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Electrocardiographic Changes after General Anaesthesia Induction in a Patient with Subarachnoid Haemorrhage and Thyroid Heart Disease: A Case Report

Perubahan Elektrokardiografik Setelah Induksi Anestesi Umum pada Pasien Perdarahan Subarachnoid dengan Penyakit Jantung Tiroid: Laporan Kasus

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Abstract

Electrocardiographic (ECG) abnormalities are commonly observed in patients with subarachnoid hemorrhage (SAH) and patients and are linked to worse outcomes. This case report documents the rapid normalization of severe ECG abnormalities following general anesthesia induction in a high-risk SAH patient with hyperthyroid heart disease. We report an 81-year-old woman with Fisher grade IV SAH and hyperthyroidism-associated cardiomyopathy who presented obtunded (GCS 7) with refractory atrial fibrillation at 116 bpm, diffuse T-wave inversions, and elevated troponin I. Following a multidisciplinary discussion, an External Ventricular Drain (EVD) was placed under general anesthesia using fentanyl, propofol, and rocuronium. This resulted in the immediate restoration of sinus rhythm, resolution of repolarization changes, and stabilization of blood pressure. Despite comprehensive ICU management—including analgesia, sedation, osmotherapy, steroids, and diuretics—the patient's condition deteriorated within 24 hours, developing refractory intracranial hypertension and leading to death. This case highlights the autonomic-modulating and antiarrhythmic properties of anaesthetic agents in SAH-related neurocardiogenic dysfunction and underscores the need for further research into optimal anaesthesia strategies and patient outcomes.

Keywords: atrial fibrillation; fentanyl; propofol; subarachnoid haemorrhage

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Abstrak

Kelainan elektrokardiografi (EKG) umum dijumpai pada pasien dengan perdarahan subaraknoid (PSA) dan berhubungan dengan luaran yang lebih buruk. Laporan kasus ini mendokumentasikan normalisasi yang cepat dari kelainan EKG yang parah setelah induksi anestesi umum pada pasien PSA berisiko tinggi dengan penyakit jantung hipertiroid. Seorang wanita berusia 81 tahun dengan PSA *grade* IV menurut klasifikasi Fisher dan kardiomiopati terkait hipertiroidisme, yang datang dalam kondisi obtundasi (GCS 7) dengan fibrilasi atrium refrakter pada 116 denyut per menit, inversi gelombang T yang difus, dan peningkatan troponin I. Setelah diskusi multidisiplin, dipasang *External Ventricular Drain* (EVD) di bawah anestesi umum menggunakan fentanyl, propofol, dan rokuronium. Hal ini mengakibatkan pemulihan irama sinus secara langsung, resolusi perubahan repolarisasi, dan stabilisasi tekanan darah. Meskipun telah dilakukan penatalaksanaan ICU yang komprehensif, termasuk analgesia, sedasi, osmoterapi, steroid, dan diuretik, kondisi pasien memburuk dalam waktu 24 jam, mengalami hipertensi intrakranial yang refrakter dan menyebabkan kematian. Kasus ini fokus pada sifat modulasi otonom dan antiaritmia dari agen anestesi pada disfungsi neurokardiogenik yang terkait PSA dan menekankan perlunya penelitian lebih lanjut mengenai strategi anestesi yang optimal dan luaran pasien.

Kata kunci: anestesi; fentanyl; fibrilasi atrium; perdarahan *subarachnoid*; propofol

Introduction

Subarachnoid hemorrhage (SAH), can be caused by trauma or spontaneously due to aneurysmal rupture. Incidence of spontaneous aneurysmal SAH adds up to 3–23 cases per 100,000/year, with a relatively high mortality rate (18.4%).¹ SAH management includes nimodipine, triple-H therapy (hypertension, hypervolemia, and hemodilution), balloon angioplasty, cerebral aneurysm coiling or clipping, or external ventricular drain (EVD) placement. EVD is indicated in patients with increased intracranial pressure (ICP) and hydrocephalus after SAH to alleviate the pressure. SAH surgery must consider the preservation of cerebral perfusion pressure, maintenance of ICP, and optimal surgical exposure without much brain retraction. Prevention of ICP swing during surgery can be anticipated using sufficient hypnotics such as propofol, opioids such as fentanyl, non-depolarizing neuromuscular blockers, and local anesthetic infiltration of the sites of pin placement.²

Electrocardiography (ECG) changes are commonly seen in SAH patients. However, the occurrence of fatal arrhythmias remains low, with atrial fibrillation (AF) reported in only 1–4% of all arrhythmia cases.³ ECG changes in SAH are frequently associated with a poorer prognosis across various studies, with high mortality and morbidity due to dysfunction of essential organs, including the brain, heart, lungs, and kidneys. The resolution of ECG changes is typically reported to occur within days up to 6 weeks.^{4,5}

