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Unanticipated Ventricular Fibrillation During Central Line Insertion In A Neurosurgery Patient: A Case Report

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Abstract
Durring neurosurgical procedure, Central venous catheterization is frequently required for the safe administration of medications that may damage peripheral veins, for administration of ionotropes or vasopressors, for the placement of temporary transvenous pacemakers, and for providing invasive hemodynamic monitoring. Although various complications are well-documented with the insertion of central venous catheters, reports of cardiac arrest during their placement remain rare. We present a unique case of sustained ventricular fibrillation and subsequent cardiac arrest occurring during ultrasound-guided central venous catheterization in a patient undergoing surgery for a brain tumor. This case highlights a rare yet potentially fatal complication of central venous access placement, particularly in neurosurgical patients, and underscores the importance of prompt and effective management.

Keywords: *debrillation, cardiac arrest, heart-brain crosstalk, brain tumour, neurosurgery, ventricullar fibrillation, central venous cannulation*

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I. Introduction

Heart-brain cross-talk is a growing field of research, with many case reports describing cardiovascular manifestations associated with neurological insults since the 1900s [1]. While intraoperative cardiac arrest during skull pin application, positioning, and tumour handling in neurosurgical patients is well-documented, ventricular fibrillation leading to cardiac arrest during central venous catheter (CVC) insertion in neurosurgery patients under anesthesia is rarely mentioned.

Central venous catheterization, in neurosurgery patients commonly performed for central venous pressure monitoring, administration of vasoactive drugs and fluids, and transvenous pacing, can be done using a landmark-guided technique or under ultrasound guidance [2]. The Seldinger method, often used for this procedure, involves exchanging catheters over a guide wire. If the guide wire is unintentionally threaded into the heart chambers, it can irritate the myocardium, causing ventricular arrhythmias [3]. These arrhythmias are usually transient and resolve with a slight guidewire withdrawal.

Here, we describe an unusual case of sustained ventricular fibrillation and subsequent cardiac arrest during ultrasound-guided central venous catheter placement in a neurosurgery patient. This case highlights albeit rare, but potentially fatal complications of central venous access in brain tumor patients and warrants a high level of concern by anesthesiologists. Moreover, knowledge of the risk factors and early treatment can minimize the high mortality risk.

II. Case Presentation

A 45-year-old male, weighing 60 kg and standing 170 cm tall (BMI: 20.76 kg/m²), presented to the neurosurgery outpatient department with a history of headaches for the past month. A magnetic resonance imaging (MRI) revealed a left parietal high-grade glioma without mass effect. The patient had no significant medical or surgical history and was scheduled for an elective left parietal craniotomy and lesion excision.

During the pre-anesthetic checkup, his Glasgow coma scale (GCS) score was E4V5M6. He was afebrile, with a heart rate (HR) of 70 bpm and blood pressure (BP) of 124/72 mm Hg. Central nervous examination showed orientation to time, place, and person, with no focal neurological deficits. The

cardiorespiratory examination was normal, and baseline biochemical and hematological investigations were within normal limits. (Table 1). ECG showed normal sinus rhythm. (Figure 1)

Serial Number	Parameter	Value	Reference range
1.	Haemoglobin	13.4 g/dL	12-15 g/dL
2.	White blood cells	7200 cells per microliter	4000-11000 cells per microliter
3.	Platelets	222000 per microliter	150000-400000 cells per microliter
4.	Blood urea	25 mg/dL	10-39 mg/dL
5.	Serum creatinine	1.1 mg/dL	0.6-1.3 mg/dL
6.	Serum sodium	137 mEq/L	135-145 mEq/L
7.	Serum potassium	4.1 mEq/L	3.5-5 mEq/L
8.	Serum chloride	101 mEq/L	92-106 mEq/L
9.	Prothrombin time	10.5 seconds	9-12 seconds
10.	INR	1.1	0.9-1.2
11.	aPTT	29 seconds	24-32 seconds

TABLE 1: Preoperative biochemical and hematological investigations.

