



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

Dr Sukriti Khatri¹, Dr Dev Kumar Harkawat^{2*}, Dr Manish Kaushik³

¹Postgraduate Resident, Department of Anaesthesiology, Jaipur National University Institute For Medical Sciences and Research Centre, Jaipur, Rajasthan, India.

²Professor, Department of Anaesthesiology, Jaipur National University Institute For Medical Sciences and Research Centre, Jaipur, Rajasthan, India.

³Assistant Professor, Department of Surgical Oncology, Jaipur National University Institute For Medical Sciences and Research Centre, Jaipur, Rajasthan, India.

Corresponding Author*: Dr Dev Kumar Harkawat, Professor, Department of Anaesthesiology, Jaipur National University Institute For Medical Sciences and Research Centre, Jaipur, Rajasthan, India.

Email ID: dev.harkawat@gmail.com

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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 female with carcinoma of the esophagus, scheduled for total esophagectomy, developed sudden malignant ventricular tachycardia (VT) within 30 seconds of initiating intraoperative ICG nebulisation. The arrhythmia was accompanied by cardiovascular collapse. Surgery was immediately halted, and the patient was repositioned from prone to supine. Cardiopulmonary resuscitation (CPR), electrical defibrillation, and appropriate resuscitative measures successfully restored normal sinus rhythm and hemodynamic stability. Considering the severity of the event, the surgical procedure was abandoned. Following stabilization, the patient was successfully extubated in the operating room and transferred to the Surgical Intensive Care Unit (SICU) for further monitoring and management.

Conclusion: This case highlights a severe, hyper-acute cardiovascular event temporally associated with nebulised ICG and emphasizes the need for vigilant monitoring and immediate resuscitative preparedness during its administration.

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.

Acute Intraoperative Ventricular Tachycardia

Approximately two hours after commencement of surgery, at the surgeon's request for improved anatomical visualization, side nebulisation with ICG was initiated through the airway. Within approximately 30 seconds, sudden ventricular tachycardia was observed on the cardiac monitor, 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.

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.

Two mechanisms were considered in the diagnostic reasoning. First, Hyper-acute Alveolar Absorption, 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.

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.
~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.

Second, the extremely short interval between exposure and cardiovascular collapse raises the possibility of an immediate hypersensitivity-mediated reaction, potentially accompanied by coronary vasospasm. This interpretation is supported indirectly by previous intravenous ICG reactions. Chu et al. reported severe hypotension, increased airway pressure, and ventricular tachycardia shortly after intravenous ICG administration; markedly elevated serum tryptase supported an anaphylactic mechanism[3]. Sasaki and Murata similarly reported immediate severe hypotension following ICG administration, with elevated histamine and tryptase and subsequent intradermal testing supporting ICG-induced anaphylaxis[8]. In the present case, an anaphylactoid response with coronary vasospasm and consequently a Kounis-like mechanism was therefore considered; however, Kounis syndrome cannot be established without supporting evidence of an allergic reaction and myocardial ischemia.

Alternative causes of perioperative VT must also be considered. Preoperative ECG, electrolytes, and other routine investigations were normal, LVEF was preserved at 55%, and the operation had proceeded uneventfully before ICG exposure. Severe metabolic acidosis and hypokalemia were detected following resuscitation and corrected, but their contribution to arrhythmia initiation cannot be established because contemporaneous pre-event values were unavailable. Thus, the approximately 30-second temporal relationship provides the strongest evidence for an ICG-associated event, while causality and the precise pathophysiological mechanism remain unproven.

Clinical Lessons and Recommendations

Nebulised ICG should be administered under continuous cardiovascular monitoring with immediate access to advanced resuscitation facilities, also thoroughly screen patients for prior iodine allergies before using any form of ICG. 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

The patient was extubated in the operating room without immediate neurological deficit and was transferred to the Surgical Intensive Care Unit (SICU) under strict, continuous monitoring to guard against a biphasic anaphylactic reaction or recurrent arrhythmia:

Cardiovascular Surveillance

Continuous 12-lead ECG telemetry tracking for ischemia or QT prolongation. Serial cardiac enzymes (Troponin I

and CK-MB) were ordered at 6-hour intervals to check for myocardial injury. A bedside 2D ECHO was scheduled to re-evaluate LVEF.

Anaphylaxis Investigation

Immediate serum tryptase levels were drawn to confirm immune-mediated mast cell activation. An absolute contraindication to ICG and iodine dyes was flagged in her chart.

Electrolyte Targets

Serum potassium (>4.0 mEq/L) and magnesium (>2.0 mg/dL) are being maintained at high-normal limits to preserve cardiac electrical stability.

Conclusion

Malignant ventricular tachycardia developed within approximately 30 seconds of initiating nebulised ICG during total esophagectomy. Although the close temporal association suggests a possible ICG-related adverse reaction, the precise mechanism remains uncertain. This case emphasizes vigilant monitoring and immediate resuscitative preparedness when nebulised ICG is used.

Informed Consent

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

Conflict of Interest

The authors declare no conflicts of interest.

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