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Case report

A rare case of *Amanita vidua* poisoning

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ABSTRACT

The similarities between poisonous and edible mushrooms, combined with the inexperience of foragers, increase the risk of mushroom poisoning. Amatoxin-containing mushrooms are responsible for most fatal mushroom poisonings. However, there is limited clinical data regarding the toxin content of certain *Amanita* species, such as *Amanita vidua*, and the severity of poisoning they may cause if consumed. This case report presents the first documented instance of poisoning from *A. vidua* in the literature. A 72-year-old woman mistakenly consumed a toxic *A. vidua* mushroom, believing it to be the non-toxic *Amanita vaginata*. Approximately 10 hours later, she arrived at the emergency room with symptoms including nausea, vomiting, and diarrhea. All laboratory parameters were normal on the first day. However, the patient subsequently developed refractory circulatory shock and multiple organ failure, which began on the second day and rapidly worsened over the following days. Liver function tests peaked at 73 hours post-ingestion, showing aspartate transaminase levels of 2534 IU/L, alanine transaminase levels exceeding 942 IU/L, and lactate dehydrogenase levels above 3325 U/L. We identified *A. vidua* by both macroscopic and microscopic examinations. Additionally, high-performance liquid chromatography (HPLC) analysis of the consumed *A. vidua* mushroom detected the presence of toxic alpha- and beta-amanitins, confirming amatoxin poisoning. This study details a case of *A. vidua* mushroom poisoning that occurred in Türkiye, ultimately resulting in death despite treatment. This is significant as it represents the first known case of poisoning worldwide from *A. vidua*, highlighting its potential lethality when ingested by humans.

1. Introduction

Wild mushrooms are traditionally consumed by local people worldwide for their better taste. Poisonous and edible mushrooms have many similarities in appearance, smell, color, and taste. This similarity increases the risk of poisoning due to unnecessary courage and inexperience caused by the local people's overconfidence in this issue. (He et al., 2023, 2024; Kose et al., 2015; Yilmaz et al., 2015; Diaz, 2018).

The majority of fatal mushroom poisonings are caused by mushrooms containing amatoxin. There are different varieties of this toxin family, including alpha-amanitin, beta-amanitin, and gamma-amanitin, all of which are severely toxic (Bayram et al., 2022). These toxins have a high heat stability and are not destroyed by cooking or drying. They are also resistant to enzyme and acid degradation. Therefore, they are not inactivated in the gastrointestinal tract when ingested after cooking (Garcia et al., 2015; Yilmaz et al., 2015; Sharma et al., 2021). The primary target organ in toxicity is often the liver, and hepatic toxicity can often be fatal. Serious complications such as coagulation disorders and renal failure may also accompany the picture due to hepatic and renal involvement (Kose et al., 2015; Diaz, 2018; Vetter, 2023).

Amanita phalloides, *A. virosa*, and *A. verna* are known in the literature to cause frequent poisoning due to their high amatoxin content (Kaya et al., 2013; Yilmaz et al., 2014; Diaz, 2018; Vetter, 2023). However, there is still no precise data in the literature on whether some *Amanita* species cause poisoning. The findings of some past studies using simple analysis and identification methods also create confusion about the toxin content of some fungi. A limited number of studies show that one of these mushrooms, *Amanita vidua*, contains high levels of amatoxin. (Alvarado et al., 2022). However, there is no clinical data on the degree of poisoning that may occur in humans if this mushroom is consumed. This mushroom was recently described in detail in a study from Türkiye, and it was shown for the first time that it grows in Türkiye (Akata et al., 2024). In this case report, we present data on the first severe poisoning case reported from the region where the mushroom was identified in

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Türkiye. The patient, who was reported to have consumed a fatal amount of this mushroom, was hospitalized after poisoning but died on the fourth day of hospitalization despite all interventions. We believe that the case is important as it is the first case of *A. vidua* poisoning reported in the literature from all over the world.