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Rhythm changes can also be attributed to hyperthyroidism, which happens in 0.5–4% in the elderly.⁶ However, the ECG alteration in SAH is attributed to the catecholamine surge resulting from the disruption of the hypothalamus-pituitary-adrenal axis, which is closely linked with increased intracranial pressure (ICP).⁷ Catecholamine surge is also linked to increased pain, which subsequently can cause arrhythmia through autonomic nervous system disturbance.^{8,9} The return of sympathetic-parasympathetic homeostasis and pain management through the administration of general anesthesia could theoretically reverse the ECG alteration. Moreover several studies reported that general anesthesia, like propofol and fentanyl, has antiarrhythmic properties.^{10,11}

What distinguishes this case from previously published reports is the immediate and dramatic normalization of refractory atrial fibrillation and ischemic ECG changes directly following general anesthesia induction in a patient with SAH complicated by previously asymptomatic thyroid heart disease. While prior case reports and studies describe gradual ECG improvement over days or weeks, rapid reversal during anesthesia induction has not been systematically documented.^{10,11} This observation not only highlights the potential neurocardiogenic mechanisms involved, but also suggests a novel clinical consideration for anesthesia management in SAH patients with complex cardiac comorbidities. Therefore this case provides unique insight, warranting dissemination to broaden understanding and guide future management strategies in similar high-risk patients.

This case study presented a case of an SAH patient with previously asymptomatic thyroid heart disease who experienced immediate ECG normalization following the administration of propofol and fentanyl.

Case Illustration

An 81-year-old female was brought to the emergency department in an unconscious state after experiencing a severe headache and generalized seizure. Her medical history included uncontrolled hypertension. The family denied any history of palpitations, sweating, or tremors. Physical examination revealed a blood pressure of 167/103 mmHg refractory to nifedipine, a heart rate of 110 beats per minute, a respiratory rate of 26 breaths per minute, and an oxygen saturation of 98% on a 15 lpm non-rebreathing mask. The GCS was E2V2M3, with no sign of lateralization. Pupils were equal in size, with sluggish direct and indirect light reflexes. Blood chemistry indicated hypokalaemia, elevated FT4 consistent with thyroid heart disease, and increased troponin I levels, along with ECG findings of T-wave inversions in leads V3-V6, consistent with non-ST elevation myocardial infarction (NSTEMI). Laboratory results are

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summarized in Table 1. ECG showed rapid ventricular response AF (RVRAF) refractory to intravenous digoxin (Figures 1 and 2). The non-contrast head CT scan revealed SAH classified as Fisher grade IV (Figure 3).

Table 1 Admission Laboratory Values in This Case

Parameter	Result	Unit	Normal Value
Hematology			
Hemoglobin	12	g/dL	11.5–15
Hematocrit	35	%	35–45
Leukocyte	19.67 (H)	10 ³ /uL	3.5–9.5
Basophil	0.1	%	0–1
Eosinophil	0 (L)	%	0.4–8
Neutrophil	93.5 (H)	%	40–75
Lymphocyte	4.7 (L)	%	20–50
Monocyte	1.7 (L)	%	3–10
Thrombocyte	192	10 ³ /uL	150–350
Electrolyte			
Natrium (Na)	130.9 (L)	mmol/L	136–145
Kalium (K)	3.45 (L)	mmol/L	3.5–5.5
Chloride (Cl)	111.8 (H)	mmol/L	96–108
Blood chemistry			
Creatinine	0.74	mg/dL	0.5–0.9
Glucose	140	mg/dL	70–140
Urea	28	mg/dL	15–40
TSH	< 0.05	uIU/mL	0.25–5
FT4	62.52 (H)	pmol/L	10.6–19.4
Troponin I HS	342.6	ng/L	

