Austin Cardiology

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Austin Publishing Group

Case Report

Lidocaine Toxicity in a Patient with Liver Cirrhosis Underwent Transcatheter Aortic Valve Implantation

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Received: February 03, 2021; **Accepted:** March 05, 2021; **Published:** March 12, 2021

Abstract

Background: Lidocaine has been commonly used in many clinical settings. Nonetheless, systemic toxicity can be life-threatening and careful attention to dosing, especially among patients with liver dysfunction is important to minimize the risk of toxicity.

Objective: The diagnosis of lidocaine toxicity is usually clinical, while rare, but may prove fatal.

Methods: Here we discuss 60 years-old man with advanced liver cirrhosis, developed lidocaine-induced cardiovascular and neurotoxicity.

Results: Our case study demonstrates a successful treatment of cardiovascular and neurotoxicity with intravenous lipid emulsion in the context of systemic lidocaine toxicity in a liver cirrhosis patient who received lidocaine as a local anesthetic.

Conclusions: A high index of suspicion should be maintained for severe toxicity even after subcutaneous administration and prompt intralipid administration may prove lifesaving.

Keywords: Aortic stenosis; Transcatheter aortic valve implantation; Lidocaine toxicity; Intravenous lipid emulsion

Introduction

Lidocaine is a widely used local anesthetic and a class Ib antiarrhythmic agent, available since 1948, and stabilizes the neuronal membrane by binding to and inhibiting voltage-gated sodium channels, thereby inhibiting the ionic fluxes required for the initiation and conduction of impulses and effecting local anesthesia [1]. The dose and site of injection are risk factors for toxicity. It has a 90% hepatic metabolism forming monoethylglycinexylidide and glycinexylidide, which may accumulate and cause central nervous system and cardiovascular toxicity, while rare, but may prove fatal [1]. Patients with impaired liver function are at a greater risk for toxicity. There are a growing number of case reports regarding lidocaine toxicity [2-5]. However, the potential for severe toxicity after subcutaneous, rather than intravenous administration is not well recognized. We report a case of lidocaine-induced cardiovascular and neurotoxicity in a patient with advanced liver cirrhosis admitted for elective Transcatheter Aortic Valve Implantation (TAVI).

Case Presentation

A 60-year-old man with severe symptomatic aortic stenosis was admitted for TAVI .His medical history was significant for alcoholic liver cirrhosis (Child C) with portal hypertension and gastric varices. His left ventricular systolic function was normal. His coronary arteries were normal. Regular medications included aspirin 100 mg per day, propranolol 10 mg twice daily, spironolactone 25mg, and metformin 850 mg daily. On admission, the patient was in no apparent distress. His blood pressure was 145\65 mmHg, heart rate 80 beats per minute and regular, respiratory rate 18 per minute and temperature 36.7°C. His physical examination was notable for 3/6 systolic murmur at the right upper sternal border with diminished S2, ascites and mild leg edema.

The TAVI procedure was started with bilateral groin local anesthesia, using lidocaine 2% in a total dose of 600 mg. Shortly thereafter, the patient became confused and lethargic and his blood pressure dropped to 70/40 mmHg. A neurologic examination showed no motor deficits. The procedure was interrupted and an urgent brain CT angiography revealed no evidence of bleeding or stroke. The patient became progressively hypotensive and required increasing doses of noradrenaline. Upon admission to the intensive care unit, the patient was still hemodynamically unstable and lethargic. Mechanical ventilation was initiated. The ECG revealed no specific changes. An echocardiogram showed a hyperdynamic left ventricular function and his known severe aortic stenosis. The laboratory evaluation showed no change in the hemoglobin level and a mild combined metabolic and respiratory acidosis. An ascites puncture revealed no evidence of spontaneous bacterial peritonitis.

Lidocaine toxicity was suspected and the patient given intravenous lipid infusion 20% in addition to supportive care with saline and vasopressors. A few hours later, the patient stabilized, was successfully extubated and gradually weaned from noradrenaline. After 4 days the patient was moved to internal medicine for further treatment of his liver disease and rehabilitation. The TAVI procedure was further postponed until stabilization of his encephalopathy.

Discussion

Lidocaine toxicity has been suggested as a contributing factor

Citation: Dogosh AA, El Nasasra A, Zahger D and Cafri C. Lidocaine Toxicity in a Patient with Liver Cirrhosis Underwent Transcatheter Aortic Valve Implantation. Austin Cardiol. 2021; 6(1): 1026.

to adverse outcomes, although its role has not been demonstrated definitively [6]. Lidocaine toxicity is dosage-dependent and directly relative to its plasma concentration and is alterable by many patient factors (e.g., decreased congestive heart, liver failure, and co-medications) [7]. Because lidocaine is metabolized by the cytochrome P450 system, the potential for drug interactions is high. As lidocaine concentration increases to toxic levels, initial complaints of a metallic taste, restlessness, circumoral and tongue numbness, lightheadedness, and tinnitus occur.

The recommended maximum dose for subcutaneous infiltration of lidocaine without epinephrine is 4.5 milligrams per kilogram (mg/kg) and for lidocaine with epinephrine is 7 mg/kg [8]. Addition of vasoconstrictors such as epinephrine can dramatically slow the absorption from the site of injection, improving safety and prolonging the anesthesia, which is why higher doses of some agents are possible with a vasoconstrictor additive. Our patient's weight was 80 kg and he received a lidocaine dose of 600 mg subcutaneous in the groin region, equivalent to 7.5 mg/kg. This dosage exceeds the previously described toxic doses, and therefore, warrants the development of subsequent local anesthetic systemic toxicity. In patients with liver cirrhosis or other chronic liver disease, the dose should be reduced.

For the treatment of lidocaine toxicity the American Society of Regional Anesthesia and Pain Medicine (ASRA) recommends an initial bolus dose of 1.5 mL/kg of 20% lipid emulsion, followed by a continuous infusion of 0.25 mL/kg/minute. If cardiac stability is not restored, the infusion rate can be doubled and it is recommended to continue the infusion until cardiac stability has been restored for at least 10 minutes [9]. The mechanism of action of intravenous lipid infusion in the management of poisoning is not completely understood. Nonetheless, the most widely accepted theory by which intravenous lipids reverse the cardiac toxicity of lidocaine is by building a "lipid sink" in which the infused lipids bind the lipophilic lidocaine, before it is slowly released back into circulation to be metabolized [10]. Furthermore, the lipids may also serve as a cardiotonic agent by increasing cardiac output without an increase in heart rate [11].

In our case, the patient received a high dose of lidocaine in the presence of advanced liver cirrhosis, leading to shock with cardiovascular collapse and neurologic symptoms. After the rapid lipid infusion this symptoms resolved within hours.

Conclusion

Systemic toxicity of lidocaine can be life-threatening and the use of intravenous lipid infusion as an antidote is not well recognized. Careful attention to dosing, especially among patients with liver dysfunction is important to minimize the risk of toxicity. Our aim is to ensure that interventional cardiologists are able to diagnose and treat lidocaine toxicity. A high index of suspicion should be maintained for severe toxicity even after subcutaneous administration and intravenous lipid infusion is crucial to prevent mortality.

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