2. Case report

A 72-year-old woman weighing 65 kg was admitted to the emergency department with complaints of nausea, vomiting, and diarrhea. In her anamnesis, she stated that she had eaten a natural mushroom collected from the region where she lived at noon, and her complaints of nausea, vomiting, and diarrhea intensified in the evening of the same day. She consumed these mushrooms in a single meal and did not have repeated consumption. The patient experienced mild nausea 3–4 hours after eating the mushroom. At first, he did not take these symptoms seriously and thought they would go away alone. Unfortunately, the symptoms worsened in the following hours. The patient had no chronic systemic disease except type 2 diabetes mellitus and presented to the emergency department approximately 8–10 hours after eating these mushrooms. Initial vital signs and physical examination were normal. Initial laboratory tests included liver function tests, renal function tests, and coagulation parameters. In the tests, aspartate aminotransferase (AST) 20 IU/L, alanine aminotransferase (ALT) 14 IU/L, lactate dehydrogenase (LDH) 181 U/L, BUN 25.4 mg/dL, creatinine 0.99 mg/dL, total bilirubin 0.46 mg/dL, direct bilirubin 0.2 mg/dL, total protein 79 g/dL, albumin 44 g/dL, activated partial thromboplastin time (APTT) 21.8 seconds, prothrombin time (PT) 10.1 seconds, international normalized ratio (INR) 0.8. Complete blood count showed leukocyte count (WBC) 9.73 10^3/μL, neutrophil 7.49 10^3/μL, and hemoglobin 11.2 g/dL. Hydration was provided by giving 0.9 % sodium chloride and 5 % dextrose (against the risk of hypoglycemia), and the patient was hospitalized for follow-up. Acetylcysteine infusion treatment and silymarin treatment, if available in case of elevated liver function tests, were recommended by the National Poison Information Center. The next day, in the evening hours, hypotension developed unresponsive to hydration, and she was admitted to the internal medicine intensive care unit. Hydration support, noradrenaline infusion (5–10 μg/min), and simultaneous acetylcysteine treatment (150 mg/kg for 1 hour, followed by 50 mg/kg for 4 hours, followed by 150 mg/kg for 16 hours) were started.

On the second day of intensive care unit follow-up, the patient was conscious, breathing spontaneously, and receiving oxygen therapy with a mask. Her hemodynamics stabilized with noradrenaline infusion. However, nausea, vomiting, and diarrhea persisted. Multiple vasoactive drug support treatment was initiated upon the persistence of hemodynamic problems. Amiodarone (5 mg/kg) and digoxin (8 μg/kg) were given for atrial fibrillation with high ventricular response. Liver enzyme levels started to increase, and multiple organ failure (renal, hepatic, and circulatory shock) developed. A temporary hemodialysis catheter was inserted into the left femoral vein, and 3 L of therapeutic plasma exchange was performed with fresh frozen plasma for 2 hours. Continuous renal replacement therapy (in venous-venous hemodiafiltration mode) was started after plasmapheresis support due to the persistence of metabolic acidosis. Invasive mechanical ventilation support was given after elective intubation due to increased vasoactive requirement and deterioration in general condition. Empirical antibiotic treatment (piperacillin/tazobactam and vancomycin combination) was initiated due to high procalcitonin levels (7 ng/mL).

On day 3, vasoactive supportive drug therapies were increased in the patient who still had multiple organ failure and refractory circulatory shock. In addition to high doses of noradrenaline (10–20 μg/kg/min), hydrocortisone (200 mg/day), adrenaline, terlipressin (1.5 mg/4 hours), and dobutamine (0.5–1 μg/kg/min) infusion were added. In the follow-up of the patient, drug doses were gradually increased with invasive blood pressure monitoring. The second session of therapeutic plasma exchange was performed under multiple vasoactive drug support, followed by continuous venovenous hemodiafiltration due to deep metabolic acidosis. As the general condition of the patient deteriorated considerably and the procalcitonin level increased, antibiotic treatment was extended to meropenem + vancomycin. Liver function tests of the patient, which started to increase on the second day after eating mushrooms, peaked at the 73rd hour with AST, 2534 IU/L; ALT, >942 IU/L. The ALT value is shown this way because it exceeded the maximum measurement limit of the kit used for the test. Fig. 1 shows the dramatic increase in AST levels following hepatotoxicity caused by poisoning.

