CASE-REPORT

DOI: 10.31736/jist/v14.i1.2018.40-44

Ultrasoundguided Percutaneous Transhepatic Gall bladder Aspiration (PTGBA) in the treatment of Mushroom poisoning

Journal Homepage :www.jist.org.in; Email: article@jist.org.com

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ARTICLE INFO

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How to Cite this article: cle: Dutia A, Boruah DK, Mitchell T, Deori P. Ultrasound Guided Percutaneous Transhepatic Gall bladder AspiraTIon (PTGBA) in the Treatment of Mushroom Poisoning.Journal of Indian Society of Toxicology 2018;14(1):1-5.= DOI: 10.31736/jist/v14.i1.2018.40-44

Keywords:
mushroom poisoning;
ultrasound guided aspiration;
percutaneous transhepatic gall bladder aspiration;
PTGBA.

Conflicts of Interest and Fundings: Declared none

Received on 11th May 2018
Accpeted on 30th May 2018
Published on 30th July 2018

ABSTRACT

Mushroom poisonings (Mycetismus) are rare conditions have been reported frequently from North East India during the monsoon seasons. In one of the earlier studies we noted a high mortality (43.75%) even after using all the conventional agents in literature. Studies in dogs have shown that the toxins are excreted in the bile and much of the toxin excreted in this way is reabsorbed into the liver with the bile. Interruption of the cycling of the toxin by bile duct cannulation can reduce the toxic effects of α-amantin in dogs. We report three males, (58 , 28 and 24 years old respectively) who came to our institute with mushroom poisoning presenting with dehydration, acute liver failure, raised liver enzymes and hyperbilirubinemia. Though they were given conventional treatment their raised liver enzymes indicated high risk for mortality. After proper consent, ultrasound guided percutaneous transhepatic gall bladder aspiration (UPTGA) was performed in all three of them under strict aspeptic and antisepticconditions. Bile was aspirated and preserved for toxicological analysis. The patients improved both symptomatically and biochemically after the procedure. No complications were noted in these patients and they were discharged after a few days of observation in our hospital. We conclude that ultrasound guided percutaneous transhepatic Gall bladder Aspiration (PTGBA) is a simple bedside procedure that can be safely done in high risk patients of amanita mushroom poisoning.

INTRODUCTION

Mushroom poisonings(Mycetismus) are rare conditions, mostly reported from specific geographic areas. Poisonous varieties of mushrooms are numbered approximately 100 among the more than 5000 species, out of which only about 32 species have been associated with fatalities.[1] At least 300 species of mushrooms have been reported from India.[2]The poisons can be broadly divided into protoplasmic poisons, neurotoxins, gastrointestinal irritants and alcohol dehydrogenase inhibitors. The protoplasmic and gastrointestinal poisons are most commonly encountered.Greater than 90% of cases of fatal poisoning are caused by ***Amanita phylloides***(death cap) or ***Amanita verna*** (destroying angle).Mushroom poisonings have been reported frequently from North East India during the monsoon seasons.[3,4]Sharma TC et

al,[5] has noted that in North-eastern India mushrooms are highly coveted item of food but the knowledge of edible mushroom in Assam is confined only to the ethnic tribes of the state. Still mushroom poisoning cases are reported during the monsoon seasons in young and old patients of various socioeconomic and ethnic groups of people.There are no universally accepted guidelines to treat mushroom poisoning, though various medications have been tried. Enjalbert F et al,[6] in 2002 reviewed 2108 hospitalized amatoxin poisoning exposures as reported in the medical literature from North America and Europe over the last 20 years and concluded that benzylpenicillin (Penicillin G) alone and in association was the most frequently utilized chemotherapy. But this treatment showed little efficacy. Thioctic acid and steroids showed no benefit. Silybin, administered either as mono-chemotherapy or in drug combination and N-acetylcysteine as mono-chemotherapy may have some benificial role. The mortality in mushroom poisoning varies from region to region and is mostly dependent on the type of mushroom ingested, facilities available like liver transplant and critical care management. Though there have been very few mushroom poisoning published in literature from India, most have shown high mortality. In one of the earlier studies we noted a high mortality (43.75%) even after using all these agents.[3] Das AK noted that we needed our own treatment protocol for mushroom poisoning patients.[7]Studies in dogs have shown that the toxins are excreted in the bile, which leads to cellular damage to the duodenum.[8] Much of the toxin excreted in this way is reabsorbed into the liver with the bile. Interruption of the cycling of the toxin by bile duct cannulation can reduce the toxic effects of α-amantin in dogs (Fauser and Faulstich 1973).[9]We have shown in our previous publications that elevated transaminases level in blood and prothrombin time are high predictors of mortality in mushroom poisoning cases in upper Assam. [10,11]