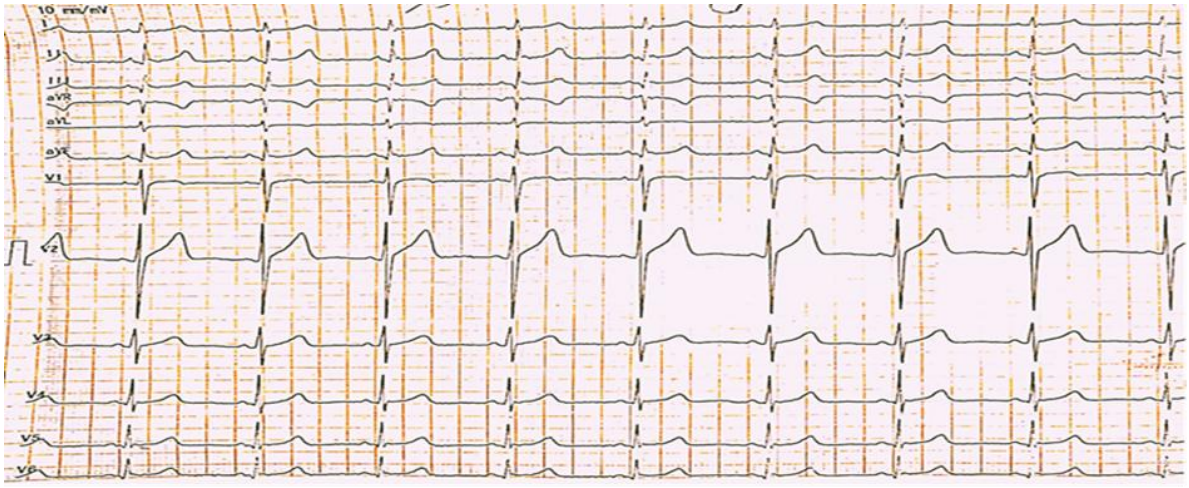

FIGURE 1: Preoperative ECG, showing normal sinus rhytham.

The patient was cleared for surgery under American Society of Anesthesiologists (ASA) Physical Status II. Routine premedication included alprazolam 0.25 mg and pantoprazole 40 mg the night before, with another dose of pantoprazole on the day of surgery. The patient and family were informed about the prognosis and potential need for postoperative mechanical ventilation. After confirming nil by mouth status on the morning of surgery, the patient shifted to the operation theatre. ASA standard, monitors were attached, including a 5-lead electrocardiogram (ECG), pulse oximetry, temperature probe, and non-invasive

BP monitoring. An arterial cannula was placed in the left radial artery under local anesthesia with 2% lignocaine. After pre-oxygenating with 100% oxygen for 3-5 minutes, general anesthesia was induced using injection propofol (100 mg), fentanyl (120 mcg), and vecuronium (6 mg) and intubated with a 7.5 mm standard cuffed endotracheal tube. Anaesthesia was maintained with 50% oxygen, 50% air, and isoflurane with a minimum alveolar concentration (MAC) of 0.8-0.9.

Due to the tumor's proximity to major blood vessels, a central venous catheter (CVC)(Arrow Polyurethane Triple Lumen, 7Fr x 16 cm, J tip guidewire, 60 cm, Czech Republic) was placed for potential inotrope support and fluid resuscitation. The patient was positioned in Trendelenburg (20 degrees) with the head tilted left (45 degrees), and the right side of the neck was prepped for internal jugular vein (IJV) cannulation with anterior approach by the anesthesia resident. The procedure took around 5 minutes. Vitals during cannulation were HR 94/min, BP 136/84 mm Hg, and ECG showed sinus rhythm. Under aseptic precautions and ultrasound guidance (Sonosite II, Fujifilm, USA), the right IJV was cannulated. The guidewire was inserted after confirming backflow.

While inserting the guidewire at a depth of about 20 cm, ventricular premature complexes (VPC) appeared on the monitor. The guidewire was withdrawn 4-5 cm until the VPCs disappeared. However, while threading the CVC catheter over the guidewire, ventricular tachycardia occurred with a HR of 164 bpm and BP of 56/28 mm Hg. The guidewire and CVC catheter were immediately removed, but the rhythm progressed to ventricular fibrillation, leading to cardiac arrest before start of surgery. (Figure 2).

