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Case Report

Anaesthesia Section

Dilemmas to Proceed with High-risk NORA Endoscopy: A Case of Patient with Bradycardia

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ABSTRACT

Colorectal cancer is a leading cause of cancer-related morbidity and mortality worldwide. Early diagnosis through colonoscopy remains a cornerstone in improving prognosis. However, performing such diagnostic procedures in elderly patients with significant co-morbidities poses unique challenges. The elderly population, often characterised by age-related physiological changes and multiple co-existing conditions, is especially vulnerable to perioperative complications, including cardiovascular instability, altered drug metabolism, and neurological disturbances. Authors present a rare and clinically significant case of a 62-year-old male patient with suspected colorectal malignancy and baseline sinus bradycardia, referred for diagnostic colonoscopy. Notably, this patient had previously been deferred from undergoing the procedure due to severe perioperative bradycardia at an outside hospital. Comprehensive preoperative evaluation, including Electrocardiography (ECG), echocardiography, and Holter monitoring, revealed no malignant arrhythmias but persistent physiological bradycardia. During the first colonoscopy attempt, profound bradycardia and hypotension occurred following standard sedation, requiring pharmacologic intervention and the eventual abortion of the procedure. A second attempt revealed intraprocedural hypocalcaemia as a contributing factor to both bradycardia and abnormal limb movements, which was successfully managed with intravenous calcium. A third, carefully adjusted sedation protocol-using preprocedure atropine and ketamine-based sedation-resulted in a stable and successful colonoscopy without complications. This case uniquely underscores the need for an individualised, multidisciplinary anaesthetic strategy in elderly patients with baseline conduction abnormalities undergoing Non Operating Room Anaesthesia (NORA). It highlights the interplay between bradycardia, hypocalcaemia, and sedation, emphasising the importance of proactive management, tailored drug selection, and readiness for cardiac intervention in diagnostic procedures with potential physiological risks.

Keywords: Anaesthesia, Hypocalcaemia, Intraoperative complications, Multidisciplinary care, Sedation

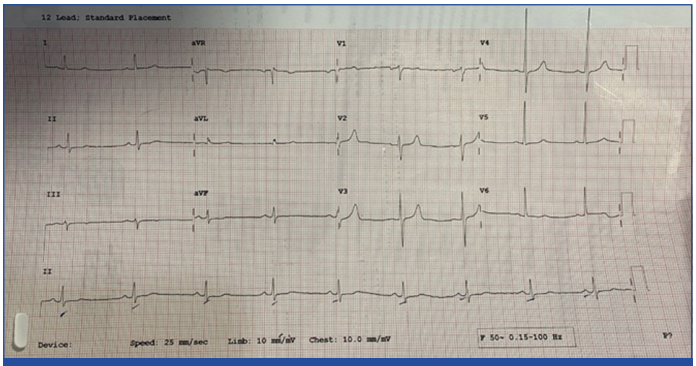
CASE REPORT

A 62-year-old male farmer, classified as American Society of Anaesthesiologists (ASA) physical status III, presented with complaints of gradually progressive, dull, non radiating lower abdominal pain for one month, associated with loss of appetite and a 5 kg weight loss. He also noticed swelling in the periumbilical region. The pain was intermittent, increased after meals, and was not relieved with over-the-counter analgesics. He had a history of chronic alcohol consumption and tobacco chewing for over 15 years but no previous major medical or surgical illnesses. Notably, this patient had previously been deferred from undergoing the procedure due to severe perioperative bradycardia at an outside hospital.

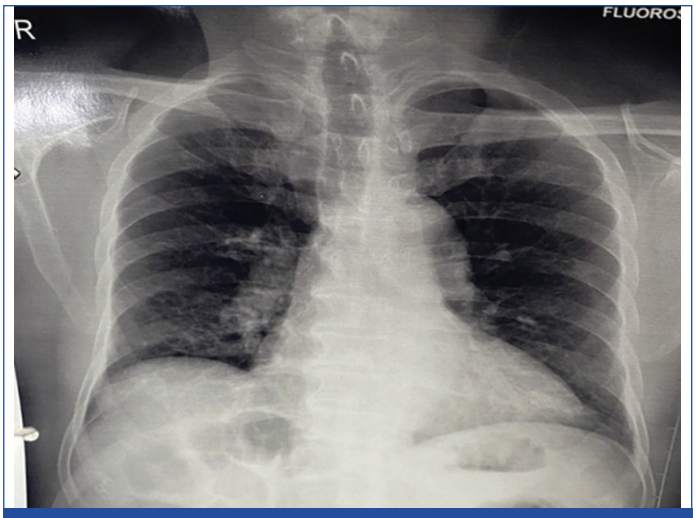
On physical examination, he was haemodynamically stable with a pulse rate of 56 bpm, blood pressure of 130/80 mmHg, and SpO_2 of 99% on room air. A reducible umbilical hernia was noted. Laboratory investigations showed haemoglobin of 8 g/dL, serum albumin of 2.8 g/dL, and HbA1c of 6.1%, with elevated tumour markers: CEA 19.79 ng/mL and CA 19-9 of 34.47 U/mL. ECG revealed sinus bradycardia [Table/Fig-1] and 2D echocardiography showed preserved ejection fraction (60%) with no structural abnormalities. Preoperative chest X-ray was unremarkable [Table/Fig-2].