Mitchell ST,Deori PK and Long C have reported drainage of the gall-bladder in six Assamese patients of mushroom poisoning undergoing surgical open cholecystostomy.[12,13] Mitchell ST and Deori PK had communicated their findings with Deepjyoti Payeng in Assam Medical College, who took the initiative of performing this procedure in some of the selected ill patients of Mushroom poisoning. Mitchell ST recommended performing simple ultrasound-guided gallbladder aspiration rather than cholecystostomy, as risks of bile peritonitis, bleeding, and infection associated with previous were expected to be lower. So we report a case series where this novel technique was performed in three patients who presented with acute liver failure following mushroom poisoning.

Technique of Percutaneous transhepatic Gall bladder Aspiration (PTGBA): Under all antiseptic precautionunder local anaesthesia the procedure were performed. After local site injection of 10 ml of lignocaine, 18-20GVygon needle was inserted under ultrasound guidance into the body of distended gall bladder across the normal hepatic parenchyma and avoiding direct puncture of Gall bladder wall. Gradually the stellate of the Vygon needle was removed and 20cc disposable syringe was attached into the needle. Then gradually bile was aspirated from gall bladder until gall bladder gets contracted on USG or non-aspirable. This aspiration technique was repeated every alternate day preferably upto 7 days on basis of clinical outcome or clinical improvement.

Case 1

A 58 year old male, was admitted in our hospital with altered behaviour, 78 hours after ingestion of a bowl full of cooked wild mushrooms with rice and other cooked vegetables. His wife (46 years) and daughter (24 years) also ingested the same. He and his daughter were referred to our tertiary centre after his wife died in a primary care hospital where they were initially admitted. On taking proper history he said that his first symptom was nausea and vomiting, 38 hours after ingesting the mushroom. His first symptoms were diarrhoea and pain abdomen. He developed jaundice on fourth (4^{th}) day following the poisoning. Upon admission to our tertiary care hospital he was started on intravenous fluids (Normal saline and Ringers Lactate), Syrup Silymarin, Vitamin K and Tablet N-acetylcysteine 600 mg as per institutional protocol. His serum bilirubin was 4.1 mg/dl (conjugated 2.75 mg/dl). His aspartate aminotransferase (AST) was 3055 IU/dl , alanine aminotransferase (ALT) was 4363 IU/dl, Alkaline Phosphatase (ALP) was 232 IU/dl, serum Gamma-glutamyltranspeptidase(GGT) level was 82 IU/dl. High AST and ALT is a strong indicator of mortality.[10]His Prothrombin time was 14.1 and INR was 1.23.He was diagnosed with acute liver failure with grade III Hepatic Encephalopathy (HE).As hepatic transplant was no feasible option in our setting, we had thealternative option of Ultrasound guided percutaneous transhepaticneedle aspiration of Gall bladder, the idea being removal of the toxin in the bile and preventing its entero-hepatic recycling and re-

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absorption. After full informed consent including risks of biliary peritonitis and other complications he was taken up for the procedure of PTGBA following which he improved clinically. This procedure was repeated for a second time on third post procedure day. His biochemical parameters and clinical condition, both improved significantly (Table 1). After supportive treatment for another week he was discharged from our hospital without any complication.

