HYPERKALEMIA – AN IMPORTANT DIFFERENTIAL DIAGNOSIS OF WIDE COMPLEX TACHYCARDIA – A CASE REPORT

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ABSTRACT

Wide complex tachycardia in Electrocardiogram (ECG) is a diagnostic challenge in patients presenting to the Emergency Department, with no prior work up. Differentiating a WCT resulting from hyperkalemia opposed to primary Ventricular Tachycardia (VT) is vital in the immediate management. Here we are presenting a case of a 65 year old lady whose ECG pattern on arrival when connected to the monitor showed a wide complex tachycardia pattern and initiated treated as ventricular tachycardia, later turned out to be due to hyperkalemia resulting from acute or chronic kidney disease and treatment modified. Immediately correcting the potassium was life saving for this patient. Immediate administration of intravenous (IV) calcium gluconate reverted the ECG back to sinus rhythm and patient was taken up for emergency dialysis. Hyperkalemia should be considered as an important differential diagnosis of wide complex tachycardia especially with a heart rate of less than 140 beats per minute.

Keywords: Wide Complex Tachycardia, Hyperkalemia, Ventricular Tachycardia

INTRODUCTION

Wide complex tachycardia's present diagnostic challenges for emergency physicians (Delbridge and Yealy, 1995). Common teaching in medicine is that when faced with a wide complex regular tachycardia, the treating physician should always treat the patient for VT (Ohlow *et al.*, 2005). Often other causes are easily missed unless carefully looked for and the management entirely changes. Here we present a case report where the patient presented with acute onset breathing difficulty and decreased urine output and when connected to the cardiac monitor showed a WCT pattern with a heart rate between 120 and 130 beats per minute. The patient was initially managed as stable ventricular tachycardia and later when VBG (Venous Blood Gases) report showed an increased potassium value of 8.9 mEq/L, the treatment plan was changed and modified to treat severe hyperkalemia.

CASES

A 65-year-old woman was referred from another hospital to the ED at night with complaints of acute dyspnea with decreased urine output. On presentation, she was in respiratory distress with a respiratory rate of 32/min, blood pressure of 90/60 mm Hg and an oxygen saturation of 80% at room air. Crepitation's were present in both her lung fields on auscultation, and the results of the abdominal examination were normal. The extremities were without cyanosis or edema. She was connected to oxygen immediately and blood samples were drawn for investigations. When the doctor-in-charge was about to take a brief history from the relatives after the initial stabilization, he had a look at the ECG monitor which showed wide QRS complex rhythm with a rate between 120 and 130 beats per min. The patient was conscious and pulse was palpable. Results of multiple 12-lead electrocardiograms revealed a wide ORS complex rhythm with a rate of 125 beats per minute and ORS duration of 165 msec (Figure 1), which led to a diagnosis of ventricular tachycardia. The patient was subsequently treated with intravenous (IV) amiodarone 150 mg over 10 minutes. Soon the VBG results arrived and it showed a potassium level of 8.9 mEq/L. Potassium corrections was started immediately with intravenous (IV) calcium gluconate and salbutamol nebulisation. The WCT pattern reverted to sinus rhythm soon after the administration of calcium gluconate (Figure 2). Intravenous (IV) dextrose with insulin was administered after that. Noradrenaline infusion was started for hypotension. The patient had also severe metabolic acidosis and

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bicarbonate therapy was started and the patient was immediately shifted to ICU for emergency dialysis. In view of hypotension, sustained low-efficiency dialysis was done. Her clinical history was reviewed which revealed intake of NSAID drugs for a prolonged period of time for her right leg pain, previous hospital admission for loss of appetite, decreased food intake and lately, breathing difficulty and nil urine output. Her serum creatinine levels were also increasing gradually. The patient had blood sent to the lab for multiple studies. The patient's complete blood count (CBC) revealed a white blood count of 20.3 thou/cu mm, hemoglobin of 11 g/dL, a hematocrit of 33.3%, and a platelet count of 450 thou/cu mm. Her metabolic panel revealed sodium of 137 mmol/L, potassium of 9 mmol/L, a urea of 182 mg/dL, and a creatinine of 5.24 mg/dL. Arterial blood gas revealed a pH of 7.062, pCO₂ of 24.4mmHg, and pO₂ of 158mmHg (maintaining saturation 100% at 6L of oxygen). International normalized ratio (INR) was 1.05. This led to a diagnosis of acute or chronic kidney disease with severe hyperkalemia, fluid overload and metabolic acidosis. After dialysis, she became symptomatically better. Her potassium level came down to 5.7 mmol/L and serum creatinine level came down to 4.3 mg/dL. Her vitals and urine output started improving and came to normal limits by the 4th day of admission.

