DOI: 10.7860/JCDR/2026/80131.22840

Case Report

Anaesthesia Section

Sinus Bradycardia as a Manifestation of Iron Deficiency Anaemia: A Case Report and Anaesthetic Implications

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ABSTRACT

Iron Deficiency Anaemia (IDA) is the world's leading nutritional disorder. While the conventional cardiovascular response to anaemia involves tachycardia due to sympathetic stimulation, anaemia-related autonomic dysfunction may paradoxically present as bradycardia. Such an atypical presentation can complicate perioperative management, particularly in patients scheduled for major surgery. Hereby, the authors present a case of a 54-year-old postmenopausal woman with an ovarian tumour and symptomatic IDA who exhibited autonomic dysfunction manifesting as sinus bradycardia. Her haemoglobin level was 4.7 g/dL, with markedly low ferritin levels consistent with chronic iron deficiency. She was transfused with three units of packed red blood cells, after which her haemoglobin improved to 11.2 g/dL. Combined epidural–general anaesthesia was administered for tumour resection. Intraoperatively, episodes of bradycardia and hypotension occurred but were successfully managed with intravenous glycopyrrolate and fluid boluses. The postoperative course was uneventful, with no recurrence of bradycardia. The present case highlights the importance of recognising anaemia-induced autonomic dysfunction as a potential perioperative complication. Careful anaesthetic planning, preoperative optimisation, vigilant haemodynamic monitoring, and timely intervention can result in favourable outcomes in such challenging situations.

Keywords: Echocardiography, Electrocardiography, Heart conduction system, Haemodynamics, Hypotension, Oxygen consumption

CASE REPORT

A 54-year-old postmenopausal multiparous woman (G4P4L4) presented with dull, aching abdominal pain and constipation for 15 days. She also complained of generalised weakness and passage of bloody stools. There was no history of fever, weight loss, or chronic illness. On examination, the patient was alert and oriented. Her vital signs revealed a blood pressure of 130/80 mmHg and a pulse rate of 54 beats per minute, which was relatively low considering the severity of her anaemia. Cardiovascular and respiratory system examinations were unremarkable. Abdominal examination revealed a palpable solid-cystic mass extending from the infraumbilical region to the epigastrium, suggestive of a large abdominopelvic mass.

Initial laboratory investigations showed a haemoglobin level of 4.7 g/dL, serum ferritin of 4.0 ng/mL, and serum iron of 55 µg/dL. Vitamin B12 levels were elevated (1000 pg/mL), likely due to prior supplementation or an acute-phase response. Electrocardiography revealed sinus bradycardia (heart rate: 52/min) with flattening of T waves in lead aVL. Transthoracic echocardiography demonstrated a normal ejection fraction (60%), grade I diastolic dysfunction, and trivial mitral regurgitation. Contrast-enhanced Computed Tomography (CECT) of the abdomen and pelvis revealed a large right ovarian tumour with mild ascites and anterior displacement of the urinary bladder.

The bradycardia was not attributable to structural cardiac abnormalities or intrinsic conduction system disease, as evidenced by electrocardiographic and echocardiographic findings. Other reversible causes, including electrolyte imbalance, hypothyroidism, and medication effects, were systematically excluded through appropriate laboratory and clinical evaluation. The bradycardia was therefore attributed to autonomic dysfunction secondary to chronic, severe IDA, which can impair baroreceptor sensitivity and alter vagal tone, leading to paradoxical bradycardia and labile blood pressure responses.

Given the heightened perioperative risk associated with severe anaemia and autonomic instability, preoperative optimisation was prioritised. The patient received three units of packed red blood cells over 48 hours, resulting in an increase in haemoglobin to 11.2 g/dL. Following cardiology and general medical evaluations, she was deemed fit for surgery, with a heart rate of 62 beats per minute. Potential anaesthetic and intraoperative complications were explained in detail, and high-risk informed consent was obtained.

Considering the anticipated challenges, a combined epidural–general anaesthetic technique was planned to provide effective analgesia, attenuate the stress response, and allow titration of anaesthetic depth. Under strict aseptic precautions, an epidural catheter was placed at the L2-L3 intervertebral space using the loss-of-resistance technique. Premedication included intravenous glycopyrrolate (0.2 mg), midazolam (1 mg), and butorphanol (1 mg) to reduce vagal tone and provide anxiolysis and analgesia. Following preoxygenation, general anaesthesia was induced with propofol (100 mg) and vecuronium (6 mg). Tracheal intubation was performed using a 7.0 mm cuffed endotracheal tube. Anaesthesia was maintained with isoflurane in a 50:50 oxygen–nitrous oxide mixture, remifentanil infusion at 0.6 µg/kg/min, and intermittent boluses of vecuronium. After administration of a test dose, epidural boluses of 10 mL of 0.25% bupivacaine were given.

Central venous catheterisation of the right internal jugular vein was performed to facilitate central venous pressure monitoring and fluid management. During surgery, significant blood loss resulted in sudden-onset bradycardia (heart rate 37-39 bpm) and hypotension (blood pressure 70/50 mmHg). These were promptly managed with intravenous glycopyrrolate (0.2 mg) and fluid boluses, leading to stabilisation of haemodynamics (heart rate 78 bpm, blood pressure 100/70 mmHg). The estimated intraoperative blood loss was 650 mL, and two units of packed red blood cells

were transfused intraoperatively. The total duration of surgery was approximately four hours, and no further complications were encountered.

