Amanita Phalloides Intoxication Misdiagnosed as Acute Appendicitis: A Case Report

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ABSTRACT

Background: Acute appendicitis is the most common surgical emergency of the childhood. Its diagnosis is really challenging. A constellation of through history taking, physical examination, radiographic investigations, and laboratory analysis should be used to balance the risk of delayed operative intervention against the removal of a normal appendix. Here a case with misdiagnosis of acute appendicitis instead of Amanita Phalloides intoxication was presented.

Case: A 6-years-girl was referred with history of constant, not-colicky lower quadrant and periumblical abdominal pain from 24 hours ago; associated with nausea and vomiting. In physical examination generalized tenderness particularly in lower quadrants was found. leukocytosis (WBC=22000) with a shift to left (PMN=91%) and hypoglycemia was found too. Alvarado score for diagnosis of acute appendicitis was 7 out of 10. In laparotomy, the appendix appeared normal. Blood study revealed INR>7 during operation which was partially corrected with FFP infusion. The patient developed decreased levels of consciousness and was transferred to ICU. Further history taken from her family by the clinical toxicologist revealed that she had eaten wild mushrooms. After examination of the remaining mushrooms by an experienced biologist, the diagnosis of *Amanita Phalloides* intoxication was established. The patient died tomorrow regardless of appropriate interventions.

Conclusion: Mushroom poisoning should be taken into account in evaluation of the patients suspected to have acute appendicitis. If diagnosis of mushroom poisoning was made earlier by proper history taking and a high index of suspicion, the patient would receive the available treatment modalities earlier and her chance of survival would increase.

Keywords: Acute Appendicitis, Amanita Phalloides, Intoxication.

INTRODUCTION

In 1886, Reginald Fitz coined the term "appendicitis" and proposed that early surgical intervention is essential in treatment of the disease. More than 100 years later, acute appendicitis still remains one of the most common causes of acute abdominal pain necessitating surgery. Generally, if the patient presents the typical signs and symptoms of right lower quadrant (RLQ) pain and tenderness, nausea, vomiting, and anorexia, in the setting of fever and leukocytosis, especially with a left shift, the decision to operate cannot be contested (1).

Furthermore, appendicitis represents a real, everyday working problem for the primary physician and those who care for the children. It is the most common surgical emergency of the childhood. Appendicitis may present at any age, although it is a rare, but one of the most challenging diagnoses among very young children. Delayed presentation and misdiagnosis of acute appendicitis are frequent occurrences in this age group and result in

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increased incidence of appendiceal perforation (2).

The evaluation of patients with suspected acute appendicitis is clinically complex. A constellation of history, physical signs, radiographic investigation, and laboratory analysis is used to balance the risk of delayed operative intervention against the removal of a normal appendix (non-therapeutic laparotomy). The cornerstone of the diagnosis of acute appendicitis has traditionally been the combination of history and physical examination. The overall accuracy for the clinical examination in diagnosing acute appendicitis has been reported to be 70% to 87% (54% to 70% in children and 50% to 70% in women of childbearing age) (3).

Cases of wild mushroom poisoning with *Amanita phalloides* (*A. phalloides*), the 'death cup' mushroom, occur sporadically, usually during late summer and autumn months, when climatic conditions favor fungal growth. Given the high mortality of A. phalloides poisoning (30–90% of untreated cases), case recognition and prompt optimal treatment are essential (4-7).

This study presents a case referring with acute abdomen that underwent laparotomy while the final diagnosis was poisoning with death cap.

CASE PRESENTATION

A 6-years-girl referred to the pediatric surgery ward with history of abdominal pain beginning from around 24 hours before. Her pain was constant, not-colicky in nature, and was located in lower quadrant and periumblical regions. She was complaining from nausea and vomiting, which had begun since two days ago and was aggravating with ingestion. She had had two episodes of vomiting and was also complaining from anorexia. The patient had referred to OPD ward the day before and had taken some medications, but her symptoms had not improved. In her past medical history, only surgery for cataract had been noted a year before.

In physical examination, the patient was conscious, calm, emaciated, and dehydrated. She had tachycardia and her oral temperature was 38°C. No remarkable findings were established in head and neck and chest examination. Generalized tenderness particularly in lower quadrants was found in abdominal examination. Rectal examination was unremarkable.

In preliminary lab study, leukocytosis (WBC=22000) with a shift to left (PMN=91%) was seen. Hematocrit, platelet count and U/A were reported normal.