Among other parameters, the LDH value reached its peak at the 86th hour (>3325 U/L), and the D-Dimer was 4527 ng/mL. Coagulation parameters, which started to deteriorate on the second day, reached their peak at the end of the third day (APTT 98.9 seconds; PTZ 44.2 seconds; INR 4.1). Total bilirubin peaked at 2.2 mg/dL, and direct bilirubin was 1.62 mg/dL.

On the morning of the fourth day, the patient developed asystole, and cardiopulmonary resuscitation was started with adrenaline. Despite 45 minutes of resuscitation, spontaneous circulation was not restored, and the patient died. Indeed, when the patient was first admitted to the intensive care unit, his Apache II score was 29 (expected mortality rate 67 %), and her SOFA score was 8, while the SOFA score measured on the last day was 18.

While the patient was being monitored in the intensive care unit, we visited the place where she collected the mushrooms with his relatives (Türkiye-Uşak, Yeşildere village, on calcareous soils, under the Valonia oak (*Quercus ithaburensis* Decne subsp. *macrolepis* (Kotschy) Hedge & Yalt.), 38° 35′ 21″ N, 29°17′ 56″ E, 1005 m, June 02, 2023, ANK Akata & Kaya 007). Some of the mushrooms described were collected. Relatives of the patient confirmed that these mushrooms were among the suspected mushrooms eaten by the patient. The distinction between *A. vidua* and *A. vaginata* was confirmed by macroscopic evaluation by an experienced mycologist (Fig. 2a). We believe that the patient may have mistaken the edible *A. vaginata* mushroom for the toxic *A. vidua* mushroom, potentially consuming the poisonous *A. vidua* instead. As illustrated in Fig. 2b, it can be challenging for inexperienced individuals to differentiate between these two mushrooms.

A. vidua (Gasch, G. Moreno & P.-A. Moreau) is a rare ectomycorrhizal fungus native to Mediterranean oak woodland habitats. It features a smooth, whitish to cream-colored cap that often has yellowish or pinkish tints and lacks any veil remnants. As it matures, the cap flattens and usually exhibits a small umbo. Its white stipe is marked by a prominent ring and a sack-like volva. The gills are free from the stem and range in color from white to cream. This species produces a white spore print, with smooth spores that are shaped from subglobose to broadly ellipsoid. Mushroom expert Akata et al. (2024), who contributed to our study, recently conducted a comprehensive taxonomic description of *A. vidua* (Akata & Kaya 007) specimens collected from this region. This

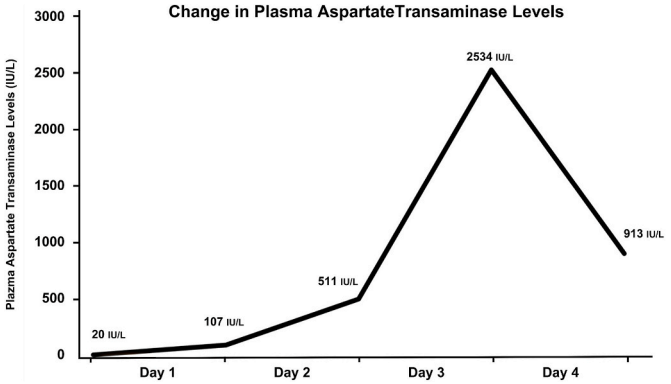


Fig. 1. Dramatic increase in aspartate transaminase (AST) levels after ingestion of *Amanita vidua* mushroom.



Fig. 2. *Amanita vidua* and *Amanita vaginata* mushrooms from the patient's previous collection site. **a**, Both mushrooms side by side. (The ones with longitudinal striated edges are *Amanita vaginata* mushrooms, while the others are *Amanita vidua*) **b**, A large number of both mushrooms were collected in one basket. It does not seem easy to tell them apart.

description serves as the primary reference for mycological studies related to this species in our country. The macroscopic and microscopic appearance of the *A. vidua* mushroom is shown in Fig. 3a and b.

