"MUSHROOM" POISONING DUE TO *AMANITA PHALLOIDES*

BY

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Edible fungi have been a favourite culinary delicacy for centuries. Mushrooms were recognized as a valuable form of food by the ancient Babylonians and early Romans, who employed them in large quantities not only as a delicacy for the tables of the rich but also as a staple article of diet of the poorer classes.

Although the ancients were possessed of considerable knowledge regarding edible fungi and poisonous varieties were well recognized, many fatalities must have occurred. Records of death from fungus poisoning, often amongst eminent people, figure prominently in contemporary writings, especially those of Pliny; but the earliest record of fatal poisoning dates back over two thousand years. In the fifth century B.C., when Euripides the poet was at Icarus, a woman with her two grown-up sons and a married daughter "were destroyed by pitiless fate in one day" after eating fungi gathered from the fields. In this instance it is probable that *Amanita phalloides* (Death Cap) was the cause of the tragedy; for besides being the most poisonous fungus known it is responsible for more than 90% of all deaths due to mushroom poisoning and is attended by a mortality rate of from 50 to 70% (Ford, 1923). Ramsbottom (1945) states that a death rate as high as 90% has been claimed. Although this percentage is unduly high as an overall figure, there are many examples of a 100% fatality rate in particular groups in which all members partaking of a meal have succumbed (Ford, 1923).

Mushroom poisoning is a commonplace on the European continent, while 200 fatal cases are said to occur annually in Germany alone. In this country but few cases have been reported in the literature. Thus Dubash and Teare (1946), recording four fatal cases, could trace only 21 reported cases of mushroom poisoning with 14 deaths in Great Britain between 1930 and 1942. Birch (1946), quoting from the Registrar General's record, could find only 38 cases of fatal fungus poisoning in England and Wales between 1920 and 1945.

As the time of year is now upon us when *A. phalloides* will again be abundant in the woods and adjoining pastures, and when many continentals are still in our midst, two further cases of Death Cap poisoning seem worthy of report.

The Present Cases

Two German prisoners of war—H. W., aged 29, and J. W., aged 30—were admitted to an R.A.F. hospital on the morning of Sept. 13, 1946, complaining of intermittent seizures of cramp-like abdominal pain accompanied by severe attacks of vomiting and violent purgings. They gave the following story: On the morning of Sept. 11 they had gathered "mushrooms" from a near-by wood; and the next day, after preparation, the fungi were boiled in water for three-quarters of an hour before being fried. The failure of the cooking "mushrooms" to blacken a silver coin was considered proof that the meal would be safe; and each man ate approximately half a pound of mixed fungi. They found the meal most palatable and did not complain of ill effects until nine and twelve hours later respectively. In both cases initial symptoms consisted of severe vomiting, soon to be followed by intermittent attacks of griping upper abdominal pain and diarrhoea. Symptoms continued throughout the night, and on admission to hospital on Sept. 13, eighteen hours after ingestion, diarrhoea and vomiting had taken place fifteen times in H. W. and ten times in J. W.

The patients were first seen personally on Sept. 14, the day after admission. Diarrhoea with vomiting had occurred almost

two-hourly in J. W. Fluids by mouth and an intestinal sedative had been prescribed. On questioning the patients the history of ingestion of "mushrooms" followed by a silent period of many hours before the onset of symptoms made a diagnosis of poisoning by *A. phalloides* virtually certain. Treatment was therefore conducted along these lines without waiting for more definite proof. Additional evidence, however, was soon furnished by the patients themselves. One volunteered that the types of fungus they had gathered were chiefly those with large bulbous stems surrounded in their lower parts by a membrane or volva (see Fig.)—characteristics typical of the *Amanita* group. They

Photograph of specimens of *A. phalloides* at different stages of growth. Note the well-marked volva at the base of the stem, the faint dark (green) striations on the surface of the cap, and the dead-white gill-plates seen in the oldest specimen.

also identified a specimen picked by a fellow prisoner of war (who had also partaken of a small quantity of the mushrooms but had been unaffected) as similar to some of the fungi they had eaten. This specimen proved to be *A. phalloides*. Later a personal visit to the wood accompanied by a colleague with sound botanical knowledge showed that in an unselected picking from the area concerned *A. phalloides* constituted about 10% of the fungi collected. *A. rubescens* (Warty Caps), a non-poisonous variety, were plentiful; but no common mushrooms were identified. It was roughly estimated that in half a pound of uncooked fungi each man had probably eaten one or at most two specimens of *A. phalloides*.

