Annals of Clinical Case Reports

Case Report
Published: 03 Sep, 2017

A Case of Toxic Hepatitis and Acute Liver Failure Induced by Ingestion of Raw Sansevieria

Joo Hyun Sohn[1,3], Ji Yeoun Kim[1], Tae Yeob Kim[1], Jae Yoon Jeong[1], Sun Min Kim[1] and Ju Yeon Pyo[2]*

[1]Department of Internal Medicine, Hanyang University Guri Hospital, Hanyang University, Korea

[2]Department of Pathology, Hanyang University Guri Hospital, Hanyang University, Korea

[3]Department of Gastroenterology, Hanyang University College of Medicine, Korea

Abstract

Herbal medicines are frequently used in the oriental region to treat a variety of disease and symptoms. Nearly 50% of patients with toxic hepatitis are related to these herbs and health supplements in Korea. We report, for the first time, a case of liver failure due to toxic hepatitis induced by Sansevieria ingestion. The patient took raw Sansevieria for a month and thereafter developed symptoms such as jaundice, nausea, anorexia and general weakness. The diagnosis was made after a thorough history taking, laboratory exams, imaging studies such as ultrasonography, liver biopsy, and the exclusion of other causes of hepatitis. We administered supportive treatment for acute toxichepatitis but the patient's symptoms and liver functions worsened and finally expired in 2 months. Since toxic hepatitis is frequently induced by taking herbs and health supplements and occasionally fatal, it is crucial to make early diagnosis and immediately stop each drug which is suspected to cause liver injury. This case is noteworthy because Sansevieria assumed to have hepatoprotective effect but is the first case reported for its fatal hepatotoxicity.

Keywords: Liver failure Acute; Drug-induced liver injury; Sansevieria; Toxic hepatitis

Introduction

Incidence of drug-induced liver disease (DILD) is from 2.3 up to 19.1 per 100,000 inhabitants per year, especially higher with older age and it is one of the leading causes of acute liver failure [1]. As concerns for health are growing, the use of herbs and health supplements are increasing worldwide and especially in Korea as an easier way to care for health [2]. In Korea, over 50% of patients with acute hepatitis are caused by drugs or toxins and 42-74.5%of the causes of toxic hepatitis were occupied by oriental medicines and other herbs [3-5]. Furthermore, patients with acute liver failure induced by idiosyncratic drug reactions have poor prognosis, with 60% to 80% mortality without a liver transplantation [3,4,6]. In most cases, there are no effective drugs or antidotes other than stopping the causative agent and providing supportive care [7,8].

Sansevieria is assumed to have some benefits on the liver and we could not find any reports about hepatictoxicity of raw Sansevieria [9]. For the first time, we report a case of toxic hepatitis caused by consuming Sansevieria for a month in a 79 years old female, which ultimately led to progressive liver failure and death in 2 months.

Case Presentation

In August 2012, a 79-year-old female with no other underlying chronic liver disease visited emergency room with newly developed jaundice and was admitted. About six months prior to her visit, she underwent percutaneous transhepatic biliary drainage and percutaneous transhepatic gallbladder drainage with balloon dilatation for calculous cholecytitis and cholangitis. After the procedure and treatment of antibiotics, her cholecystitis and cholangitis were improved and then the patient and her family refused operation. Thereafter ursodeoxycholic acid (UDCA) was prescribed for several months with no recurrence of clinical symptom and signs of cholecystitis or cholangitis. Several weeks before this admission, she arbitrarily stopped taking UDCA and instead she insisted on taking raw Sansevieria for a month, which was believed, without proven efficacy, to have hepatoprotective effect. She denied taking other medicines or herbs and alcohol drinking. She also denied any abrupt or colic abdominal pain and febrile illness during the past several months.

OPEN ACCESS

Correspondence:
Joo Hyun Sohn, Department of Internal Medicine, Hanyang University Guri Hospital, 153 Gyeongchun-ro, Guri 471-701, Korea, Telephone: 82315602225; Fax: 82315552998; E-mail: sonjh@hanyang.ac.kr

Received Date: *25 May 2017*
Accepted Date: *30 Aug 2017*
Published Date: *03 Sep 2017*

Citation:
Sohn JH, Kim JY, Kim TY, Jeong JY, Kim SM, Pyo JY. A Case of Toxic Hepatitis and Acute Liver Failure Induced by Ingestion of Raw Sansevieria. Ann Clin Case Rep. 2017; 2: 1429.