HPLC analysis of *the A. vidua* mushroom consumed by the patient showed the presence of toxic alpha- and beta-amanitin chromatographically, as in our previous similar studies (Kaya et al., 2013; Yilmaz et al., 2014). Thus, the patient's intoxication due to amatoxins was confirmed (Fig. 4).

More detailed toxin content and quantity analysis of the fungus was not performed at this stage as it is the subject of a further analytical study, but it is planned to be done in future studies.

3. Discussion

Amatoxins can be found in *Amanita*, *Galerina*, *Lepiota*, and other mushroom species (Garcia et al., 2015; Kaya et al., 2013; Yilmaz et al., 2014; Diaz, 2018; Akata et al., 2020; Vetter, 2023). The local population often confuses these deadly *Amanita* mushrooms with edible mushrooms that grow in the same geographical area and habitat, leading to severe poisoning. Experienced mushroom experts can only distinguish these species based on distinct and critical morphological characteristics. The original taxonomic description of *A. vidua* specimens, whose presence was first demonstrated in our country, was recently detailed in a study by Akata et al. (2024). In our study, which presents the first fatal poisoning case involving *A. vidua* from our country and the world, we also confirmed the presence of alpha and beta-amanitin in the mushroom using HPLC analysis. However, in future studies, it would be beneficial to elucidate further the amatoxin content of *A. vidua* grown in our country and compare it with similar amatoxin-containing mushrooms such as *A. verna*.

The patient stated that he had collected and consumed mushrooms many times before, but that this was the first time he had experienced such symptoms. Based on information obtained from the patient and his relatives, the area where he had collected the mushrooms was visited. Two different types of mushrooms similar to those described were observed in the area, and all were collected. Macroscopic and microscopic examination revealed that some mushrooms were edible *A. vaginata*, while others were poisonous *A. vidua* mushrooms. This explains why the patient had not been poisoned before, as he had likely consumed edible *A. vaginata* mushrooms on multiple occasions.



Fig. 3. Macroscopic and microscopic characteristics of *Amanita vidua* mushrooms collected from the patient's mushroom-picking site. **a**, Fruiting body. **b**, Spores in Melzer's reagent.

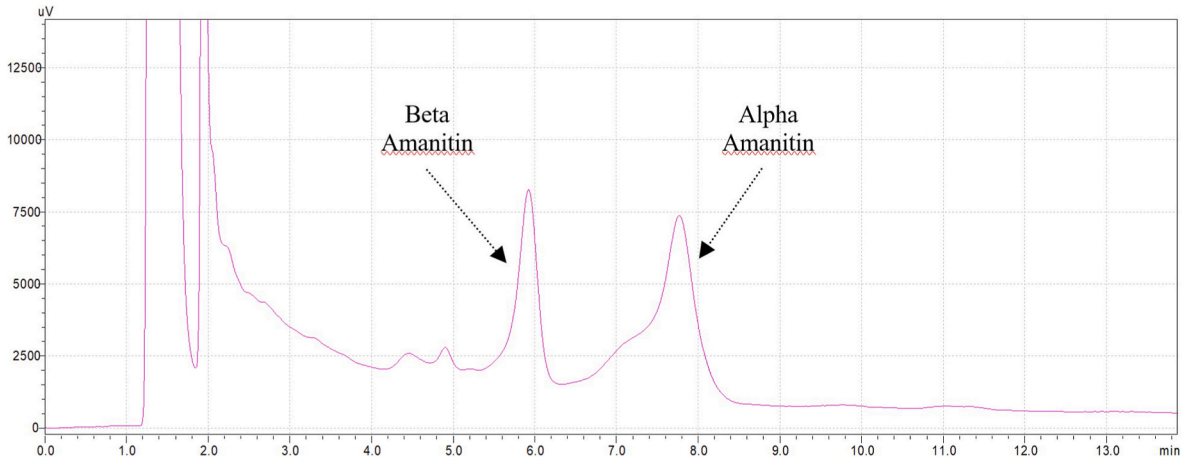


Fig. 4. HPLC analysis of the *Amanita vidua* mushroom to detect alpha- and beta-amanitin.