Clinical Examination

Seen on Sept. 14, the second day, both patients, of good physique and development, were exhausted and somewhat dehydrated, especially H. W., in whom symptoms had begun earlier and were more severe. Thirst was marked. Mental aberrations or nervous involvement—a muscarine-like effect that is sometimes observed—were not a feature in either case, except that H. W. was a little disorientated and excitable, although able to co-operate and give a rational history. There was no sign of jaundice or haemorrhagic tendency in either case, while lacrimation, sweating, and salivation were absent. The tongue was dry and coated and the breath heavy. The skin and especially the extremities felt cold, but were without lividity or cyanosis; the temperature was subnormal and the pulse small and rapid. On abdominal examination H. W. complained of tenderness under the right costal margin; in neither case was the liver or spleen palpable. No abnormal physical signs were found in the nervous system, and the rest of the examination was essentially negative. The urine, normal to routine tests, was small in amount and concentrated. Vomitus and excreta contained little solid matter, considerable mucus, but no macroscopic blood in the early stages.

Treatment and Management

All efforts to obtain supplies of antiphallenic serum proved unsuccessful. Gastric and high colonic lavage with 1% saline solution was performed three times on the day after admission

and daily for the next three days. On each occasion lavage was continued until the return was clear. This involved as much as three gallons of fluid during the early part of treatment. Two ounces (57 cc.) of 50% magnesium sulphate were left in the stomach. The effect of lavage produced an immediate and striking diminution of vomiting and diarrhoea, which ceased in the case of J. W. after the first treatment.

On Sept. 14, 5% glucose in normal saline, four to five litres in 24 hours, intravenously, was begun; this was continued for four days, together with 15 units of soluble insulin and 10 mg. of vitamin K four-hourly in an attempt to prevent or minimize liver damage, which was fully anticipated. Apart from ice to suck, all fluid requirements were met by parenteral administration. Atropine 1/100 gr. (0.65 mg.) and morphine 1/4 gr. (16 mg.) were prescribed to control restlessness and pain during the first three days.

On the sixth day unlimited glucose drinks and small quantities of citrated milk were substituted for the intravenous drip, but four-hourly insulin injections, combined with vitamin K and vitamin B₁, 2,000 units and 10 ml. of 10% solution of calcium gluconate parenterally, were continued for a further forty-eight hours. A light diet rich in protein and carbohydrate was then prescribed and steadily increased until discharge from hospital on Oct. 21, when the patients were taking a normal diet.

Clinical Course of Case 1 (H. W.)

Although gastric and colonic lavage decreased the frequency and severity of vomiting and diarrhoea, both persisted until the evening of the third day, when these symptoms gave way to complaints of upper abdominal pain. At this time the patient was unduly drowsy and lethargic. Examination revealed tenderness under the right costal margin, but the liver was not found to be enlarged. On the fourth day definite jaundice of the sclerotics was noted, but the skin was normal in colour. The urine, small in amount, was dark amber and concentrated. The patient, however, felt much better; drowsiness was less marked and he was more alert; nor was there any complaint of abdominal pain or sign of liver enlargement. Clinical improvement from this point was rapid despite an intermittent pyrexia, which reached 102° F. (38.9° C.) on Sept. 17 and persisted with gradual defervescence until Sept. 27. All signs of jaundice had cleared by Sept. 21, and urinary output, which had previously been unsatisfactory, rapidly improved.

Laboratory Investigations.—Sept. 14:—Urine: N.A.D. Sept. 17:—Blood: Hb, 94% (Sahli); R.B.C., 4,280,000; W.B.C., 5,400 per c.mm. (polymorphs 77%, lymphocytes 20%, monocytes 3%). Urine: Urobilinogen +; albumin and sugar, nil; no deposit. Sept. 18:—Total plasma proteins: 5.5 g. per 100 ml.; albumin-globulin ratio, 1.6:1. Oct. 2:—Electrocardiogram: Physiological; no evidence of bundle-branch block (a feature reported by Hyman, 1928). Oct. 4:—B.S.R.: 35 mm. in first hour (Westergren). Oct. 5:—Total plasma proteins: 6.5 g. per 100 ml.; albumin-globulin ratio, 5:1. Oct. 7:—Serum bilirubin: Less than 0.5 mg. per 100 ml. Oct. 17:—B.S.R.: 14 mm. in first hour (Westergren).

Clinical Course of Case 2 (J. W.)

On the second day, after the first gastric and colonic lavage, for which three gallons (13.64 litres) were required, the patient felt more comfortable, while vomiting and diarrhoea ceased completely from this time. The patient still complained of pain under the right costal margin, but there was no jaundice or evidence of hepatic enlargement.