DISCUSSION

Wide QRS complex tachycardia still presents a diagnostic challenge with a 12-lead ECG. ECG based differential diagnoses include Ventricular Tachycardia (VT) vs. Supraventricular Tachycardia (SVT) with aberrant conduction, pre-existing Bundle Branch Block, intraventricular conduction disturbances, and preexcitation. VT is the most important differential diagnosis because of its unfavorable prognosis. An accurate diagnosis with immediate treatment is usually required. A delayed diagnosis of VT or a misdiagnosis followed by inappropriate intravenous administration of drugs used for the treatment of SVT, such as verapamil and adenosine, can cause severe hemodynamic deterioration and may provoke ventricular fibrillation and cardiac arrest (Ahn, 2013). In most instances, less common causes like hyperkalemia can be easily missed. The WCTs resulting from hyperkalemia's are rarely faster than 140 beats per minute, usually have extremely wide and bizarre QRS morphologies, and do not demonstrate any rapid deflections within the ORS complex (Subramanian and Brady, 2008). Laboratory tests are the gold standard in diagnosing changes in the serum electrolyte concentration, delays may be experienced in obtaining results. Hence, in many cases, early diagnosis and empiric treatment of hyperkalemia is dependent on the physician's ability to recognize the electrocardiographical manifestations of hyperkalemia (Chew and Lim, 2005). If the patient is at risk for developing hyperkalemia, such as preexisting renal disease or at risk for developing acute kidney injury, such as recent NSAID or toxin intake, and if the ECG shows a WCT, calcium gluconate can be administered at the earliest and definitive dialysis can be lifesaving. Wide complex tachycardia is an indicator of life threatening hyperkalemia. As the hyperkalemia worsens and the potassium level approaches to reach 10 mmol/L, sinoatrial conduction no longer occurs and passive junctional pacemakers take over the electrical stimulation of the myocardium. If hyperkalemia continues unabated, the ORS complex continues to widen and eventually blends with the T wave, producing the classic sine-wave electrocardiogram. Once this occurs, ventricular fibrillation and asystole are imminent (Parham et al., 2006). A usual recommendation for treating a case of a stable WCT is to manage it as if it were VT, in accordance with the consideration of "first does no harm". Procainamide and lidocaine are probably the most effective agents for acute therapy in ventricular tachycardias. However, hyperkalemia potentiates the blocking effect of lidocaine on cardiac sodium channel and may precipitate asystole or ventricular fibrillation. Even, Amiodarone which is primarily a class III antiarrhythmic agent has got some sodium blocking property (Ohlow et al., 2005).

Conclusion

Hyperkalemia should be considered as an important differential diagnosis of wide complex tachycardia, especially if heart rate is less than 140 beats per minute. Diagnosing hyperkalemia by ECG alone is a diagnostic challenge. The diagnosis of hyperkalemia must be considered in any patient with clinical risk factors that would predispose them to its development. Early recognition and prompt treatment with calcium gluconate and hemodialysis can completely reverse this life threatening condition.

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Abbreviations ECG: Electrocardiogram ED: Emergency Department VBG: Venous Blood Gases ICU: Intensive Care Unit WCT: Wide Complex Tachycardia VT: Ventricular Tachycardia SVT: Supraventricular Tachycardia BBB: Bundle Branch Block NSAID: Non-steroidal Anti-inflammatory Drug

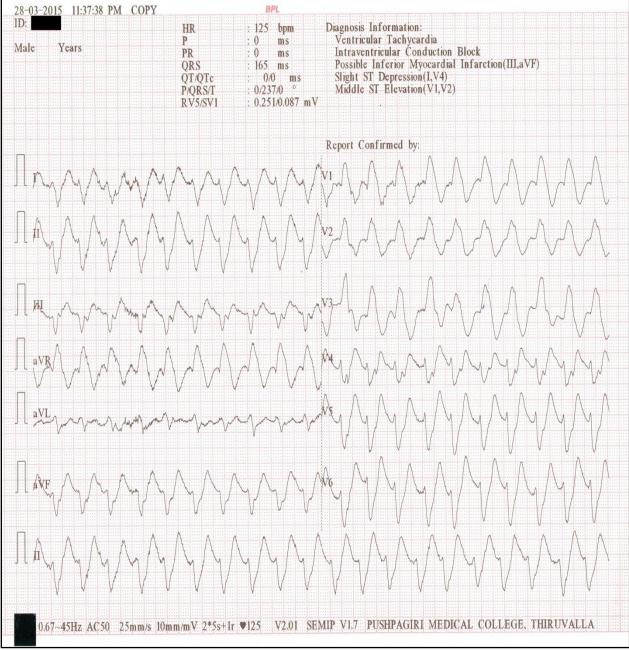


Figure 1: 12 lead ECG showing Wide Complex Tachycardia

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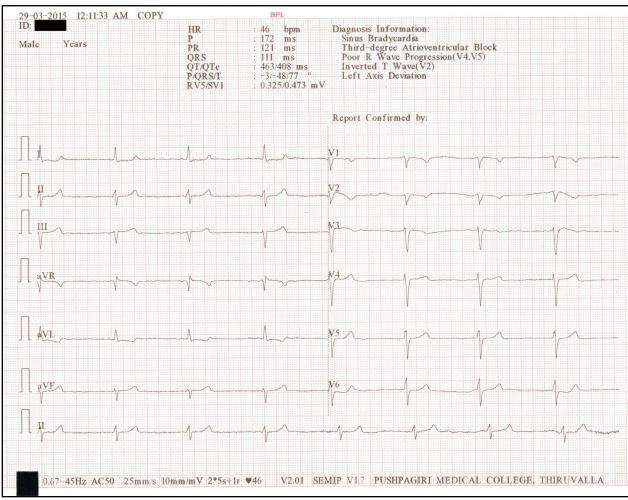


Figure 2: 12 lead ECG after giving IV calcium gluconate

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