Holter monitoring detected occasional supraventricular and ventricular ectopics, with a lowest heart rate of 46 bpm and no high-grade arrhythmias.

First colonoscopy attempt - day 1: The patient was confirmed nil per os and was shifted to the colonoscopy suite at 08:00 AM. Standard monitoring (ECG, non invasive blood pressure, and SpO2) was initiated, and a 20G intravenous line was secured. At 08:20 AM, glycopyrrolate 0.2 mg was administered intravenously as a prophylactic measure. Sedation was initiated at 08:25 AM with



[Table/Fig-1]: ECG showing bradycardia.



[Table/Fig-2]: Chest X-ray.

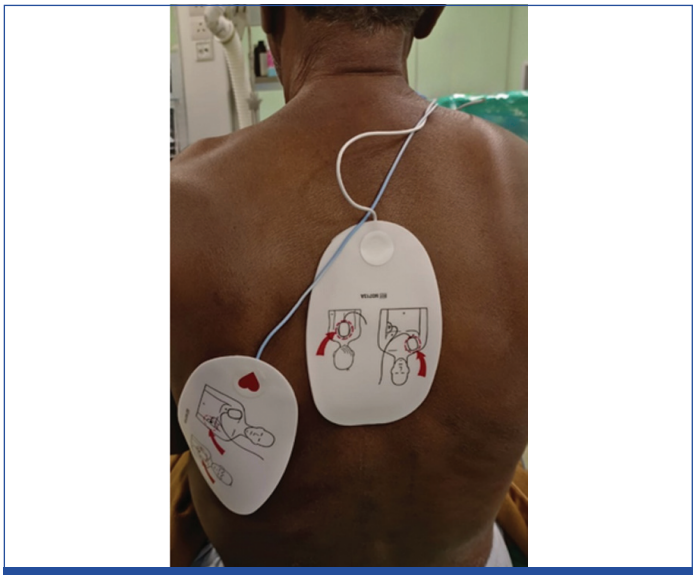
midazolam 1 mg intravenously, fentanyl 50 µg intravenously, and propofol 30 mg intravenously as a bolus.

Baseline vital signs prior to sedation were: heart rate 56 bpm, blood pressure 156/81 mmHg (mean 105 mmHg), SpO_2 at 100%, and respiratory rate at 15/min [Table/Fig-3]. Within two minutes of the sedative administration, the patient developed bradycardia (HR 35 bpm) and hypotension (BP 80/50 mmHg). These haemodynamic changes were attributed to the vagotonic effect of fentanyl and the sympatholytic effect of propofol. Immediate management included intravenous atropine 1 mg, mephentermine 6 mg, and a 200 mL bolus of normal saline, after which the patient's vital signs stabilised within 3-4 minutes. The colonoscopy was aborted, and the patient was observed in the Post-Anaesthesia Care Unit (PACU) for 30 minutes, during which recovery remained uneventful.

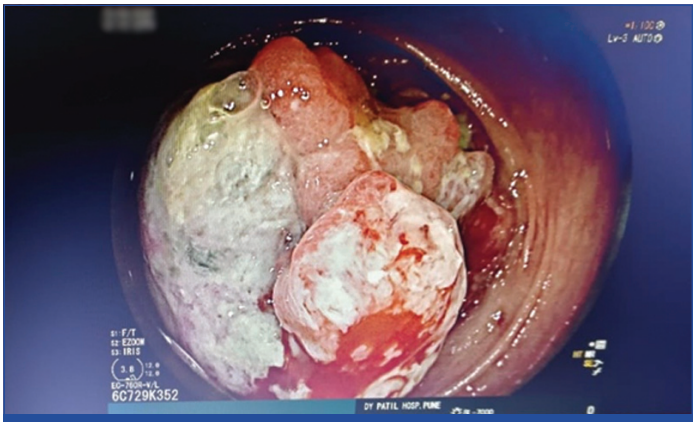
[Table/Fig-3]: Monitor display showing baseline presedation vital parameters: heart rate 56 bpm, blood pressure 156/81 mmHg (mean 105 mmHg), SpO_2 100% and respiratory rate 15/min.

Further evaluation and second attempt - day 3: Following the aborted attempt, a cardiology consultation confirmed that the bradycardia was physiological. A repeat Holter monitor showed no arrhythmias. Atropine 1 mg was administered intramuscularly 20 minutes before the second colonoscopy. Sedation was modified to include midazolam 1 mg intravenously, ketamine 40 mg intravenously, and propofol 20 mg intravenously. Intra-procedurally, the heart rate dropped to 40 bpm, and involuntary movements in the left upper limb were observed. Venous blood gas analysis revealed euglycaemia but low ionised calcium levels. A diagnosis of corrected hypocalcaemia was made and treated with calcium gluconate 1 g intravenously in 100 mL normal saline over 20 minutes, after which both the bradycardia and abnormal movements resolved. Due to the patient's instability, the procedure was again postponed. This episode emphasised the significance of electrolyte imbalances in bradycardic patients.

