available in the clinical details reviewed. Approximately 30 seconds after nebulisation began, ventricular tachycardia was observed, accompanied by acute cardiovascular deterioration.

The surgical procedure was immediately halted, 100% oxygen was administered, and airway patency was confirmed. An intravenous bolus of amiodarone 150 mg was administered for the initial ventricular tachycardia. As the arrhythmia persisted and the patient's hemodynamic status deteriorated, she was urgently repositioned from prone to supine. The rhythm progressed from ventricular tachycardia with a pulse to hemodynamically unstable VT, prompting synchronized cardioversion and rapid intravenous crystalloid administration for hypotension. Subsequently, the patient developed pulseless ventricular tachycardia.

High-quality cardiopulmonary resuscitation was immediately initiated in accordance with advanced cardiovascular life support principles. Unsynchronized biphasic defibrillation at 200 J was delivered, with four cycles documented. Intravenous epinephrine 1 mg and corticosteroids were administered, and a norepinephrine infusion was titrated according to the mean arterial pressure. Following resuscitative measures, sinus rhythm was successfully restored and the patient regained hemodynamic stability. In view of the severity of the cardiovascular event, the surgical procedure was abandoned.

Post-event Management and Outcome

Serial arterial blood gas analyses performed following resuscitation demonstrated severe metabolic acidosis with hypokalemia. These abnormalities were corrected with

sodium bicarbonate and potassium chloride. The epidural infusion was withheld. After cardiovascular and respiratory stabilization, the patient was successfully extubated in the operating room and transferred to the Surgical Intensive Care Unit for further monitoring and management. In the SICU, serum tryptase measured 35.5 ng/mL at 2 hours after the event and 5 ng/mL at 24 hours. This acute elevation exceeded the commonly used event-related threshold of $(1.2 \times \text{baseline}) + 2 \text{ ng/mL}$ (8 ng/mL in this patient), consistent with substantial mast-cell activation. Troponin I and CK-MB were within the laboratory's negative range. Post-event transthoracic echocardiography showed normal cardiac findings with a left ventricular ejection fraction of 55%. Coronary angiography revealed no significant coronary abnormalities.

The available clinical record indicates that the patient had no immediate neurological deficit following the event. Further oncological resection was planned for reconsideration by the multidisciplinary team following cardiac and allergy evaluation.

Discussion and Diagnostic Reasoning

Indocyanine green (ICG) has a favorable overall safety profile, although severe immediate cardiovascular and hypersensitivity reactions are well documented after

intravenous exposure. In a series of 1,923 ICG angiographic procedures, Hope-Ross et al. reported severe reactions in approximately 0.05% of administrations[2]. Obana et al., evaluating 3,774 angiograms in 2,820 patients, reported adverse reactions in 0.34%, including two episodes of hypotension requiring treatment for shock[6]. Bjerregaard et al. subsequently described profound hypotension immediately following low-dose intravenous ICG during otherwise uncomplicated surgery[7].

The present case differs importantly in the route and temporal pattern of exposure. Malignant ventricular tachycardia developed approximately 30 seconds after initiation of nebulised ICG, after nearly two hours of otherwise uneventful surgery. Experience with this route remains limited. Thammineedi et al. evaluated aerosolized ICG in 37 patients undergoing minimally invasive esophagectomy; tracheobronchial fluorescence was achieved in 35 patients (94.6%), with no intraoperative or postoperative complications attributed to ICG[4]. More recently, Rajan et al. reported successful tracheobronchial fluorescence in 30 patients receiving awake nebulised ICG, again without reported ICG allergy[5]. These relatively small feasibility cohorts, however, cannot exclude infrequent serious adverse reactions.

