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Case Report

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Coexistence of Brucellosis and Mushrooms Intoxication in a Patient Presenting with Toxic Hepatitis

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ABSTRACT

Introduction

Elevated liver enzymes are a laboratory finding that we frequently encounter, especially in patients who apply to the internal medicine outpatient clinic with complaints of nausea, vomiting and abdominal pain. When the etiological reasons are examined in patients living in rural areas in our country, it is understood that liver enzyme elevations due to mushroom eating and consumption of raw milk products are seen more.

Coexistence of rare brucella and mushroom poisoning will be explained in our case.

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Introduction

Elevated liver enzymes are a common concern at internal medical clinics. Paracetamol intoxication is one of the most frequent causes of enzyme elevation. In our country, especially in rural areas, mushrooms intoxication is also an important etiological factor [1]. Brucellosis is among the most common causes of zoonosis in the world. In rare cases, it may result in hepatic enzymes elevated [2]. We present a rare case who presented with a history of eating mushrooms and accompanied by brucella.

Case Presentation

A 50-year-old female patient was admitted to the hospital with nausea, fatigue, anorexia, headache after eating mushrooms. In addition, we learned that family members applied to the hospital with the suspicion of mushroom poisoning. The patient was conscious and vital functions were stable. On physical examination, abdominal palpation was normal. Complete blood count (CBC) revealed neutropenia and thrombocytopenia. Peripheral smear was compatible with CBC. Liver function tests were found to be high (other parameters are summarized in Table). The patient was evaluated with hepatobiliary, Portal Doppler Ultrasonography and contrast-enhanced abdominal tomography. In her imaging, the liver size was increased, diffuse density loss secondary to steatosis, and a millimetric calcific focus in the subcapsular region at the level of liver segment 8, was observed (Figure). It was observed that the increase in liver enzymes continued and the patient has vomiting. Oral intake was stopped and then intravenous hydration was started. It was learned that the patient lived in the village and was engaged in ovine breeding. It was also determined that take part of birth small cattle and consumed cheese made from raw milk. It was revealed that she had severe back, joint and headache complaints about a week ago.

A sero-agglutination test Rose Bengal was reported as weakly positive at a titer of 1:160. Rifampicin and doxycycline were administered. A significant decrease in liver enzymes was observed on the first day after treatment. In the follow-ups, liver enzyme levels regressed. When these results were assessed together, it was thought that liver enzyme elevation developed secondary to Brucella infection.