Alvarado score for diagnosis of acute appendicitis was 7 out of 10. In abdominal sonography, few apristaltic bowel loops in RLQ suggesting loops around the inflamed appendix as well as some lymph nodes in RLQ, echogenisity of mesentery lipids and little free fluid in pelvis were reported; however, no closed appendiceal loop was seen. The findings put forward diagnosis of acute appendicitis.

During admission, the patient developed convulsion that improved with DW50% infusion. Concomitant serum glucose analysis was 28 mg/dl. Neurologic examination and brain CT scan were normal. Owing to the history of cataract, low body weight, and hypoglycemia, the possibility of a metabolic disorder was taken into account and appropriate diagnosis and management were deferred till improvement from the acute phase of her disease.

The patient was sent to the operating room for laparotomy. In laparotomy, the appendix appeared normal and only little clear serous fluid in pelvic cavity and lymphadenopathy in meso of small intestine were observed. The procedure came to a stop with normal homeostasis and without any complication; however, the anesthesiologist noticed oozing from the site of IV line. Blood study revealed INR>7 which partially was corrected with FFP infusion. At the same time, blood glucose, BUN, Cr, platelets, and PTT were in the normal range.

The following day, the patient developed decreased levels of consciousness again and did not respond to hypertonic glucose infusion. She was transferred to ICU and toxicologic consultation was requested. Further history taken from her family by the clinical toxicologist revealed that she had eaten mushrooms obtained from a jungle near her house. After examination of the remaining mushrooms by an experienced biologist, the diagnosis of *Amanita Phalloides* intoxication

was established. Appropriate interventions got started; nevertheless, the patient expired the next day with an outstanding hepatic failure.

DISCUSSION

Toxins have had major roles in human societies for thousands of years. Interactions between surgeons, both generalists and subspecialists, and those taking care of poisoned patients have been extensive throughout history. The advancement of the science of toxicology, the development of regional poison control centers, the development of emergency medicine, and the development of medical toxicology as a subspecialty have led to more appropriate and creative interactions among medical toxicologists, emergency physicians, and surgeons. Throughout the history of mankind, surgeons have used their skills to evaluate the effects of toxins. Such interactions should not be confined to parenteral substance use, inhalational and insufflational substance use, alcohol and drug abuse as they relate to traumatic events, occupational risks, envenomations, suicide, complications of gastrointestinal decontamination, thermal burns and smoke inhalation, ingestion of acids and burns, toxins leading to organ transplantation, and substance withdrawal (8). The presented case was a prototype. Although clinical judgment still remains the most important diagnostic tool for acute appendicitis (9), other differential diagnoses should be noticed, as well. More accurate history taking and physical examination may assist earlier diagnosis in our case.

Mushroom poisoning refers to the severe and often deadly effects of various toxins that are found in certain types of mushrooms. Mushrooms are fungi, saprophytic in nature, that use organic material from dead plants and animals (10). From a clinical and diagnostic perspective, in humans, Lampe (1978) grouped toxic mushrooms according to the six main syndromes encountered:

1- A diverse group is usually responsible for transient gastrointestinal irritation associated with a variety of toxins.

2- The Amanita phalloides group containing cyclic polypeptides is responsible for the most serious and often fatal intoxication, including delayed, irreversible cytotoxicity predominantly affecting the liver and kidney.

3- The Gyromitra group containing methyl hydrazine derivatives causes similar but less severe cytotoxicity.

4- A group induces muscarine-related symptoms such as salivation and increased gastrointestinal motility.

5- A group causes sensitivity to alcohol by inhibiting acetaldehyde dehydrogenase.

6- The hallucinogenic mushrooms category can be subdivided into the Psilocybe group causing a hallucinogenic syndrome, and a group causing delirium associated with sleep or coma. Both *A.* pantherina and *A.* muscaria fall into the latter group (11).

Most cases of fatal mushroom poisoning in the world occur after the ingestion of Amanita species, primarily of *Amanita phalloides*. The toxic agents of *A. phalloides* are cyclopeptides which form two groups of toxicologically different compounds: The amatoxins, which are lethal within 2±8 days and highly toxic (LD50 0.4±0.8 mg/kg, in some animal species, per orally) and the phallotoxins, which are lethal within 2±5 hours and less toxic than the amatoxins (LD50 2±3 mg/kg, but only when they are injected). The third group of active agents, the virotoxins, are mono-cyclic heptapeptides. All groups of toxic peptides contain a tryptophan residue substituted at position 2 of the indol ring by a sulfur atom. Amatoxins and phallotoxins are bicyclic, cross-linked by the 2'-bound sulfur group (12).