Case 2

28 year old male, a tea garden labour attended our tertiary care centrefour (4) days after ingestion of few spoon full mushroom cooked with vegetables taken with one family member. He presented with diarrhoea and pain abdomen on third day of ingestion of mushrooms. On admission he was dehydrated and icteric. He was having confusion and was diagnosed with grade II HE.His hemoglobin 10.3 g/dl, Total count was 5,600/cmm. ESR was 23 mm AEFH. His serum bilirubin was 5.98 mg/dl (conjugated 0.5 mg/dl). His AST was 1175 IU/dl, alanine ALT was 2250 IU/dl ALP was 141 IU/dlGGT was 172 IU/dl. His Prothrombin time was 12 and INR was 1.04. He was intravenous fluids (Normal saline and Ringers Lactate), Syrup Silymarin, Vitamin K and Tablet N-acetylcysteine 600 mg.PTGBAwas done with full informed consent on 5th day post mushroom poisoning. Following this procedure his condition improved without any complication and he was discharged within one week's time. He came for review check up after one month and we found him in complete good health with no complications.

Case 3

A 24 year old male was referred to our institute on second day of ingestion of bowlful mushroom cooked per boiled with three other family members, two of whom died before reaching our hospital.He presented within 24 hours of ingestion of the mushroom. His Bilirubin was 3.23 mgl/d (conjugated 1.67mg/dl),ASTwas 461 IU/dl ,ALT was 699 IU/dl,ALP was 196 IU/dl, GGT was 126 IU/dl. His Prothrombin time was 15.3 and INR was 1.33. With two of his four family members dead due to mushroom poisoning we offered him the option of PTGBA. His biochemical parameters improved significantly after the procedure. He was discharge after seven days of observation when he recovered clinically.

DISCUSSION

Ganzert et al,[14] retrospectively analyzed the outcome of a large series of amatoxin intoxication cases and found that predictors of deathwere the prothrombin index in combination with the serum creatinine level on 3–10 days after ingestion.Passo B et al,[15] and Teutsch C et al,[16] both described that liver transplantation improve mortality in amatoxin-induced acute liver failure in selected patients and before development of grade 4 hepatic encephalopathy. But access to liver transplant is trivial, more so in developing countries. Overall, the prognosis of amanitin induced acute liver failure remains quite poor. Mitchell T,[12,13] noted that drainage of the gall-bladder, in combination with sustained aggressive IV hydration, appears to be a promising treatment alternative for patients with amatoxin mushrooms poisoning in developing countries where injectable Silibin is not available. He reported six patients from rural India who underwent open surgical cholecystectomy and one American patient who underwent percutaneous cholecystectomy. Alpha-amantin(HPLC) content in bile samples from India ranged from 3.06 to 11.67 mcg/ml where as it was 22.3 mcg/ml in day 0 (day of ingestion) sample of the American patient. He also commented that simple gall-bladder aspiration is a much simple method than open surgical or percutaneous cholecystectomy, with minimal side effects and complications. In our cases, we have shown that PTGBA can be safely performed in patients with high risk of mortality from amatoxin mushroom poisoning. Madhok M er al,[17]had also noted that nasobiliary drainage by endoscopic cholangiography (ERCP) has been successfully used to remove amatoxins from enterohepatic circulation but is not preferred routinely.ERCP is technically challenging and may not be widely available in developing countries.We have shown that Percutaneous transhepatic Gall bladder Aspiration (PTGBA) was successfully used in three patients of amanita mushroom poisoning who had high risk of mortality.[10] PTGBAhadexcellent results and no major complication. This procedure may be considered in the management of seriously ill mushroom poisoning patients when standard medical therapy fails to show improvement and the option of endoscopic cholangiography (ERCP) or liver transplant seems trivial.

