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TRANSCUTANEOUS PACING IN PERIPHERAL SETUP

Cardiology

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ABSTRACT

Transcutaneous cardiac pacing is a temporary method of pacing that may be indicated in patients with severe symptomatic or hemodynamically unstable bradyarrhythmias. It is particularly helpful in patients with reversible or transient conditions atrioventricular block in the setting of inferior wall myocardial infarction, or when transvenous pacing is not immediately available as in a peripheral setup. The use of transcutaneous pacing for a prolonged duration to bridge and manage bradyarrthmia secondary to dyselectrolytemia in a case of sepsis in a peripheral setup has not been researched exclusively. This article aims to highlight such a case where use of transcutaneous pacing in an elderly male patient on mechanical ventilator and maximal inotropic support in sepsis and multiorgan dysfunction syndrome resulted in weaning off ventilatory and inotropic support and normalizing trend of organ failure.

KEYWORDS

Case Report

A 76 years old male, a known case of Cerebrovascular accident (left hemiparesis) 10 years back (not on any medication) presented to the casualty with altered sensorium manifesting as decreased oral intake and verbal output of 2 days duration. The patient became bed bound in a day and was brought by relatives to the emergency. No history of trauma, fever, vomiting, diarrhoea, cough, chest pain, dyspnoea on exertion or abnormal body movements.

On examination, the patient was drowsy but arousable , blood pressure was 90/60 mmHg, respiratory rate was 20/min and pulse rate was 48/min. General physical examination revealed pallor and dry oral cavity. Clubbing, cyanosis or lymphadenopathy was absent. From the aforementioned history, the neurological examination revealed GCS: E1V1M5, bilateral plantar reflex was mute. Both superficial and deep tendon reflexes were absent. No sensory and cranial nerve involvement, autonomic disturbance, or involvement of bladder was there. O n P/A exa mination , abdomen was soft, nontender but bowel sounds were decreased. CVS examination revealed bradycardia. Respiratory examination revealed bilateral basal crepititions.

Investigations at presentation revealed polymorphonuclearleucytosis (TLC: 20,200: P: 90%), azotemia (S Cr: 1.6mg/dl), RBS: 600mg/dL, Na/K: 140/5.8mmol/L, Arterial Blood gas analysis: Metabolic acidosis (pH : 7.20/HCO3: 9mmol/L/pCO2: 23.4mm Hg), ECG : sinus bradycardia

Management and semiology: In view of the low GCS, the patient was immediately shifted to the Intensive Care Unit and put on mechanical ventilator. He was started on broad spectrum intravenous antibiotics, insulin infusion and anti hyperkalemic measures. He continued to worsen requiring escalating doses of inotropic support. Despite correcting the hyperkalemia, and on inotropic support the patient continued to have worsening bradycardia with hemodynamic compromise. The patient was given a trial of atropine with no response. He was then put on transcutaneous pacing and a heart rate of 70 was achieved with 60mA current. He was maintained on 65 mA for the next 48 hours with repeated failed trials of terminating pacer rhythm. The patient became anuric with worsening renal parametres and developed iscahemic hepatitis. Bedside Echocardiography was suggestive of global hypokinesia and the IVC diameter was assessed to be normal. The patient received renal modified doses of antibiotics and central venous pressure guided fluids. On assessing the rhythm after 48 hours of continuous transcutaneous pacing, the patient developed normal sinus rhythm. He continued to have improving renal parametres and started producing urine on day 5 of admission. The inotropes were tapered off and the patient was extubated on Day 7 with normal sensorium and vital parametres. He was then referred to a higher centre for further evaluation and management by nephrologist and cardiologist.