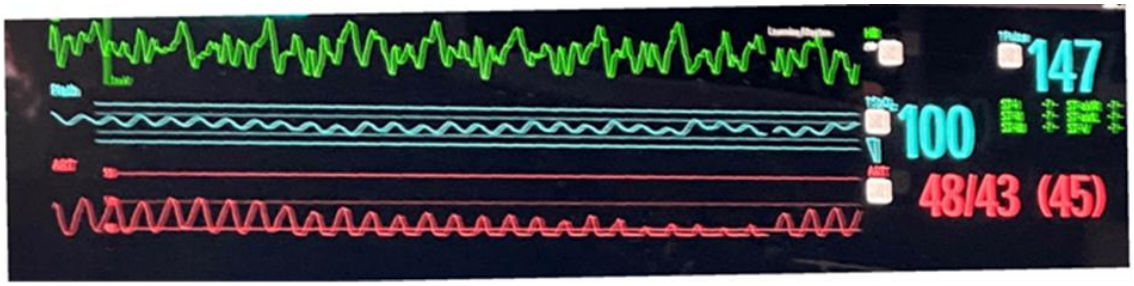

FIGURE 2: Intraoperative ECG, showing ventricular fibrillation with haemodynamic instability.

Chest compressions were started, 100% oxygen was administered, and preparations for external defibrillation began. Defibrillator pads were placed in the right infraclavicular and left inframammary regions. A 200-joule biphasic shock was delivered, resulting in sinus rhythm and return of spontaneous circulation (ROSC). The table and the patient's head were returned to a neutral position. Injection lignocaine 90 mg was administered. An arterial blood gas test showed normal results. Injection magnesium sulfate 2 g. IV was given over 30 minutes. Meanwhile, cardiologist called inside OT, and intraoperative transthoracic echocardiography showed normal cardiac contractility with no regional wall motion abnormalities. The patient's relatives were informed about the incident. Following a detailed cardiac evaluation and documentation, a joint decision was made by the neurosurgeon, anesthesiologist, cardiologist, and the patient's relatives to proceed with the surgery. The patient's guardian was provided with information regarding the high risk of perioperative major adverse cardiovascular events, and high-risk informed consent was obtained from patients' relatives before proceeding surgery. Before start of surgery, patient was again monitored for 30 minutes, during which a central venous cannula was placed in the right femoral vein under ultrasound guidance.

Intraoperatively, the patient was maintained with 50% oxygen, 50% air, and isoflurane with a MAC of 0.8-0.9. Analgesia was maintained with a fentanyl infusion (10 mcg/hr), and muscle relaxation with vecuronium (1 mg/hr). The surgery lasted for four hours without complications. Post-procedure, arterial blood gas analysis showed normal results.

Postoperatively patient shifted to the neurosurgery intensive care unit (ICU), where vital signs and neurological status were monitored using the GCS. Once the patient was fully awake, alert, and responsive to verbal commands with complete motor recovery, extubation was performed on postoperative day one. The postoperative GCS was 15/15, and this was monitored daily for five days. During ICU stay, a 12-lead ECG and transthoracic echocardiogram were performed, revealing no cardiac abnormalities. (Figure 3)

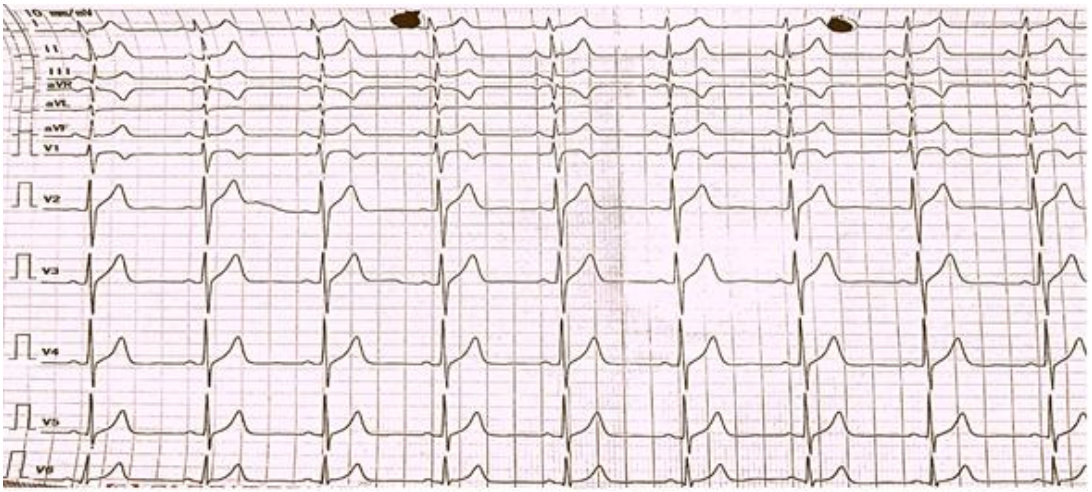

FIGURE 3: Postoperative ECG, showing normal sinus rhytham.