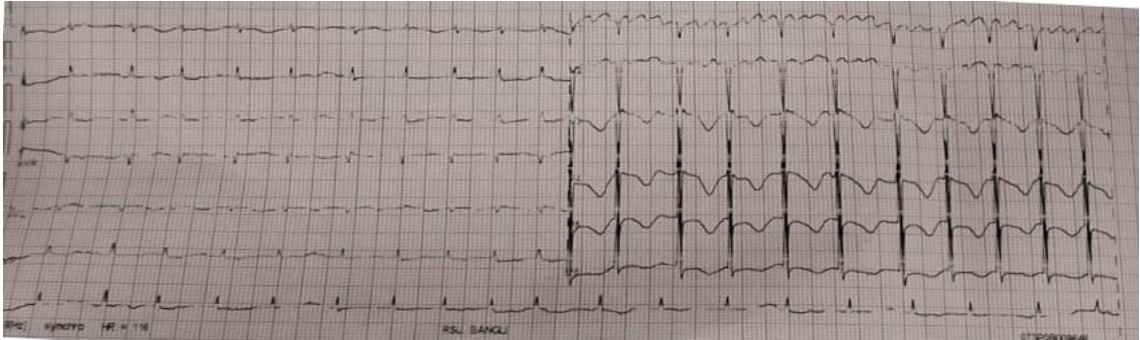


Figure 1 Initial ECG on Admission Showing RVRAF

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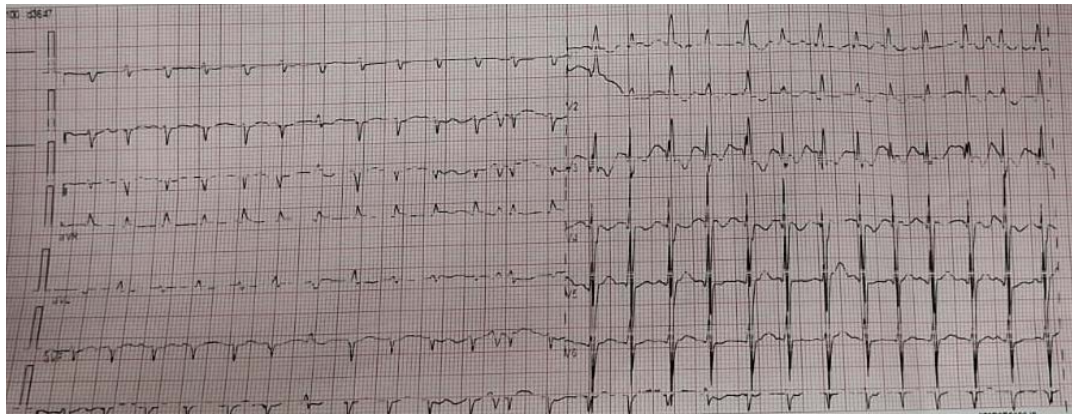


Figure 2 ECG Two-Hour Post-Digoxin Administration Showing Persistent RVRAF



Figure 3 Non-Contrast Head CT Scan Showing Hyperdensity Through The Subarachnoid Space, Lateral, Third, and Fourth Ventricles, and Hydrocephalus, Consistent with Fisher Grade IV

The patient was diagnosed with SAH Fisher grade IV and Hunt-Hess grade IV, NSTEMI, RVRAF, hypokalemia, and thyroid heart disease, with ASA score of 4E. Intravenous isosorbide dinitrate (ISDN) was initiated at a rate of 2 mg/hour, along with continuous intravenous furosemide at 5 mg/hour. A loading dose of digoxin 0.5 mg IV was administered, followed by a maintenance dose of 0.25 mg IV every 6 hours. Placement of an external ventricular drain (EVD) was planned subsequently. The cardiologist initially recommended postponing the EVD insertion; however, the procedure was carried out following discussions with the patient's family. Anaesthesia induction was done using 4 mcg/kgBW fentanyl, 30 mg propofol, and 0.8 mg/kgBW rocuronium. Immediately following induction, the heart rhythm, T-wave inversions, and blood pressure normalized to 120/60 mmHg, allowing EVD insertion. The EVD was inserted at the right Kocher point, and ICP reduction was achieved, marked by the

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return of brain pulsations. Postoperatively, the patient was managed in the ICU with fentanyl 0.25 mcg/kgBW/hour continuous IV, dexmedetomidine 0.4 mcg/kgBW/hour continuous IV, dexamethasone 10 mg three times daily, mannitol 6x100 cc, furosemide 20 mg three times daily, paracetamol 1 g three times daily, ibuprofen 400 mg three times daily and lansoprazole 30 mg twice daily. Despite these measures, the patient's condition deteriorated within 24 hours, leading to death.



Figure 4 ECG 2 Hours Post-Surgery Showing Normalization of Heart Rhythm, Rate, And T-Wave Inversions

Discussion

In this case, an 81-year-old patient with subarachnoid hemorrhage (SAH) complicated by thyroid heart disease presented with RVRAF and NSTEMI, which resolved immediately after induction of general anesthesia using propofol and fentanyl. This rapid normalization of ECG abnormalities and hemodynamic stabilization was observed concurrently with the administration of these anesthetic agents.