Following surgery, the patient was extubated and transferred to the intensive care unit for close monitoring. Epidural analgesia was continued using 0.125% bupivacaine at a rate of 10 mL/h to ensure effective pain control and attenuation of sympathetic stress responses. Two units of fresh frozen plasma were administered to correct any perioperative coagulopathy. No further episodes of bradycardia or hypotension were observed. She was shifted to the ward after 36 hours and showed steady clinical recovery. At one-month and three-month postoperative follow-up, her heart rate remained within normal limits, with complete resolution of bradycardia and no recurrence, indicating sustained autonomic stability and favourable long-term outcomes.

DISCUSSION

Bradycardia secondary to dysautonomia is a rare but clinically significant complication of severe IDA. While anaemia typically leads to tissue hypoxia and reduced oxygen delivery, resulting in compensatory tachycardia, paradoxical bradycardia may occur, particularly in chronic or severe cases. Dysautonomia, characterised by excessive vagal activity or diminished sympathetic tone, can override the expected chronotropic response. In the present case, the presence of sinus bradycardia in the absence of structural heart disease, combined with profoundly low ferritin levels, strongly suggested anaemia-induced autonomic dysfunction [1,2]. Other potential causes of vagal stimulation, such as surgical manipulation of visceral structures, endotracheal tube stimulation, peritoneal stretch, or high neuraxial blockade, were considered but were either absent or insufficient to account for the sustained bradycardia observed [3].

The mechanisms by which IDA impairs autonomic function are multifactorial and complex. Chronic anaemia can result in sustained hypoxic stress, adversely affecting the sinoatrial node and other components of the cardiac conduction system. Iron plays a crucial role in mitochondrial integrity and energy metabolism in cardiac myocytes; its deficiency may therefore compromise electrophysiological stability. Additionally, iron deficiency impairs baroreceptor sensitivity and autonomic reflexes. Reduced myoglobin availability in both cardiac and skeletal muscle further limits oxygen utilisation, potentially suppressing sinoatrial node activity. Chronic sympathetic underactivity combined with increased central vagal outflow, possibly due to altered neurotransmitter metabolism in iron-deficient states, provides a plausible mechanistic link between IDA and bradycardia. These changes may manifest clinically as bradycardia, orthostatic hypotension, or other atypical cardiovascular responses, particularly under perioperative stress [4,5].

The IDA may contribute to bradycardia through multiple interrelated mechanisms. Autonomic imbalance, characterised by impaired sympathetic activity and exaggerated vagal tone, has been documented in iron-deficient states, predisposing patients to sinus node suppression and reduced heart rate. Iron is essential for maintaining mitochondrial function and optimal energy metabolism in cardiac myocytes; deficiency compromises the energy supply required for normal pacemaker activity. Furthermore, alterations in intracellular calcium handling, including Reduced ryanodine Receptor (RyR2) expression and diminished Sarco/endoplasmic Reticulum Ca^{2+}-ATPase (SERCA) pump activity, impair excitation–contraction coupling and further depress sinoatrial node function. These pathophysiological changes may act synergistically, resulting in paradoxical bradycardia despite the hypoxic stimulus of anaemia, particularly in chronic or severe cases [6-8].

From an anaesthetic perspective, such patients require meticulous attention and thorough preoperative optimisation, with emphasis on correcting anaemia to restore oxygen-carrying capacity and prevent maladaptive physiological responses. Packed red blood cell transfusion increased the haemoglobin concentration to a safer level, enabling more stable intraoperative haemodynamics. Anaesthetic management focused on minimising sympathetic fluctuations while ensuring adequate analgesia without excessive cardiovascular depression. A combined epidural-general anaesthetic technique was therefore selected. Epidural anaesthesia provided effective analgesia and reduced systemic anaesthetic requirements, while general anaesthesia ensured airway protection and optimal surgical conditions. Agents known to precipitate bradycardia, such as opioids, were administered cautiously in titrated doses. Glycopyrrolate was used both as a vagolytic premedication and as a rescue agent during intraoperative bradycardia [9,10].

During surgery, the patient experienced a severe episode of bradycardia, most likely precipitated by vagal stimulation superimposed on underlying autonomic dysfunction. Prompt administration of intravenous anticholinergic agents and fluid boluses successfully stabilised haemodynamic parameters. This event underscores the importance of continuous intraoperative monitoring and preparedness to manage acute autonomic disturbances. Invasive haemodynamic monitoring is advisable in such high-risk cases. The present case adds to the limited existing literature and reinforces the need for clinicians to recognise and anticipate autonomic instability in anaemic patients presenting with unexplained bradycardia [11].

CONCLUSION(S)

Anaemia-induced autonomic dysfunction is a rare but potentially serious perioperative condition. The present case highlights the need for heightened clinical suspicion when encountering atypical cardiovascular presentations, such as bradycardia, in patients with severe iron deficiency anaemia. Comprehensive preoperative evaluation, timely correction of anaemia, and individualised anaesthetic planning are crucial to minimising perioperative morbidity. Vigilant monitoring and prompt management of autonomic instability can significantly improve outcomes. Increased awareness of this uncommon entity will assist Anaesthesiologists and surgeons in delivering safer perioperative care, particularly during major abdominal surgeries.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: May 13, 2025
- Manual Googling: Sep 15, 2025
- iThenticate Software: Sep 17, 2025 (2%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Apr 23, 2025**
Date of Peer Review: **Jul 26, 2025**
Date of Acceptance: **Sep 20, 2025**
Date of Publishing: **Apr 01, 2026**