ISSN: 2474-1655

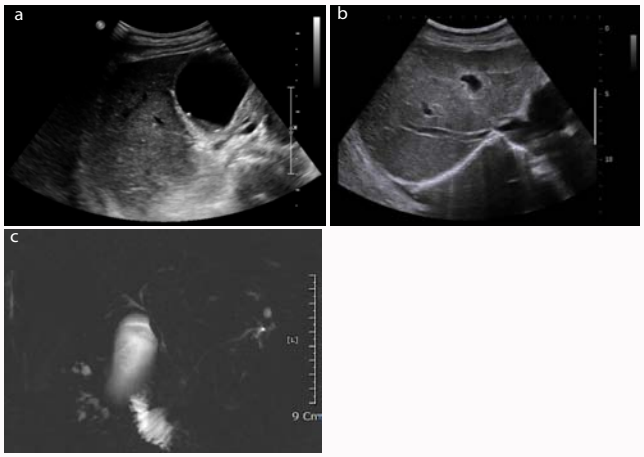

Figure 1: Imaging study of the patient. (A) Ultrasonographic image showsbile sluge and small gallstones in gallbladder without gallbladder wall thickening. (B)It also shows heterogeneously increased hepatic parenchymal echogenicity and no signs of chronic liver disease. (C) Magnetic resonance cholangiogram shows no obstructive lesion or duct dilatation in common bile duct.

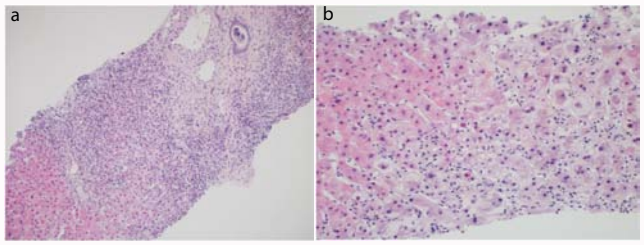

Figure 2: Microscopic findings of liver biopsy specimen. (A) Histologic examination shows extensive piecemeal necrosis and severe lobular and portal inflammation but no portal fibrosis (H&E, x100). (B) There is severe hepatocyte ballooning degeneration with apoptosis and cholestasis (H&E, x200).

On physical examination, she was icteric and ill looking. Initial vital signs at emergency room were: blood pressure of 120/70 mmHg, pulse of 72 beats per minute, respiratory rate of 20 breaths per minute, and temperature of 36.8°C. Mild right upper quadrant discomfort with tenderness was noted.

The laboratory tests were as follows: hemoglobin 11.8 g/dL, WBC 3,400/mm^3 (segment 72%, lymphocytes 22%, and eosinophil 1%), platelet 140,000/mm^3, serum AST/ALT ratio 9.9 (797/79 U/L), r-GTP 295 U/L, ALP 140 U/L, bilirubin (total/direct) 14.2/8.8 mg/dL, LDH/CPK 726/39 (U/L). Prothrombin time INR was 1.38. Serologic tests for viruses causing acute hepatitis such as HAV Ab IgM, HBs Ag, anti-HBc IgM, HCV Ab IgG, HCV RNA PCR, HEV Ab IgM, HEV Ab IgG, CMV Ab IgM, CMV Ab IgG, HSV Ab IgM, HSV Ab IgG, EBV Ab IgM, and EBV Ab IgG were all negative. Immunological profiles for anti-smooth muscle antibody, anti-nuclear antibody, and anti-mitochondria antibody were also all negative. There were no bacterial growth in blood and urine.

Ultrasonography examination showed bile sludge and small gallstones in gallbladder without gallbladder wall thickening, heterogeneously increased hepatic parenchymal echogenicity and no signs of chronic liver disease (Figure 1A and B). In abdominal contrast enhanced computed tomography showed no stones in common bile duct but pneumobilia due to previous balloon dilatation and did not show any enlarged lymph nodes or hepatosplenomegaly. Magnetic resonance cholangiogram also showed stones in the gallbladder, but did not give any evidence for cholecystitis and any obstructive lesion such as bile sludge and stones in the common bile duct (Figure 1C).

On her 17th day after admission, transjugular liver biopsy was performed to exclude other causes of hepatitis. Histological examination showed severe hepatocyte degeneration with piecemeal necrosis and severe cholestasis. There were severe lobular and portal inflammation but no portal fibrosis was found (Figure 2). This result is compatible with severe acute hepatitis with no underlying chronic liver disease.