Postoperative electrolyte levels (sodium, potassium, magnesium, calcium, phosphorus) were within normal limits. The patient had an uneventful recovery and was discharged on day 7. On follow-up after 1 month, the patient was doing well without any neurological deficit.

III. Discussion

The heart can be influence and affected by neurological conditions like, acute ischemic stroke (AIS), traumatic brain injury (TBI), and intracranial space-occupying lesions [1]. Brain tumors have been linked to atrial fibrillation (AF), bradycardia, and cardiac syncope [4]. Brain tumor patients undergoing neurosurgical procedures often have an irritable myocardium due to the complex brain-heart interactions. This is a consequence of the neuroanatomical connections between the central nervous system and the cardiovascular system, involving the parasympathetic ganglia and the intermediolateral gray columns of the spinal cord, which influence heart rate and rhythm. The right side of the brain primarily controls heart rate, while the left side is more associated with the development of arrhythmias. In patients with right-sided strokes, the loss of parasympathetic control on the right side is associated with sinus tachyarrhythmias, whereas patients with left insular strokes are typically characterized by sympathetic predominance, potentially resulting in cardiac dysrhythmias. [5,6] Moreover, activation of the hypothalamic-pituitary- adrenal (HPA) axis triggers a catecholamine surge by stimulating the sympathetic chain of ganglia at the spinal cord level. Sympathetic neurons extend through the cervical and upper thoracic ganglia to form post- ganglionic neurons that innervate the sinoatrial and atrioventricular nodes. The resultant activation of the cardiac sympathetic fibers stimulates the baroreceptors, sending activating signals back to the medulla, thus forming a self-propagating cycle that relays signals back to the heart via autonomic nerves, resulting in a catecholamine storm. These catecholamines can cause cardiac damage and induce various types of arrhythmias in these patients [1,5,6]. Likewise, our patient, with a long-standing left parietal high-grade glioma, likely had increased cardiac susceptibility for arrhythmias.

During the placement of central venous pressure catheters or the manipulation of guidewires, the further stimulation of this already irritable myocardium can lead to rare but serious consequences, such as life-threatening arrhythmias and even cardiac arrest. Arrhythmias can typically arise from direct contact of the guidewire with the sinoatrial node, right atrium, atrioventricular node, or right ventricle. While these arrhythmias are often self-limiting, the risk of developing potentially fatal ventricular arrhythmias, although less than 1%, is still significant and requires close monitoring and prompt intervention [2,3].

Studies have reported an incidence of arrhythmias during central venous catheter (CVC) insertion in adult patients, with rates of 14.5% for atrial arrhythmias and 3.6% for ventricular arrhythmias [7]. Furthermore, Arafa Ramadan et al. documented a case of asystole during catheter insertion that necessitated the implantation of a pacemaker, demonstrating that while guidewire-related complications are uncommon, they can still lead to substantial morbidity and even mortality in these vulnerable patients [8].

Surgery and anesthesia both stress the patient. Laryngoscopy and endotracheal intubation trigger reflex sympathetic stimulation, significantly increasing serum epinephrine and norepinephrine levels. This can cause complications such as ventricular arrhythmia, myocardial ischemia, left ventricular failure, a sudden rise in intracranial pressure, and acute cerebral edema or brain herniation. The pressor response, characterized by this heightened sympathetic drive, can persist for 5-10 minutes following intubation [9]. During this high serum catecholamine period, stimulating the heart with a guidewire can trigger catastrophic, life-threatening cardiac arrhythmias. We cannulated the internal jugular vein (IJV), 5 minutes after intubation, and direct stimulation of the endocardium by the guidewire likely precipitated the observed ventricular fibrillation.

Routinely used anesthetic drugs may induce ventricular arrhythmias. Propofol has been demonstrated to exhibit both proarrhythmic and antiarrhythmic properties [10]. Alone fentanyl or vecuronium as such does not affect cardiac conduction but given a combination may produce bradycardia. Isoflurane may prolong QT and QTc interval leading to arrhythmia but less as compared to sevoflurane and desflurane [11]. Therefore, in our case except for isoflurane, it is unlikely that other agents had caused arrhythmia. Also, no drug bolus or new drug was given at the time of CVC cannulation.