A. vaginata can be distinguished by its white color, striped cap edge, and stem without a ring. However, due to its similar morphology and habitat, non-professional mushroom collectors can easily confuse it with the toxic *A. vidua*. In our case, the confusion led the patient to consume *A. vidua* mushrooms containing amatoxin instead of the *A. vaginata* mushrooms he had previously collected and consumed regularly.

The symptoms of amatoxin poisoning can vary from mild gastrointestinal issues to severe outcomes, including death. Key factors influencing this condition include the type of mushroom consumed, the quantity ingested, and the concentration of toxins (Diaz, 2018; Vetter, 2023; Yilmaz et al., 2015; Kose et al., 2015). Symptoms are typically categorized into three stages. In the first stage, gastrointestinal symptoms predominate, often manifesting as nausea, vomiting, abdominal pain, diarrhea, and low blood pressure (hypotension). The second stage is the hepatic stage, when liver enzyme levels rise. In the final stage, the patient experiences progressive acute liver and kidney failure, leading to a worsening of their condition. Due to extensive liver damage, serum levels of AST, ALT, and LDH increase, and coagulopathy develops, which affects the coagulation factors. Additionally, encephalopathy, hepatic coma, and cerebral edema may occur, often resulting in death (Garcia et al., 2015; Wennig et al., 2020; Vetter, 2023).

In this case, the patient initially presented to the emergency department with gastrointestinal symptoms (nausea, vomiting, diarrhea). Despite treatment, the patient's hemodynamics deteriorated, and hypotension developed in the following period. The increase in liver enzyme levels, which started on the second day, accelerated dramatically and peaked on the third day. Subsequently, the patient developed renal failure accompanied by anuria. Rapid deterioration of liver function led to impaired coagulation factors and coagulation parameters. Considering the patient's current comorbidities, newly prescribed medications, the possibility of food poisoning, mycotoxic syndrome (such as cholinergic syndrome), and potential infection-related sepsis, we carefully reviewed various differential diagnoses. Echocardiographic findings were not particularly significant, and there were no prominent symptoms of cholinergic syndrome, such as bradycardia, bradypnea, bronchorrhea, lacrimation, muscle weakness, fasciculations, or blurred vision. Clinical and laboratory findings did not indicate the presence of sepsis due to an infection. Shortly after admission to the hospital, we initiated continuous hemodialysis, plasmapheresis, and vasoactive agents to manage the developing multiple organ failure, including circulatory shock, heart failure, renal failure, and liver failure. The most striking detail in the patient's history and clinical findings, obtained from both the patient and their relatives, was the history of mushroom consumption. As is well known, poisoning from particular mushroom species can cause severe toxicity affecting the heart, kidneys, and especially the liver, leading to life-threatening conditions and hemodynamic instability. While the patient was in the intensive care unit, we determined that the mushrooms consumed were toxic *A. vidua* mushrooms containing amanitin. Subsequent analyses confirmed the presence of amanitin in the mushrooms, thereby confirming that the poisoning was indeed caused by amanitin. The patient's highly progressive and severe clinical course indicates exposure to mushroom poisoning containing high concentrations and large amounts of amatoxin. The lack of targeted treatments remains one of the most significant challenges in managing mushroom poisoning. Although various agents are used in treatment, their efficacy remains inadequate. This situation also highlights the lack of a clear consensus on how to treat amatoxin poisoning. Until recently, numerous drugs have been tested in both experimental and clinical studies in the hope of finding a treatment (Kaya et al., 2016; Vetter, 2023; Bayram et al., 2022; Wang et al., 2023). Most treatments, including supportive care and antioxidant therapies, aim to prevent or limit existing liver damage. Treatments such as penicillin G, silibinin, silymarin, cimetidine, thioctic acid, various antioxidants, vitamin C, acetylcysteine, erdostein, indocyanine green, hormones, and steroids are still used clinically and experimentally (Tong et al., 2007; Ward et al., 2013; Kaya et al., 2016; Wennig et al., 2020; Bayram et al., 2022; Vetter, 2023). Since some of these treatments are not routine medications, difficulties may be encountered in obtaining them in daily practice. In our case, due to the rapid progression of the clinical process, obtaining the silymarin medication recommended by the National Poison Control Center was impossible, and it could not be used on the patient. Instead, supportive therapy and acetylcysteine therapy were administered.