Table 1: Timeline and Intraoperative Management

Time/ Stage	Events and Clinical Observations
Baseline/Induction	Standard ASA monitoring instituted (NIBP, SpO ₂ , ECG, temperature). Right internal jugular central venous catheter secured. Thoracic epidural catheter placed at T8–T9.
Airway and positioning	Double-lumen tube placed. One-lung ventilation initiated and fiberoptically confirmed. Patient positioned prone for the thoracic phase; OLV reconfirmed.
Surgical start	Laparoscopic/thoracoscopic surgery commenced uneventfully.
Approximately 2 h	At the surgeon's request for anatomical visualization, side nebulisation with ICG was initiated through the airway. Pre-event: BP 136/82 mmHg, pulse 92/min, SpO ₂ 100%, EtCO ₂ 30–32 mmHg; VCV, tidal volume 300–400 mL, RR 14–15/min, FiO ₂ 100%, PEEP 5 cmH ₂ O. ICG preparation/device/dose.
~30 s after ICG initiation	Sudden ventricular tachycardia appeared on the monitor with acute cardiovascular deterioration.
Initial management	Surgery immediately halted; 100% oxygen administered; airway patency confirmed; IV amiodarone 150 mg administered.
Persistent/deteriorating VT	Patient urgently repositioned from prone to supine. VT became hemodynamically unstable with a pulse; synchronized cardioversion and rapid crystalloid administration undertaken.
Pulseless VT	High-quality CPR initiated. Unsynchronized biphasic defibrillation at 200 J performed, with four cycles documented. Epinephrine 1 mg and corticosteroids administered; norepinephrine infusion titrated according to MAP.
Resolution	Sinus rhythm restored with hemodynamic recovery. Surgical procedure abandoned.
Post-event care	Serial ABG showed severe metabolic acidosis with hypokalemia. Sodium bicarbonate and potassium chloride administered. Epidural infusion withheld. Patient successfully extubated in the operating room and transferred to SICU. SICU tryptase 35.5 ng/mL (2 h) and 5 ng/mL (24 h); negative troponin I and CK-MB; echo LVEF 55%; coronary angiography without significant abnormalities.

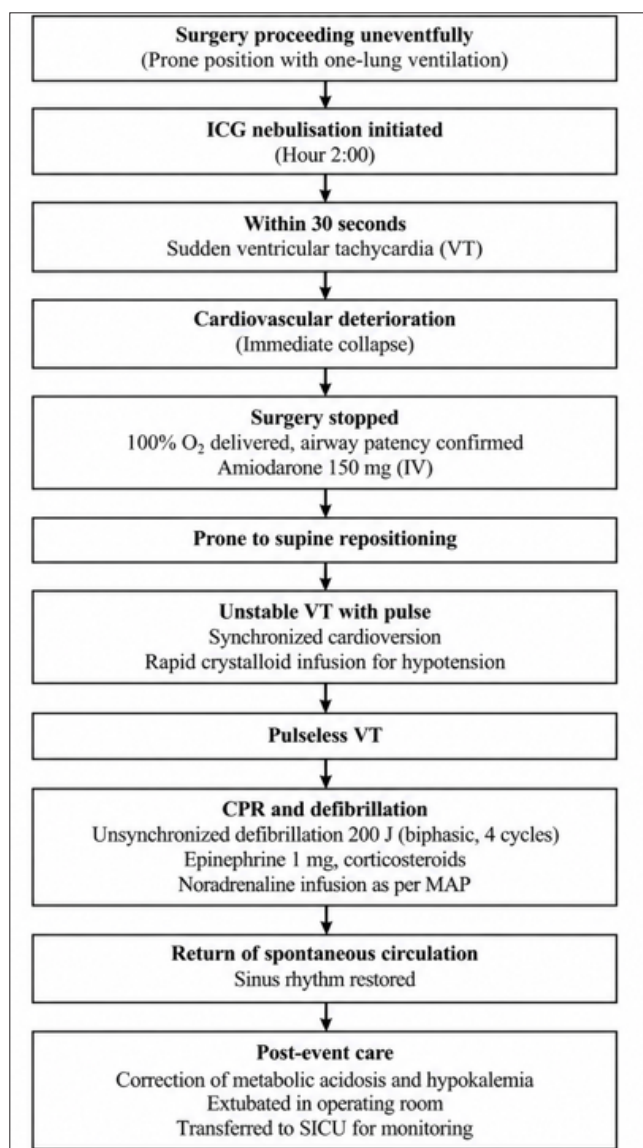


Figure 1. Clinical sequence of the acute event following nebulisation

Two mechanisms were considered in the diagnostic reasoning. First, rapid pulmonary absorption was considered because the patient had hyperinflated lungs and mild COPD. Direct delivery to the respiratory tract could theoretically permit systemic absorption of ICG; however, pharmacokinetic evidence demonstrating rapid, bolus-like absorption of nebulised ICG particularly in COPD is currently lacking. This mechanism should therefore be regarded as a hypothesis rather than an established explanation.