Patients with elevated intracranial pressure (ICP) due to brain tumors have a high chances of ECG changes and arrhythmias. Sethuraman et al. found that 32% of these patients experienced ECG changes, compared to 6% of those without elevated ICP [12]. Additionally, cardiovascular disturbances can be affected by changes in intracranial pressure (ICP) related to head and neck positioning, particularly in patients lying on the steep portion of the intracranial compliance curve. Mavrocordatos et al. investigated the effects of various positions on ICP, noting that positions with the head lowered or rotated increased ICP [13]. In our patient, during right internal jugular vein (IJV) cannulation, we positioned the head slightly lowered (15 to 20°) and rotated to the left (30-45 degrees) to facilitate IJV enlargement. This maneuver could potentially raise ICP, thereby increasing susceptibility to arrhythmias upon direct guide wire stimulation of the myocardium.

In a study by Tak-Yu Lee et al. they identified significant risk factors for guidewire-induced arrhythmias, including guidewire insertion length over 20 cm, height below 170 cm, and female gender [7].

They recommended using a marked J wire to limit guidewire insertion to 20 cm or less or employing fluoroscopic guidance during catheter insertion. Our patient was a male of height 170 cm, and had a marked J wire inserted up to the maximum limit of 20 cm. However, the substantial length of double and triple-lumen catheters, along with their guide wires, can lead to over-insertion, especially in inexperienced hands.

Additionally, catheter manipulation can inadvertently displace the wire despite the operator's perception of firm placement. To enhance procedural safety, various methods are recommended for precise guidance on the optimal guidewire or CVC insertion depth [14]. These range from simple height-based formulas to advanced techniques such as fluoroscopy, intra-atrial electrocardiography, transesophageal echocardiography, and transthoracic echocardiography [14,15,16]. For clinical practice, Saugel B, et al. recommend a six-step systematic approach for ultrasonography(USG)-guided central venous access [17]. Implementing these can significantly improve procedural safety.

Ventricular fibrillation is a critical cardiac arrhythmia that necessitates immediate defibrillation. The guidelines recommend early defibrillation with 120-200 joules using a biphasic defibrillator or 360 joules with a monophasic defibrillator. Prompt defibrillation improves survival significantly compared to delays of 2 minutes or more [16]. In our case, the patient received immediate defibrillation along with cardiopulmonary resuscitation, leading to the return of spontaneous circulation.

Therefore to ensure optimal patient safety, we suggest that the availability and functionality of a defibrillator be added to the world health organization (WHO)- surgical safety checklist, not only for procedures such as CVC insertion in neurosurgery patients, but for any medical procedure. Additionally, the initial evaluation should include an electrocardiogram and serum electrolyte assessment to detect any underlying conditions or disturbances that may predispose the patient to cardiac rhythm abnormalities [18]. This comprehensive approach can help identify and address potential risk factors, ultimately improving the patient's chances of a favorable outcome.

This report emphasizes the importance of exercising caution during the placement of central venous catheters under anesthesia. While conclusive evidence regarding the optimal positioning of the catheter tip is lacking, it is advisable to avoid advancing the catheter beyond the necessary depth, taking into account factors such as the puncture site (jugular or subclavian), patient positioning, and anatomical variations. Moreover, a thorough understanding of potential complications is essential for their prompt diagnosis, timely intervention, and effective resolution.

IV. Conclusions

This case report underscores the rare but potentially fatal complication of ventricular fibrillation during central venous catheter insertion in a neurosurgery patient. Heart-brain cross-talk and the inherent cardiac susceptibility in brain tumor patients necessitate heightened vigilance. Measures such as deepening anesthesia during intubation to avoid stress responses, proper positioning, and using ultrasound, electrocardiogram, or transesophageal echocardiography guidance for central venous catheter insertion help prevent stimulation of the irritable myocardium by the guidewire, thereby reducing the risk of life- threatening arrhythmias. Proper technique, continuous cardiac monitoring, and immediate access to defibrillation equipment are crucial. Prompt initiation of advanced cardiac life support protocols is essential for managing sudden cardiac arrest. A personalized approach that considers patient-specific factors and neurocardiogenic interactions, is crucial to reducing life-threatening events and improving outcomes in neurosurgical patients.

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