CONCLUSION

Percutaneous transhepatic Gall bladder Aspiration (PTGBA) is a simple bedside procedure that can be safely done in high risk patients of amanita mushroom poisoning.

 ISSN: 0973-3558, e-ISSN: 0973-3566

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 DOI: 10.31736/jist/v14.i1.2018.40-44

Table 1: Clinical and laboratory parameters of the patients with mushroom poisoning.

Parameters	CASE 1 (58y/M)			CASE 2 (28y/M)		CASE 3 (24 y/M)	
	Before PTGBA	**After first PTGBA**	**After second PTGBA**	**Before PTGBA**	**After PTBGA**	**Before PTGBA**	**After PTBGA**
1. **Onset of symptoms after consumption of Mushroom**	38 hours	_____	_____	48 hours	_____	32 hours	_____
2. **Hospitalization to tertiary center after consumption of Mushroom**	84 hours	_____	_____	76 hours	_____	38 hours	_____
3. **Initial symptoms**	Pain abdomen, vomiting , diarrhea	_____	_____	Pain Abdomen, diarrhoea	_____	pain abdomen, diarrhoea	_____
4. **Progression of symptoms**	Jaundice , altered consciousness	Jaundice, senorium improved	Improved	Dehydrated, Jaundice	Improved	Dehydrated	Improved
5. **Hepatic encephalopathy**	Grade III, Type A HE	Grade I	Absent	Grade II, Type A HE	Absent	Absent	Absent
6. **Hb%**	9.3 mg/dl	9 mg/dl	8.9 mg/dl	10.3 mg/dl	10 mg/dl	8.6 mg/dl	8.6 mg/dl
7. **TLC**	4,600/cumm	4,800/ cumm	4,800/ cumm	5,600/cumm	5,500/ cumm	4,400/ cumm	5,200/ cumm
8. **ALT**	4363 IU	968 IU	734 IU	2250 IU	808 IU	699 IU	61 IU
9. **AST**	3055 IU	321 IU	195 IU	1175 IU	122 IU	461 IU	37 IU
10. **Bilirubin**	4.42 mg/dl	4.11 mg/dl	3.42 mg/dl	5.98 mg/dl	2.09 mg/ dl	3.23 mg/dl	1.68 mg/dl
11. **Prothrombin Time /INR**	14.1/1.23	-------	-------	12/1.04	---------	15.3/1.33	---------
12. **Alkaline phosphatase**	252 IU	237 IU	185 IU	141 IU	106 IU	196 IU	99 IU
13. **GGTP**	82 IU	120 IU	110 IU	172 IU	140 IU	126 IU	30 IU
14. **Serum Creatinine**	1.2 mg/dl	0.9 mg/dl	0.9 mg/dl	1.2 mg/dl	0.7 mg/dl	1.1 mg/dl	0.6 mg/dl
15. **Serum albumin/ globulin**	2.6/2.7 g/dl	2.7/3.2 g/dl	2.4/3.3 g/dl	3.6/3.8 g/dl	3.4/3.5 g/dl	4.2/3.2 g/dl	3.9/3.6 g/dl
16. **USG findings**	Hepatomegaly with periportal cuffing and edematous GB walls.		Normal	Hepatomegaly with periportal cuffing and edematous GB walls.	Normal	Hepatomegaly	Normal
17. **Medical management**	Intravenous Fluids, Silymarin, Vit K, N-acetylcysteine 600mg			Intravenous Fluids, Silymarin, Vit K, N-acetylcysteine 600mg		Intravenous Fluids, Silymarin, Vit K, N-acetylcysteine 600mg	
18. **Radiological Management**	PTGBA	2nd PTGBA	_____	PTGBA	_____	PTGBA	_____
19. **Final outcome**	_____	Improved	Survived	_____	Survived	_____	Survived

 ISSN: 0973-3558, e-ISSN: 0973-3566