**LIDOCAINE TOXICITY MANIFESTING AS ACUTE STROKE: A RARE PRESENTATION**

**L. Hou**1, J. Xu1, E. Pagan1, K. Patel2, A. Hakeem2

1Department of Internal Medicine, Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ, USA

2Department of Cardiovascular Diseases and Hypertension, Rutgers Robert Wood Johnson Medical School, New Brunswick, NJ, USA

**Background:**Sudden cardiac death is most commonly caused by either ventricular tachycardia or ventricular fibrillation. Lidocaine has been used for the treatment of ventricular arrhythmias since 1950s. Lidocaine’s main target organs are the cardiovascular and the central nervous system. Of the two, CNS is more sensitive to the subtle electrophysiological balance. Neurological symptoms can range from dizziness to focal neurological deficit that can be misinterpreted as stroke. We present a case in which patient developed newly onset right-sided neurologic deficits on lidocaine infusion that resolved with lower infusion rate and normalized serum lidocaine level.

**Case Presentation:**A 76-year-old female with hypertension who presented with dyspnea found to have NSTEMI. She was found to have 99% stenosis at the left main distal region at the trifurcation of the LAD, ramus and left circumflex as well as a 100% mid LAD occlusion. She underwent CABG and developed recurrent polymorphic ventricular tachycardia and ventricular fibrillation post-operatively. She was given a lidocaine bolus and started on infusion. She was then thought to have a stroke as she acutely developed right-sided deficits, aphasia, and confusion. Computerized tomographic scan (CT) of her head did not show an acute bleed. Her lidocaine level was found to have 8.2 mcg/ml (1.5-5.0 mcg/ml). Subsequently, lidocaine was weaned off and the patient’s neurologic deficits resolved.

**Discussion***:* The most common side effects of lidocaine include neurological and cardiovascular deficits. Risk factors for lidocaine toxicity include congestive heart failure, low body weight, increased age, and previous myocardial infarctions. Intrathecal administration of lidocaine is frequently utilized in spinal anesthesia and neurotoxicity is frequently seen in clinical practice and reported. Neurological manifestation of lidocaine toxicity as a result of intravenous administered lidocaine is rarely reported and neurotoxicity mimicking acute stroke is exceedingly rare. We believe it is crucial to have a high degree of clinical suspicion for lidocaine toxicity when patients on lidocaine infusion develop neurological deficits.