Second, the short interval between ICG exposure and cardiovascular collapse, together with a serum tryptase rise from 5 ng/mL at 24 hours to 35.5 ng/mL at 2 hours, supports acute systemic mast-cell activation and makes a perioperative immediate hypersensitivity reaction clinically plausible. The acute concentration exceeds the event-related threshold of $(1.2 \times \text{baseline}) + 2 \text{ ng/mL}$. Nevertheless, this finding does not identify the responsible exposure, distinguish IgE-mediated from non-IgE-mediated activation, or establish ICG-specific allergy. Other perioperative agents

and exposures require assessment. An allergy-associated coronary event (Kounis syndrome) remains a speculative differential diagnosis, not an established diagnosis: cardiac biomarkers were negative, follow-up echocardiography showed preserved LVEF (55%), and coronary angiography found no significant abnormalities. Although transient vasospasm may not be visible on later angiography, objective evidence of acute myocardial ischemia was not documented. Prior reports of ICG-associated hypersensitivity after intravenous administration provide biological plausibility but cannot establish the mechanism in this case[3,8].

Alternative causes of perioperative ventricular tachycardia were considered. Hypoxemia related to one-lung ventilation, double-lumen tube displacement, ventilation-perfusion mismatch, or airway obstruction can precipitate arrhythmia; immediately before the event, however, SpO₂ was 100% and end-tidal CO₂ 30–32 mmHg with volume-controlled ventilation (tidal volume 300–400 mL, respiratory rate 14–15/min, FiO₂ 1.0, PEEP 5 cmH₂O). These observations

reduce concern for sustained pre-event desaturation but cannot exclude a brief unrecorded respiratory disturbance or hypercarbia. Surgical manipulation and autonomic stimulation, as well as anaesthetic medications and vasopressor exposure, are additional potential triggers; detailed medication timing and immediate peri-event hemodynamic trends should be reviewed before excluding them. Preoperative electrolytes were normal, but severe metabolic acidosis and hypokalemia were documented only after resuscitation. Without blood gas and electrolyte measurements immediately preceding VT, it is not possible to determine whether these abnormalities preceded or resulted from the arrest and resuscitation. The close temporal relationship with nebulisation is suggestive but does not establish ICG causality.

Post-event investigations strengthen the evidence for an acute mast-cell activation event but do not establish the identity of the trigger. The negative cardiac biomarkers, preserved systolic function, and absence of significant coronary angiographic abnormalities provide no objective confirmation of an acute coronary syndrome. A Kounis-type process therefore remains hypothetical. Formal post-event allergy evaluation, where available, would be needed to investigate potential culprit exposures.

Clinical Lessons and Recommendations

Nebulised ICG should be administered under continuous cardiovascular monitoring with immediate access to advanced resuscitation facilities. Potentially arrhythmogenic electrolyte and acid–base abnormalities should be corrected before administration. Sudden cardiovascular instability temporally associated with ICG exposure should prompt consideration of an immediate hypersensitivity reaction, alongside other perioperative causes, with timely serum tryptase sampling where clinically appropriate.

Patient Outcome and Follow-up

Following successful resuscitation, sinus rhythm and hemodynamic stability were restored. The patient was extubated in the operating room without immediate neurological deficit and transferred to the SICU for monitoring. Serum tryptase was 35.5 ng/mL at 2 hours and 5 ng/mL at 24 hours after the event. Troponin I and CK-MB were negative. Transthoracic echocardiography demonstrated normal cardiac findings (LVEF 55%), and coronary angiography showed no significant coronary abnormalities. Further oncological management was to be reconsidered after multidisciplinary cardiac and allergy assessment.

Conclusion

Ventricular tachycardia and cardiovascular collapse occurred approximately 30 seconds after initiation of nebulised ICG during total esophagectomy. A marked event-related rise in serum tryptase supports acute mast-cell activation and a possible perioperative hypersensitivity reaction, but the

specific trigger and precise mechanism remain uncertain. Kounis syndrome is speculative and was not confirmed. This case emphasizes the importance of careful assessment of alternative perioperative triggers, vigilant monitoring, and immediate resuscitative preparedness during nebulised ICG administration.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and associated clinical information.

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Conflict of Interest

The authors declare no conflicts of interest.

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