Although the lethal dose of amatoxins in humans is not clearly known, there are studies claiming that even as low as 0.1 mg/kg can be lethal (Wennig et al., 2020; Kose et al., 2015). In a previously reported case of *A. phalloides* poisoning, we stated that oral consumption of amatoxin above approximately 0.32 mg/kg may be lethal (Yilmaz et al., 2015). As a matter of fact, it was calculated that approximately 50 mg of mushrooms were consumed in this study. In this case, we demonstrated the presence of amatoxin in the mushroom content consumed by the patient chromatographically by HPLC. The progressive and severe course of the patient's clinic condition suggests that much higher amounts of mushrooms may have been consumed. If the amatoxin content and concentrations in *A. vidua* mushrooms in this region are analyzed in more detail in future studies, it will be possible to estimate this amount more accurately. We are planning further studies to determine the toxin composition and amount of this mushroom in more detail. Although *A. vidua* is considered to be in the same group as toxic and amatoxin-containing fungi, only one recent study (Alvarado et al., 2022) was found in the literature in which toxin content or quantity was evaluated. With our planned study, it will be possible to compare the

toxin contents and amounts of *A. vidua* mushrooms from different regions of the world. Due to technical limitations in the laboratory at the time of the patient's poisoning, amanitin analysis could not be performed on the patient's blood or urine, which can be considered a limitation of our study. However, the patient was poisoned by a mushroom containing amanitin. The patient's medical history and subsequent clinical and laboratory findings also indicate this. Additionally, the macroscopic and microscopic evaluation of the mushrooms consumed by the patient revealed that the patient had ingested the toxic *A. vidua* mushroom. Subsequent analytical studies confirmed that this mushroom contained alpha and beta-amanitin, as determined by HPLC. Currently, we are working on a project to develop a rapid diagnostic test that can detect the presence of amanitin in patients' serum or urine using a specific immunochromatographic method for similar poisonings.

There is no clinical data in the literature on the extent to which *A. vidua* mushroom can cause severe poisoning or is fatal if consumed by humans. This mushroom, which can grow in different parts of the world, was recently identified in a study from our country, and it was shown for the first time that it can be found in Türkiye (Akata et al., 2024). As a matter of fact, the mushroom consumed in our case was *A. vidua*, which grows in this region, and it is valuable because it is the first *A. vidua* intoxication reported in the literature, both in our country and in the world.

4. Conclusion

In our study, we presented a case of poisoning due to an *A. vidua* mushroom from Türkiye, which died despite treatment. This case is also the first clinical data in the world regarding this mushroom. Although it was seen in our study that a more aggressive treatment was needed, the treatment options for amatoxin poisoning were quite limited and inadequate, making this impossible. Our case once again demonstrates the importance of correctly identifying mushrooms and informing clinicians and local people about their toxin content by poison centers in regions where natural mushrooms are frequently consumed.

CRediT authorship contribution statement

Ertugrul Kaya: Writing – original draft, Project administration, Methodology, Formal analysis, Conceptualization. **Esra Sultan Karabulut Keklik:** Methodology, Investigation, Formal analysis. **Ilgaz Akata:** Investigation, Formal analysis, Conceptualization. **Ersan Horoz:** Writing – original draft, Methodology, Investigation, Data curation. **Ismail Yilmaz:** Writing – review & editing, Project administration, Conceptualization.

Ethical statement

The authors declare that this manuscript complies with the Elsevier Ethical Guidelines for Journal Publication.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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