Based on RUCAM score of 6 with cholestatic pattern, [10] she was diagnosed with toxic hepatitis and supportive treatment such as UDCA and steroid for cholestasis were given in parallel with sufficient calories and nutritional supply. Despite of 2 months with supportive treatment, her laboratory examinations did not show improvement (Figure 3). Her anorexia, general weakness and jaundice worsened and later she developed mental change caused by hepatic encephalopathy. Finally, she expired due to progression of liver failure.

Discussion

DILD is an increasing health problem, especially in the oriental region and is one of the leading causes of acute liver failure in paralleling with growing concerns for health and increasing use of herbs and health supplements [2]. Recently it was reported that over 50% of patients with acute hepatitis are caused by drugs and 42-74.5% were caused by oriental medicines and other herbs in Korea [3-5].

As we know, this case is the first to report the hepatotoxicity of raw Sansevieria, finally leading to death. There are few reports showing improvement of elevated liver enzyme in rats by Sansevieria [9]. However, clinical effectiveness is not sufficiently defined with large and randomized studies. Also we could not find any reports

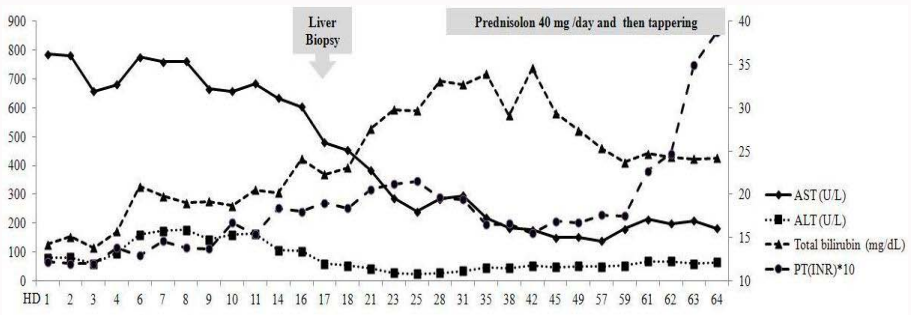

Figure 3: Clinical course of the patient.
HD: Hospital Day; PT: Prothrombin Time; INR: International Normalized Ratio

about hepatic toxicity of raw Sansevieria.

Diagnosis of DILD is very difficult because of the absence of definitive diagnostic tests and rare incidence. Histopathology is not useful to find the etiology but only shows the type and degree of hepatic injury. Therefore, the key to diagnose is to assess the temporal relationship between drug initiation and development of an abnormal liver test and to exclude other causes of liver diseases [8]. That is why detailed history taking and physical exams, laboratory findings and histopathology are needed to diagnose. In this case, we excluded other common causes of liver disease such as various hepatitis viruses, rare non-hepatitis viruses, and autoimmune liver diseases. She was hemodynamically stable before and during the admission, so ischemic reperfusion or anoxic liver injury could be excluded. She had known gallbladder stones but she denied any abdominal pain and there were no signs of cholecystitis and cholangitis by ultrasonography, computed tomography and magnetic resonance cholangiography. So the possibility of biliary stone passage was low and furthermore it could be excluded by her liver histopathology and clinical course.

As aforementioned, biochemical index for diagnosing toxic hepatitis is not established and it is not ethically allowed to artificially induce or put injured liver cell into the human body. So evaluation is usually based on the causality assessment method, especially the RUCAM scale [10]. In this case, the patient showed cholestatic-type hepatic injury with R value <2. Based on the RUCAM score, our case was classified 'probable' for a total of 6 points in combination of following factors; time to (+2), course (0), risk factors (+2), concomitant drugs (0), exclusion of other case of liver injury (+2), previous information on hepatotoxicity (0), and response to re administration (0).

In treating DILD, most important therapy is to withdraw the suspected agent. There are no other beneficial therapies reported except for the use of N-acetylcysteine in acetaminophen toxicity [11]. Corticosteroid may be used in DILD cases with evident hypersensitivity, but there are no proven benefits [12]. Using UDCA for cholestatic liver injury is also controversial [8]. In this case we used every method known but the results were fatal.