The condition appeared to have improved on the third day despite pyrexia to 101.6° F. (38.7° C.). Apart from superficial ulcers of lips and tongue there were no complaints, and the patient felt much better. Clinical examination was negative. Anuria, however, persisted throughout the day, and in the evening his condition rapidly deteriorated. He complained of generalized aching pain of sudden onset and severe right-sided upper abdominal pain, requiring morphine for relief. A bowel action, consisting of a very pale stool, was accompanied by passage of a few ounces of dark amber urine.

On the fourth day, after a noisy restless night when morphine and atropine had to be administered, the patient became more restless and very drowsy, although rousable. His chief complaint was severe abdominal pain. Jaundice of sclerotics and skin, although not deep, was obvious, and pruritus was troublesome. No haemorrhage or petechiae were seen. Abdominal examination revealed extreme tenderness in the epigastrium and under the right costal margin. The liver was enlarged one to two fingerbreadths; the spleen was not palpable. Temperature was 100° F. (37.8° C.); pulse, 64 a minute, and regular.

Restlessness, with sleep for brief periods, occurred throughout the next night; but by the fifth day his condition showed some improvement. Jaundice and hepatic enlargement were unchanged, but abdominal pain and tenderness were less. Examination of the lungs showed coarse basal crepitations, which persisted for several days; there were no respiratory symptoms. Following this acute attack the patient's condition steadily improved, but more slowly than in Case 1. Jaundice, never deep, persisted together with hepatic enlargement until Sept. 28 (16th day), when he was free from all symptoms and taking a light diet.

Laboratory Investigations.—Sept. 17: Urine: S.G. 1020; urobilinogen +; albumin +; sugar, nil; deposit—leucocytes with granular debris. Plasma proteins: Total 5.2 g. per 100 ml.; albumin-globulin ratio, 1.8:1. Blood: Hb, 101% (Sahli); R.B.C., 4,820,000; W.B.C., 5,000 per c.mm. (polymorphs 71%, lymphocytes 19%, monocytes 10%). Sept. 18:—Serum bilirubin: 1.25 mg. per 100 ml. Oct. 2:—Electrocardiogram: Physiological. Oct. 8:—Plasma proteins: Total, 6.6 g. per 100 ml.; albumin-globulin ratio, 4:1. Serum bilirubin: 0.5 mg. per 100 ml.

Both patients were confined to bed for three weeks from the time of admission to hospital; they returned to duty on Oct. 21.

Discussion

Amanita phalloides produces two main active principles—phallin and amanita toxin. The former, first described by Kobert (1891), is an alcohol-insoluble haemolysin. It is relatively unimportant in cases of poisoning, for, being an easily decomposed glucoside, it is rapidly destroyed by heat and by digestive juices. Amanita toxin, the essential poison, is an alcohol-soluble indole or aromatic phenol derivative. It is resistant to heat, to drying, and to digestive juices, and in experimental animals it produces most of the lesions which are described in fatal cases of *A. phalloides* poisoning in man (Ford, 1908, 1923).

The susceptibility of different individuals to the amanita toxin varies, but surprisingly small amounts of the fungus and even a portion of a single specimen may prove fatal, especially when eaten raw (Plowright, 1879). The case histories and course of the illness reported above indicate that

Table showing the comparative relationship of early and delayed symptoms and signs

Case	Period of Gastro-intestinal Symptoms			Period of Delayed Hepato-renal Damage						
	Silent Period (Hours)	Severity of D. and V.	Total Length of Symptoms (Hours)	Abdominal Pain and Tenderness	Duration of Jaundice (Days)	Pruritus	Liver Enlargement	Urine	Serum Bilirubin	Total Plasma Proteins. Alb./Glob. Ratio
1	9	+++ 15 attacks before admission	69	+	Slight (6 days)	Nil	Nil	Urobilin. +; albumin, nil; no deposit	—	Sept. 18: 5.5 g.; ratio 1.6:1 Oct. 5: 6.2 g.; ratio 5:1
2	12	++ 10 attacks before admission	38	+++	+(13 days)	+	1-2 finger-breadths	Urobilin. +; albumin +; polys. and deposit +	1.25 mg./100 ml.	Sept. 17: 5.2 g.; ratio 1.8:1 Oct. 8: 6.6 g.; ratio 4:1

only a small quantity of *A. phalloides* had been eaten by the patients—a contingency made all the more probable by the fact that a third individual partaking of the meal was unaffected. Thermostability of amanita toxin was well demonstrated by its resistance to boiling and frying for almost an hour (at a maximum temperature of about 140° C.); the silver-spoon test recommended by Atkinson (1900) proved a misleading and dangerous criterion by which to judge whether the "mushrooms" were safe or not; and, finally, *A. phalloides* was palatable and even pleasant to eat.