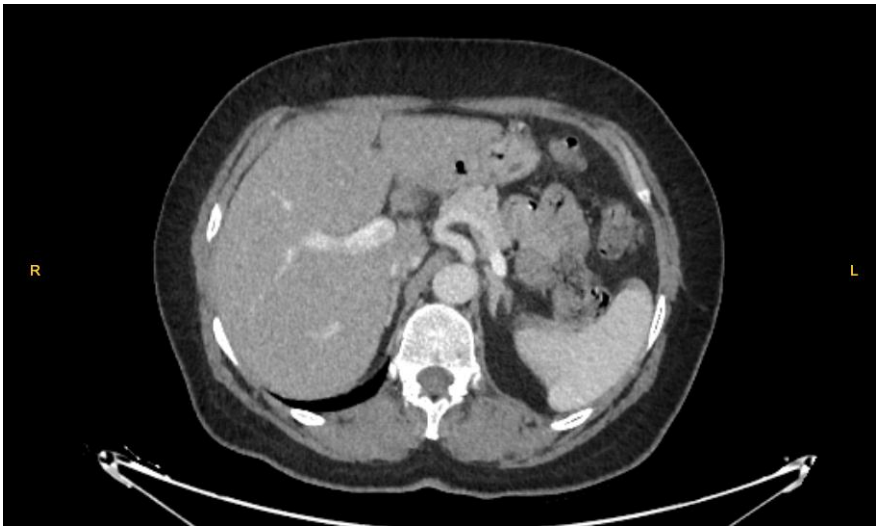

Figure: Contrast-enhanced abdominal tomography

Laboratories	Day 14	Day 10	Day 9	Day 7	Day 6	Day 4	Day 2	At Presentation	References
Glucose	75	103	93	112	86	76	79	108	70-99 mg/dl
Urea	36	38		29	12	19	23	32	19-49 mg/dl
Creatinine	0.64	0.67	0.54	0.49	0.34	0.40	0.48	0.59	0.5–1.1 mg/dl
Albumin	40.59	37.71	37.40	37.48	38.38	37.95	37.15	41.75	32-48 gr/L
ALP	107	123	158	178	180	176	186	235	53-141 U/L
ALT	66	137	349	412	458	481	490	346	<50 U/L
AST	24	45	310	456	560	791	928	748	<35 U/L
GGT	49	61	94	113	121	165	98	58	<38 U/L
LDH	220	232	293	372	362	379	525	607	120-246 U/L
CK	46		47		45		169	766	33-211 U/L
Total Bilirubine	0.50	0.40	1.015	1.325	1.80	1.50	0.80	0.493	0.3-1.2 mg/dL
Direct Bilirubine	0.26	0.22	0.657	0.649	0.90	0.67	0.30	0.180	<0.3 mg/dL
Sodium	145	139	139.8	138.1	136	136	139	139.0	132-146 mEq/L
Potassium	4.80	4.03	3.5	3.6	3.38	2.96	3.41	3.7	3.5-5.5 mEq/L
Calcium	8.67	8.71	8.29	8.34	8.70	8.49	8.80	8.47	8.7-10.4 mg/dL
Clorine		103	106	106	101	103	103	104	99-109 mEq/L
Hemoglobin	11.9	12.3	12.5	13.4	13.3		14.1	13.6	12-15.6 g/dL
Hematocrit	36.4	37.4	38	40.7	40	40	43.1	44.2	35-45 %
Leukocytes	5.82	5.34	4.70	4.98	4.95		3.75	3.08	3.9-10.2 x10^9/L
Neutrophils	2.57	2.09	2.04	2.61	2.61		1.59	1.03	1.5-7.7 x10^9/L
Platelets	202	263	240	245	221		127	107	150-450 x10^9/L
INR					1.11	1.13	1.00	1.04	

Table: Laboratory results of the patient

(ALP alanine aminotransferase, AST aspartate aminotransferase, GGT gamma-glutamyltransferase, INR international normalized ratio, CK creatinine kinase, LDH lactate dehydrogenase)

Discussion

There are many different etiological factors that cause liver dysfunction. Mushroom poisoning, which can lead to liver failure and death, is one of the most important causes. It is reported that the cause of death of more than 90% of patients with mushroom poisoning is acute liver failure [3]. Usually the first symptoms are associated with the gastrointestinal tract. Liver dysfunction is seen in the late stage. Detection of amatoxins by ELISA (enzyme-linked immunosorbent test) may be useful in early diagnosis [4]. Medical history plays an extremely critical role in early diagnosis and treatment.

Numerous infectious agents, although rare, can cause acute liver dysfunction. Brucella is not primary hepatotropic, but it can cause liver enzyme elevation. Organ involvement is observed in approximately 30% of disease. Anemia, leukopenia and thrombocytopenia may be occured due to hematologic system damaged. In addition, hepatic or splenic abscesses accompanied by elevated transaminase can be detected. Moderate hepatosplenomegaly, and sometimes acute hepatitis may develop. Acute hepatic failure is observed very rarely when an underlying etiological factor is present [5].

In this case, viral hepatitis, vascular events and autoimmune disease were considered in the differential diagnosis. When epidemiological, demographic, clinical features, laboratory findings and diagnostic imaging were evaluated together, these diagnoses were ruled out. Mushroom intoxication and brucellosis were considered in the differential diagnosis with the results obtained. Fungal intoxication could not be clearly excluded in the diagnosis due to amatoxin analysis could not be performed and intoxication was suspected in family members. Since the sero-agglutination test Rose Bengal was weakly positive and the patient responded to antibiotherapy, liver enzyme dysfunction caused by brucella was considered in the diagnosis.

Conclusion

In patients presenting with a history of eating mushrooms, it is very important to keep in mind other rare causes that may cause liver dysfunction apart from intoxication.

Declaration

The authors have no conflicts of interest to declare.

References:

1) Öncel, S. (2016). Brusella enfeksiyonları: Değerlendirme ve yönetim. Kocaeli Üniversitesi Sağlık Bilimleri Dergisi, 2(3), 25-30.

2) García Casallas, J. C., Villalobos Monsalve, W., Arias Villate, S. C., & Fino Solano, I. M. (2018). Acute liver failure complication of brucellosis infection: a case report and review of the literature. Journal of medical case reports, 12(1), 62.

3) Li Y, Mu M, Yuan L, Zeng B, Lin S. Challenges in the early diagnosis of patients with acute liver failure induced by amatoxin poisoning: Two case reports. Medicine (Baltimore). 2018 Jul;97(27):e11288.

4) Pu Y, Yao QM, Zhong JJ, Yu CM. [Three cases of mushroom poisoning caused by lethal Amanita species]. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 2021 Oct 20;39(10):787-788. Chinese.

5) Talwani R, Gilliam BL, Howell C. Infectious diseases and the liver. Clin Liver Dis. 2011 Feb;15(1):111-30.