It is notable that according to a recent article, her fatal destiny might be assumed by her initial lab results showing high AST/ALT ratio (9.9) and hyperbilirubinemia (14.2 mg/dL).In that report, it was identified AST but not ALT to be an independent predictor of bad outcome and it was also pointed out that the AST and bilirubin levels are the most important predictors of death or liver transplantation in severe DILD [13]. It is also in parallel with another result in patients with DILD showing higher aminotransferase levels, especially AST in patients who progressed to fulminant hepatic failure compared with those who did not [14]. Furthermore, it is also in line with an observation that showed a higher AST/ALT ratio in fatal cases than in survivors with severe acute viral hepatitis [15]. In addition, we could fully exclude other causes of high ratio of AST/ALT such as alcoholic hepatitis, Wilson disease, cirrhosis, acute fatty liver of pregnancy, rhabdomyolysis, etc.

With increasing interests in health and usage of herbal medicine, also the risks of developing DILI also is increasing. But there are few data regarding the incidence and clinical manifestation about DILI induced by herbal medicine. We are reporting for the first time with a case of Sansevieria induced toxic hepatitis, which was alleged to be hepatoprotective, to arouse that toxic hepatitis can occur by Sansevieria. Although most of the patients do not regard herbal medicines or dietary supplements as medicine and do regard those as not toxic material, physicians must keep in mind that any health supplements such as Sansevieria can cause hepatotoxicity, especially with high suspicion.

In conclusion, we report a case of toxic hepatitis caused by consuming Sansevieria for a month in a 79 years old female which ultimately led to progressive liver failure and death in 2 months.

References

1. Bjornsson ES, Bergmann OM, Bjornsson HK, Kvaran RB, Olafsson S. Incidence, presentation, and outcomes in patients with drug-induced liver injury in the general population of Iceland. Gastroenterology. 2013; 144: 1419-1425.
2. Park HA. Top 10 dietary supplements of korean adults from the 4th Korea national health and nutrition examination survey. Korean J Fam Med. 2011; 32: 263-266.
3. Kang SH, Kim JI, Jeong KH, Ko KH, Ko PG, Hwang SW, et al. [Clinical characteristics of 159 cases of acute toxic hepatitis]. Korean J Hepatol. 2008; 14: 483-492.
4. Kim JB, Sohn JH, Lee HL, Kim JP, Han DS, Hahm JS, et al. Clinical characteristics of acute toxic liver injury. Korean J Hepatol. 2004; 10: 125-134.
5. Suk KT, Kim DJ, Kim CH, Park SH, Yoon JH, Kim YS, et al. A prospective nationwide study of drug-induced liver injury in Korea. Am J Gastroenterol. 2012; 107: 1380-1387.
6. Hoofnagle JH, Carithers RL Jr, Shapiro C, Ascher N. Fulminant hepatic failure: summary of a workshop. Hepatology. 1995; 21: 240-252.
7. Navarro VJ, Senior JR. Drug-related hepatotoxicity. N Engl J Med. 2006; 354: 731-739.
8. Verma S, Kaplowitz N. Diagnosis, management and prevention of drug-induced liver injury. Gut. 2009; 58: 1555-1564.
9. Chigozie IJ, Chidinma IC. Positive moderation of the hematology, plasma biochemistry and ocular indices of oxidative stress in alloxan-induced diabetic rats, by an aqueous extract of the leaves of Sansevieria liberica Gerome and Labroy. Asian Pac J Trop Med. 2013; 6: 27-36.
10. Danan G, Benichou C. Causality assessment of adverse reactions to drugs--I. A novel method based on the conclusions of international consensus meetings: application to drug-induced liver injuries. J Clin Epidemiol. 1993; 46: 1323-1330.
11. Bunchorntavakul C, Reddy KR. Acetaminophen-related hepatotoxicity. Clin Liver Dis. 2013; 17: 587-607.
12. Suk KT, Kim DJ. Drug-induced liver injury: present and future. Clin Mol Hepatol 2012; 18: 249-257.
13. Bjornsson E, Olsson R. Outcome and prognostic markers in severe drug-induced liver disease. Hepatology. 2005; 42: 481-489.
14. Ohmori S, Shiraki K, Inoue H, Okano H, Yamanaka T, Deguchi M, et al. Clinical characteristics and prognostic indicators of drug-induced fulminant hepatic failure. Hepatogastroenterology. 2003; 50: 1531-1534.
15. Gitlin N. The serum glutamic oxaloacetic transaminase/serum glutamic pyruvic transaminase ratio as a prognostic index in severe acute viral hepatitis. Am J Gastroenterol. 1982; 77: 2-4.