Previous case reports and studies have described various cardiac disturbances following SAH, including repolarization abnormalities, arrhythmias, and myocardial injury triggered by

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increased intracranial pressure (ICP). These reports generally describe a gradual resolution of ECG changes over days to weeks rather than an immediate reversal during anesthesia induction. For example, studies have documented that ECG abnormalities in SAH patients often persist and resolve slowly as ICP normalizes and neurocardiogenic stress decreases.^{4,5}

What sets this case apart is the immediate reversal of refractory atrial fibrillation and ischemic ECG changes directly following induction with propofol and fentanyl, suggesting an acute pharmacologic effect rather than spontaneous recovery. This observation has not been widely reported or systematically studied in patients with SAH, particularly those with coexisting thyroid heart disease, which can itself predispose to arrhythmias.

The high mortality rate associated with SAH is primarily due to two factors: delayed cerebral ischemia and multi-organ dysfunctions.¹² SAH can induce cardiac disturbances manifesting as neurocardiogenic symptoms, including repolarization abnormalities, arrhythmias, and myocardial injury.¹³ These symptoms arise from SAH-induced elevated ICP, which can cause hypothalamic ischemia. Elevated ICP can be seen in our patient through the elevated blood pressure, which was refractory to nifedipine, tachycardia, and tachypnea.

Hypothalamic ischemia disrupts the hypothalamic-pituitary-adrenal axis due to the impairment of the hypothalamic paraventricular nucleus (PVN), which is responsible for activating the axis, resulting in a surge of catecholamine and an imbalance in the sympathetic and parasympathetic tone.¹² The heightened sympathetic activity can also cause myocardial injury through an inflammatory cascade and calcium influx into myocytes, leading to sustained contraction, oxidative stress, and mitochondrial dysfunction. All of these conditions contribute to myocyte necrosis, also known as contraction band necrosis.¹³

Our patient presenting with RVRAF and NSTEMI following SAH was consistent with the aforementioned pathophysiology. While our patient's hyperthyroidism could be a contributing factor to the RVRAF, the resolution of AF following propofol and fentanyl injection suggested that hyperthyroidism was not the primary cause, though it may have exacerbated the arrhythmia.⁶

Catecholamine surge also contributed to heightened pain in the patient by activating pain receptors and modulating neuronal excitability.⁸ Both acute and chronic pain are associated with an increased risk of AF as they disturb the autonomic nervous system.⁹ Consequently, the administration of fentanyl, a mu-opioid receptor agonist analgesic, might also resolve the AF in our patient by addressing the underlying pain.

The normalization of ECG changes following anesthesia induction with propofol and fentanyl also underscores the restoration of the sympathetic and parasympathetic regulation alongside the re-establishment of the hypothalamic-pituitary-adrenal axis, demonstrating the

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cardio-electrophysiologic effect of these drugs.^{5,7,13} Propofol exerts a sympatholytic effect by blocking baroreceptors and depressing the cardiac conduction system, leading to a reduction in heart rate. Both animal and human studies have shown that propofol slows conduction through the AV node, His-Purkinje system, and sinus node.¹¹ Additionally, propofol prolongs atrial action potential duration and inhibits multiple atrial repolarization channels, including the ultra-rapidly activating delayed rectifier potassium current (I_{Kur}), transient outward potassium current (I_{to}), and calcium current (I_{Ca}), which collectively help suppress the formation of atrial fibrillation.¹⁴⁻¹⁷

Fentanyl is also believed to possess antiarrhythmic and cardioprotective properties. A study in anesthetized dogs using fentanyl demonstrated an increased ventricular fibrillation threshold. Furthermore, opioid peptides enhance resistance to catecholamine-induced arrhythmias, including atrial fibrillation, and stabilize heart electrical conduction by producing nitric oxide and improving myocardial blood flow.¹⁵⁻¹⁹

Numerous case series and studies have documented the cardio-electrophysiology effects of remifentanyl, another mu-opioid receptor agonist. Remifentanyl can convert atrial fibrillation to a normal sinus rhythm by suppressing both the sinus and atrioventricular nodes, as well as increasing the action potential threshold of myocytes through the inhibition of cardiac calcium and potassium channels.^{11,17,20} Given that both remifentanyl and fentanyl are mu-opioid receptor agonists, it is reasonable to hypothesize that fentanyl may also possess similar antiarrhythmic properties as remifentanyl.

The immediate resolution of ECG abnormalities in our patient following propofol and fentanyl administration highlights the potential antiarrhythmic effects of these agents in SAH patients. This case adds to the limited evidence and is, to our knowledge, the first documented report demonstrating such acute electrophysiological improvement associated with anesthesia induction in this patient population. Further research is warranted to elucidate the underlying mechanisms and explore therapeutic implications.

Conclusion

The rapid resolution of ECG abnormalities in this SAH patient following general anesthesia with propofol and fentanyl suggests a potential role for these agents in modulating neurocardiogenic arrhythmias. However, as this report is based on a single case, further research is necessary to confirm these findings and better understand the underlying mechanisms.

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