After a relatively long period of latency (on average 8±10 hours, but in a few cases 24 hours and in extreme cases 36 hours), the symptoms of the initial phase of intoxication (excessive vomiting and diarrhea, for 1 or 2 days) are observed, leading to dehydration which culminates in the hypovolemic shock (13). Our case had vomiting and dehydration, too but did not present diarrhea. Abdominal pain is usually colicky in nature (14), but our patient had constant abdominal pain; an event which may contribute to delay in diagnosis. Concomitantly, hypoglycemia develops which can be lethal in rare cases. The patient also had hypoglycemia; however, it was erroneously assigned to an unknown metabolic process. Although the patient will feel better when the

gastrointestinal disease is over, hepatic lesions develop, as determined by the serum concentration of the liver enzymes, such as SGOT, SGPT, and LDH. Together with the increase in these enzymes, blood coagulation is severely disturbed, which may induce internal bleeding. Our patient similarly developed bleeding tendency manifested by oozing from the IV site, but no suspicion to mushroom poisoning arose. During the renal phase, the liver enzymes in the serum continue to increase. Hepatic failure can cause encephalopathy and coma. High values of creatinine and urea indicate additional damage to the kidney cells (hepatic coma combined with renal failure). Death may occur as late as 6±8 days after the ingestion of mushrooms (12).

CONCLUSION

Our case received the recommended treatments for Amanita phalloides poisoning, including supportive measures, plasmaphresis, NAC, and penicillin (10,13,15). There was no time for liver transplantation and opportunity for GI decontamination was lost. If diagnosis of mushroom poisoning is made earlier by proper history taking and a high index of suspicion, the patient will receive the available treatment modalities earlier and their chance of survival will increase; otherwise, the mortality rate will be high (10).

REFERENCESG

1. Mazeh H, Epelboym I, Reinherz J, Greenstein AJ, Divino CM. Tip appendicitis: clinical implications and management. The American Journal of Surgery 2009;197(2):211-5.
2. Mallick MS. Appendicitis in pre-school children: A continuing clinical challenge. A retrospective study. International Journal of Surgery 2008;6(5):371-3.
3. Birkhahn RH, Briggs M, Datillo PA, Van Deusen SK, Gaeta TJ. Classifying patients suspected of appendicitis with regard to likelihood. The American Journal of Surgery 2006;191(4):497-502.
4. Hanrahan JP, Gordon MA. Mushroom poisoning. JAMA: The Journal of the American Medical Association 1984;251(8):1057.
5. Aji DY, Çalişkan S, Nayir A, Mat A, Can B, Yaşar Z, et al. Haemoperfusion in Amanita phalloides poisoning. Journal of tropical pediatrics 1995;41(6):371.
6. Sabee AI, Kurkus J, Lindholm T. Intensive hemodialysis and hemoperfusion treatment of Amanita mushroom poisoning. Mycopathologia 1995;131(2):107-14.
7. Sakellariou G. Plasmapheresis as a therapy in specific forms of acute renal failure. Nephrology, dialysis, transplantation 1994;9:210-8.
8. RG L. Lewis RG. The Surgical Complications of Toxins The Journal of Emergency Medicine 1999 Nov 17(6):1055-64.
9. Bergeron E, Richer B, Gharib R, Giard A. Appendicitis is a place for clinical judgement. The American Journal of Surgery 1999;177(6):460-2.
10. Jan MA, Siddiqui TS, Ahmed N, Ul-Haq I, Khan Z. Mushroom poisoning in children: clinical presentation and outcome. J Ayub Med Coll Abbottabad 2008;20(2):99-101.
11. Satora L, Pach D, Ciszowski K, Winnik L. Panther cap Amanita pantherina poisoning case report and review. Toxicon 2006;47(5):605-7.
12. Vetter J. Toxins of Amanita phalloides. Toxicon 1998;36(1):13-24.
13. Cappell MS, Hassan T. Gastrointestinal and hepatic effects of Amanita phalloides ingestion. Journal of clinical gastroenterology 1992;15(3):225.
14. Lionte C, Sorodoc L, Simionescu V. Successful treatment of an adult with Amanita phalloides-induced fulminant liver failure with molecular adsorbent recirculating system (MARS). Rom J Gastroenterol 2005;14(3):267-71.
15. Alves A, Gouveia Ferreira M, Paulo J, França A, Carvalho Á. Mushroom poisoning with Amanita phalloides--a report of four cases. European Journal of Internal Medicine 2001;12(1):64-6.