Rev Bras Anestesiol. 2018;xxx(xx):xxx–xxx

Sociedade Brasileira de Anestesiologia

REVISTA BRASILEIRA DE ANESTESIOLOGIA

Publicação Oficial da Sociedade Brasileira de Anestesiologia
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CLINICAL INFORMATION

Fulminant hepatic failure after simultaneous kidney-pancreas transplantation: a case report

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Received 21 February 2017; accepted 20 January 2018

KEYWORDS
Liver failure;
Kidney-pancreas transplant;
Volatile agents;
Liver transplant;
Drug reaction;
Isoflurane toxicity;
Adverse drug reaction;
Inhalational anesthesia

Abstract We describe an unusual case of hyperacute hepatic failure following general anesthesia in a patient receiving a simultaneous kidney-pancreas transplant. Despite an aggressive evaluation of structural, immunological, viral, and toxicological causes, a definitive cause could not be elucidated. The patient required a liver transplant and suffered a protracted hospital course. We discuss the potential causes of fulminant hepatic failure and the perioperative anesthesia management of her subsequent liver transplantation.

PALAVRAS-CHAVE
Insuficiência hepática;
Transplante de rim-pâncreas;
Agentes voláteis;

Insuficiência hepática fulminante após transplante simultâneo de rim-pâncreas: um relato de caso

Resumo Descrevemos um caso incomum de insuficiência hepática hiperaguda após a anestesia geral em uma paciente que recebeu um transplante simultâneo de rim-pâncreas. Apesar de uma avaliação agressiva das causas estruturais, imunológicas, virais e toxicológicas, uma causa definitiva não pôde ser identificada. A paciente precisou de um transplante de fígado

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https://doi.org/10.1016/j.bjane.2018.01.017

Please cite this article in press as: Moss JL, et al. Fulminant hepatic failure after simultaneous kidney-pancreas transplantation: a case report. Rev Bras Anestesiol. 2018. https://doi.org/10.1016/j.bjane.2018.01.017

Transplante de fígado;
Reação medicamentosa;
Toxicidade por isoflurano;
Reação medicamentosa adversa;
Anestesia inalatória

que resultou em prolongamento da internação hospitalar. Discutimos as potenciais causas da insuficiência hepática fulminante e o manejo da anestesia no período perioperatório de seu subsequente transplante de fígado.

Introduction

Hepatotoxicity has long been recognized as an adverse effect of halogenated volatile anesthetic metabolism. The development of newer agents that undergo less hepatic metabolism has greatly decreased its incidence in modern anesthesia. Classically described as the onset of postoperative fever, myalgias, rash, and jaundice 48–72 h following exposure, volatile hepatotoxicity is associated with significant morbidity and mortality. To the best of our knowledge, we describe the first case of hyperacute liver failure following isoflurane exposure. Our patient developed overt liver failure within 24 h of exposure, and required a liver transplant on postoperative day 2. We describe our workup and review volatile hepatotoxicity. All patient information has been adequately deidentified in accordance with Mayo Clinic IRB and institutional policy, as well as, the Health Insurance Portability and Accountability Act.

Case report

A 60-year-old Asian female with end stage kidney disease secondary to diabetes and hypertension presented for a simultaneous kidney-pancreas transplant. Her past medical history was notable for chronic anemia, latent TB (treated with isoniazid 2 years prior), hepatitis B core antibody positive without a detectable viral load, mild pulmonary hypertension, ocular stroke, and coronary artery disease status-post drug eluding stent placement. She had a left upper arm arterio-venous fistula at an outside facility, presumably with a general anesthetic, however her medical records were not available for review. Her kidney-pancreas transplant proceeded uneventfully after induction with propofol (120 mg), fentanyl (250 mcg), and midazolam (2 mg) followed by isoflurane, hydromorphone (1 mg), and cis-atracurium (36 mg over 436 min) for maintenance of anesthesia. Expiratory isoflurane concentration was maintained between 1.0% and 1.5% throughout the case. The intraoperative anti-rejection regiment consisted of a thymoglobulin infusion and 500 mg of methylprednisolone. The patient began producing urine and the blood glucose decreased following reperfusion of the kidney and pancreas respectively. There were no prolonged hypotensive episodes. One unit of packed red blood cells was transfused intraoperatively for a hemoglobin of 7.3 $g.dL^{-1}$. The total anesthetic time was 436 min and the surgical time was 339 min. The post-anesthesia care unit course was uneventful and the patient was transferred to the floor.

The following morning, the patient was found to be very lethargic, but arousable. Laboratory results revealed elevated liver function tests (LFTs) with aspartate aminotransferase (AST) 3904 $IU.L^{-1}$ and alanine aminotransferase (ALT) 2596 $IU.L^{-1}$, up from 23 $IU.L^{-1}$ and 31 $IU.L^{-1}$ preoperatively. She was transferred to Intensive Care Unit (ICU) with a diagnosis of acute liver failure and intubated secondary to rapidly worsening encephalopathy. Urine output progressively decreased and continuous renal replacement therapy was initiated. Additionally, a metabolic acidosis developed, requiring treatment with a bicarbonate infusion. On Post-Operative Day (POD) 2, LFTs peaked, at AST 58,960 and ALT 6684, and the International Normalization Ratio (INR) also increased to 5.8 (baseline 1.0). The patient was listed as Status 1 for liver transplantation, indicating severe liver disease with a life expectancy of hours to days. Concurrently, the patient received an aggressive work-up to determine the cause of liver failure. Immunological, viral, structural, and toxicology studies were all nondiagnostic. An intense review of the patient's medications was performed to identify medication with hepatotoxic potential. On POD 2, the patient underwent a liver transplant. With the possibility of isoflurane being the cause of the patient's idiosyncratic reaction, the anesthesia team decided to forego inhalational anesthesia and opted for a total intravenous anesthesia approach. Infusions of propofol (50–100 $mcg.kg^{-1}.min^{-1}$) and midazolam (0.25–0.5 $mg.h^{-1}$) were employed, with titration to maintain the bispectral index monitor between 10 and 40. Additionally, cis-atracurium and fentanyl boluses were administered as needed. Continuous renal replacement therapy was maintained in the operating room. The orthotopic liver transplantation was performed using a piggy-back technique and was uneventful with the exception of a mild reperfusion syndrome following recirculation that required the brief addition of a vasopressin drip.

On visual inspection, the liver appeared grossly necrotic. Pathological examination of the explant revealed submassive necrosis, characterized by large areas of confluent multilobular necrosis, as well as, centrilobular and bridging necrosis and a background of hepatocellular hemosiderosis. Viral inclusions were not seen.

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