74 year old nursing home patient with known history of schizoaffective disorder, hypertension and dementia was brought to the emergency room due to altered mental status and disorientation. Vitals on admission were stable and the initial 12-lead EKG showed an irregular rhythm with a 3 second pause and a narrow QRs complex with no obvious ST-segment elevation or depression. Later the patient had several long pauses (6-7 seconds) and became hypotensive. Sinoatrial (SA) exit block was noted on the monitor (Figure 1). Pertinent labs on admission showed a sodium level of 150 mmol/L, blood urea nitrogen of 26 mg/dL, creatinine of 1.6 mg/dL, leukocytosis (25.6 x 10⁹/L) and the lithium levels of 2.5 mEq/L (normal: 0.7-1.5 mEq/L).

In addition to initiating work up of sepsis, given the sinus pause, transcutaneous pacemaker pads were placed with good ventricular capture. The patient with a presumptive diagnosis of lithium toxicity was admitted to ICU for cardiac monitoring, intravenous hydration and serial lithium levels. She responded well to intravenous hydration to 2nd degree sinoatrial exit block. The sinoatrial conduction improved completely resuming normal sinus rhythm at 24 hrs when the lithium level was 1.6 mEq/L.

Lithium salts are widely used in treatment and prophylaxis as mood stabilizers like mania, bipolar affective disorder, recurrent depression and aggressive or self-mutilating behavior. Lithium is fully absorbed after oral intake approximately in 8 hrs with peak level occurs at 2 to 4 hrs [1]. The therapeutic range varies from 0.7 – 1.5 mEq/L. The plasma elimination half life of a single dose of lithium is between 12 – 27 hrs [2,3].

Animal experiments indicate that lithium depresses the intracellular potassium concentration as well as replaces intracellular calcium which results in various electrophysiological changes including a decrease of the depolarization rate and reduced electrical impulse propagation [4]. It also has been suggested regarding lithium decreasing the sensitivity of sinus node to sympathetic stimulation [5]. Further experiments on human and animal models involving molecular pharmacology and electrophysiology are necessary to understand the sinus node/perisinus tissue electrophysiological behavior caused by Lithium.

References