The long delay interval of nine and twelve hours respectively before the onset of symptoms emphasizes the important point made by Birch (1946), that in any case of mushroom poisoning an interval of eight hours or more between ingestion and symptoms should be regarded as diagnostic of *A. phalloides* poisoning. The severity of the gastro-intestinal symptoms in the early stages of the illness contrasted with the surprisingly mild hepato-renal damage which took place later (see Table). The importance of eliminating factors in assisting recovery is indicated by the fact that the patient with the shorter silent interval and the more severe and prolonged diarrhoea and vomiting sustained less liver damage than his colleague, whose condition in the early stages was considered the more satisfactory. In addition, poisoning by *A. phalloides* combined with other species of fungus has a better prognosis than poisoning by *A. phalloides* alone, the reason for this being that other types of fungus often cause earlier diarrhoea (Vander Veer and Farley, 1935).

Summary and Conclusions

Mushroom poisoning due to *Amanita phalloides* in two German prisoners of war is described. Both patients recovered.

The severity of the gastro-intestinal symptoms in the early stages of the illness contrasted with the mildness of the delayed hepato-renal damage.

The delayed effects of poisoning were more severe in the patient with the longer silent period before the onset of symptoms and with the less pronounced and protracted diarrhoea and vomiting.

The ability of the gastro-intestinal tract to eliminate the toxin of *A. phalloides*, and the thoroughness with which this elimination is assisted by early and efficient gastric and colonic lavage, are regarded as factors of the greatest importance in determining recovery.

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TREATMENT OF GENU VALGUM

THE DISCARDED IRON

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The psychological effect, both on child and on parent, of wearing irons is usually unpleasant. There are occasionally exceptional people, like the late President of the United States, who achieve superiority in spite of their disability. That is possibly because the essential necessity for wearing irons has been revealed to them and they have been told that irons are necessary for progression or even existence.

Every orthopaedic surgeon who has served during the war must have experienced a thrill on his return to civilian practice on seeing as a young adult apparently cured a child who had been under his treatment five years previously. He may be labouring in the fields; she may be soignée with lipstick and rouge. Yet if treatment has extended over several years it is always easy to detect in these patients a difference from others. An intangible shyness or barrier is present. It might be called an inferiority complex, but it is a sequel of prolonged immobilization, with which social segregation is invariably associated. This result may be regrettable, but it is tempered by the fact that such treatment has been inescapable.

In quite a different category is the child who has to wear an instrument which proclaims to all that he or she is a "cripple," though in truth such an instrument is merely corrective and not essential. Here, leading all the rest, is the knock-knee iron or brace, an appliance of very doubtful value.

This paper is based on impressions derived from the treatment of 8,000 cases of genu valgum treated over a period of 15 years, and has been prompted by seeing children returned to East Anglia after evacuation to other parts of England where treatment was carried out with knock-knee braces and without appreciable results.

Aetiology

Jones and Lovett (1929) describe knock-knee under the heading of rickets. They, however, stress that rickets is not the only cause of knock-knee, and mention tuberculosis of the knee-joint and infantile paralysis as well as fractures in the neighbourhood of the knee-joint with malposition and epiphyseal injury. "Loose knees" in children are also mentioned, and I wish to emphasize these as being the chief cause. McMurray (1943) says: "This very common deformity begins as a rule in early childhood, following on rickets," and ten lines later on he states: "As a rule, deformity is due to this overgrowth of the inner side of the femur, the alignment and shape of the tibia remaining normal." Mercer (1943) states: "The deformity of knock-knee develops, as a rule, in early childhood, and is almost invariably due to rickets."

Few orthopaedic surgeons now believe that rickets is the commonest cause of knock-knee, yet this claim holds pride of place in the three textbooks mentioned. I think that knock-knee is due to: (1) laxity of ligaments or loose knees, as mentioned by Jones and Lovett (1929); (2) quadriceps insufficiency; and (3) the child being overweight.

It may be argued that if the knee is loose why does genu varum not occur at the knee instead of genu valgum? The femora are much wider apart at the level of the great trochanters, and slope inwards towards the knee, so that the tendency is normally to valgus. Furthermore, the external lateral ligament of the knee is a thick and very strong structure, reinforced by the ilio-tibial band, and

The Ministry of Health has issued two leaflets explaining how to get dental treatment or sight tests and spectacles under the National Health Service. Members of the public can get copies of them from the offices of the executive councils (address from any post office), at local offices of the Ministry of National Insurance, and at various other centres, such as citizens' advice bureaux.