Final attempt - day 5: A multidisciplinary discussion involving anaesthesia, cardiology, and gastroenterology teams preceded the final attempt. Intensive Care Unit (ICU) backup was arranged, with a defibrillator, Automated External Defibrillator (AED) pads, and pacing setup prepared [Table/Fig-4]. Atropine 1 mg was administered intravenously 10 minutes before sedation. Sedation included ketamine 40 mg intravenously, propofol 20 mg intravenously (with an additional 20 mg titration as needed), pentazocine 30 mg intravenously, and paracetamol 1 g intravenously. Oxygen was administered via nasal prongs at 3 L/min. The procedure was completed in 30 minutes with stable vital signs (HR 50-55 bpm, BP 100/70 mmHg). No bradycardia or seizures occurred intraoperatively. However, 15 minutes postprocedure, the heart rate dropped to 40 bpm and was successfully managed with atropine 1 mg intravenously. The patient was observed for two hours and later discharged in stable condition. Colonoscopy revealed a large ulceroproliferative lesion in the caecum [Table/Fig-5], consistent with malignancy.



[Table/Fig-4]: Automated External Defibrillator (AED) pads attached, preparation for bradycardia events.



[Table/Fig-5]: Large ulceroproliferative growth in caecum- malignant.

DISCUSSION

Colonoscopy remains pivotal for the early diagnosis of colorectal cancer, particularly in elderly patients presenting with weight loss and raised tumour markers [1,2]. Sedation in such patients is challenging due to altered drug pharmacodynamics and pre-existing cardiac conduction variations [3]. In the present case, the combination of propofol and fentanyl during the first attempt likely precipitated bradycardia by suppressing sinoatrial node activity and enhancing vagal tone [4,5]. The subsequent episode revealed hypocalcaemia as an additional reversible contributor, consistent with known cardiac and neuromuscular manifestations [6]. Holter and echocardiographic findings confirmed physiological bradycardia rather than a pathological conduction defect [7]. The modified approach during the final attempt—combining ketamine for its sympathomimetic effect with low-dose propofol and prophylactic atropine—ensured stable haemodynamics. The use of ketamine-propofol combinations has been validated as safe and effective in geriatric and haemodynamically unstable populations [3,5,8]. This case emphasises the need for vigilant monitoring, correction of metabolic disturbances, and multidisciplinary planning for high-risk sedation in NORA settings [3,8,9]. Differentiating between physiological and pathological bradycardia through Holter analysis and echocardiography is vital in guiding anaesthetic management [7,10].

A comprehensive understanding of the potential aetiologies of bradycardia, as categorised in [Table/Fig-6] [4], is essential for the perioperative physician. This case highlights the challenges of administering anaesthesia for colonoscopy in an elderly patient

with baseline bradycardia. Careful preoperative evaluation, early identification and correction of electrolyte disturbances, and judicious modification of sedative agents are crucial in avoiding perioperative complications. A multidisciplinary, individualised approach with vigilant monitoring ensures procedural safety even in high-risk patients. This underscores the need for proactive perioperative strategies in NORA settings.

Category	Examples
Intrinsic cardiac causes	-Sick sinus syndrome
	-AV (artrioventricular) block (1st,2nd or 3rd degree)
	-Myocardial infarction (especially inferior wall MI)
	-Cardiomyopathy
	-Myocarditis
	-Ageing related degeneration of the conduction system
Extrinsic causes	-Hypothyroidism
	-Hyperkalaemia
	-Hypothermia
	-Increased intracranial pressure (Cushing reflex)
	-Vagal stimulation (e.g., carotid sinus hypersensitivity, valsalva manoeuvre)
	-Sleep (physiological bradycardia)
Pharmacological causes	-Beta blockers
	-Calcium channel blockers (e.g., verapamil, diltiazem)
	-Digoxin
	-Amiodarone
	-Clonidine
Systemic conditions	-Sepsis (late stages)
	-Hypoxia
	-Anorexia nervosa
	-Chronic endurance training (Athlete's heart)
Congenital or genetic	-Congenital heart block
	--Genetic channelopathies (e.g., long QT syndrome)
Toxins and poisons	-Organophosphate poisoning
	-Cholinergic toxicity

[Table/Fig-6]: Causes of bradycardia [4].

CONCLUSION(S)

Elderly patients with asymptomatic bradycardia and electrolyte disturbances pose significant sedation challenges, particularly in urgent diagnostic NORA procedures. Present case illustrates that with thorough evaluation, tailored drug selection, and preparedness for cardiac complications, such patients can undergo colonoscopy safely. This aligns with emerging evidence that individualised sedation protocols and vigilant monitoring improve outcomes in high-risk populations.

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