
Anaphylactic Shock during Operation for Hydatid Disease

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In man infection with Echinococcus granulosus or Echinococcus multilocularis produces cystic lesions. These have been found in many organs, but are seen primarily in the liver and secondarily in the lungs. The disease is infrequently noted in the United States, but occasionally it is reported in the western and southwestern parts of the country. With the increase in world travel, however, the disease may be more frequently encountered in visitors from Central Europe, South America, and Africa, as well as immigrants from these areas.  

Nonsurgical treatment has thus far proved unsatisfactory, and several surgical procedures are currently employed for treatment of the disease.  

Rupture of the cyst during operation, with discharge of its contents into the peritoneal cavity, or manipulation of the cyst and seeding of the circulation, may be followed by an anaphylactic reaction. This is caused by the strongly antigenic hydatid fluid and may be fatal.

We report the case of a patient who suffered intraoperative anaphylactic shock secondary to manipulation of an hydatid cyst.

REPORT OF A CASE

A 36-year-old woman, a Greek immigrant, was admitted to the Naval Hospital, Boston, with a complaint of epigastric pain. A past history of ulcerative disease in 1962 was elicited; however, no further exacerbations had occurred. The patient denied any family history of allergy, hepatic disease, or other systemic illness. On physical examination the only positive finding was a palpable mass in the area of the left lobe of the liver. Hemogram was normal except for an eosinophil count of 8 per cent. Chest x-ray was within normal limits. Liver scan revealed a 3-cm filling defect in the liver below the xiphoïd process.

Ten days after admission the patient underwent operation for probable echinococcal cyst of the liver. Premedication consisted of meperidine (Demerol), 50 mg, pentobarbital (Nembutal), 100 mg, and atropine, 0.4 mg, 45 minutes prior to induction of anesthesia. Anesthesia was induced with thiopental (Pentothal), 250 mg intravenously, and the trachea was intubated following succinylcholine (Anectine), 60 mg intravenously. Anesthesia was maintained with nitrous oxide-oxygen in a mixture of 4 to 2 liters, morphine sulfate, and d-tubocurarine.

During the procedure blood-tinged hydatid fluid was aspirated from the cyst, and 95 per cent ethyl alcohol was injected directly into the cyst and allowed to remain in place for eight minutes. Five minutes after aspiration of the cyst, the systolic blood pressure fell from 130 to 100 mm Hg. Within two minutes the pressure fell to 60 mm, systolic. At this time nitrous oxide was discontinued and the lungs were ventilated with 100 per cent oxygen. Within the next five minutes peripheral blood pressure and pulse became unobtainable. Heart sounds, monitored with a precordial stethoscope, continued to be audible, and inspiratory and expiratory wheezes were noted throughout both lung fields, while compliance seemed to decrease.

An anaphylactic reaction was suspected and methylprednisolone (Solu-Medrol), 750 mg, was administered intravenously as a bolus followed by mephenetermine sulfate (Wyamine), 30 mg, and sodium bicarbonate, 44 mEq, to counteract the
expected metabolic acidosis. Mephenetermine was repeated, in the same dose, three times in the following ten minutes. Arterial blood gases at this time were: Po2, 220 torr; Pco2, 30 torr; pH, 7.46.

Approximately 25 minutes after aspiration of the cyst the peripheral pulse and blood pressure became palpable at 60 torr systolic. Methylprednisolone, 750 mg, was repeated and an isoproterenol infusion of 0.2 mg/250 ml of physiologic saline solution was begun.

No attempt was made to excise the cyst, and the abdomen was closed without further manipulation. Upon removal of the surgical drapes, erythema of the upper part of the patient’s body was noted, giving further clinical evidence of an anaphylactic reaction. The patient was taken to the recovery room with an auscultatory blood pressure of 60/40 torr and a pulse rate of 120 beats/min approximately 60 minutes after aspiration of the cyst. She was responsive to voice commands at this time and the bronchospasm had subsided.

Ventilation was maintained with a Bird Mark VII ventilator with 100 per cent oxygen. On arrival in the recovery room, arterial blood gases were: Po2, 210 torr; Pco2, 33 torr; pH, 7.33. Sodium bicarbonate, 44 mEq, was administered in an intravenous infusion of balanced salt solution. After 20 minutes isoproterenol was discontinued, with the patient able to maintain a blood pressure of 130/60 torr. Four hours after the onset of hypotension the endotracheal tube was removed and oxygen given by mask. Urinary output throughout the 1 hour, 45 minutes, procedure was 150 ml, and continued at 75-100 ml/hour for several hours postoperatively. A total of 2,600 ml of balanced salt solution (Ringer’s lactate) had been infused.

The subsequent postoperative course was uneventful, and on the tenth postoperative day the patient underwent partial left hepatic lobectomy. Diphenhydramine (Benadryl), 100 mg, and atropine, 0.6 mg, as well as hydrocortisone (Solu-Cortef), 100 mg, were administered preoperatively intramuscularly, with the addition of diphenhydramine, 100 mg, and hydrocortisone, 200 mg, to the first intravenous infusion of 1,000 ml of Ringer’s lactate solution. Gallamine (Flaxedil) was substituted for d-tubocurarine, and nitrous oxide—oxygen in a mixture of 4 liters to 2 liters was given, as well as morphine sulfate. The operation and postoperative course were uneventful, and the patient was discharged on the tenth postoperative day, 20 days after the original reaction.

**DISCUSSION**

Man, sheep, cattle, and hogs serve as intermediate hosts for the dog tapeworm, *Echinococcus granulosus*. The host becomes infected through ingestion of food or water contaminated with feces from infected animals. The ova are digested in the gastrointestinal tract of the intermediate host, the larval form passing through the intestinal wall to the liver via the portal circulation. If not cleared by the liver, the larvae will be transported to the lungs and possibly the systemic circulation. Within the organ, differentiation into a two-layered cyst occurs. The inner membrane of the cyst produces the scoles and a strongly antigenic hydatid fluid.

Individuals with echinococcal disease may become sensitized to hydatid cyst fluid. The antigenicity of the fluid is not dependent upon a viable cyst but, in fact, remains for years, even after the death of a cyst. This property provides a basis for the Casoli intradermal skin test, one of the few reliable methods of diagnosing echinococcal disease in man. Positive reactions occur in 70 to 92 per cent of individuals with the disease.

In sensitized individuals, spontaneous leakage of fluid through an incompetent cyst wall or surgical rupture may cause allergic phenomena. These vary from mild pruritus and urticaria to anaphylactic shock, convulsions and coma.

Although the literature on echinococcal disease mentions anaphylactic shock as a complication, few reviews note the incidence of such reactions in general, or the incidence during surgery. Sharma reports one reaction among 33 patients. In reviewing 90 cases of echinococcal disease at the Mayo Clinic prior to 1960, Hoffman and Judd each report one case of anaphylaxis. Jidejian observed a single anaphylactic shock in 178 patients operated upon for echinococcal cysts of the liver; however, the incidence of rupture during surgery was not noted. In 1966, Schiller reported one death from probable anaphylaxis among 30 patients with surgically ruptured cysts of the liver.

It is possible that precautions to avoid spillage of cystic fluid during operation for hydatid disease account for the apparent low incidence of anaphylactic shock intraoperatively. However, the anesthesiologist should be aware of the possibility of this complication. Treatment must be instituted immediately if survival is to be expected. The immediate concern is to restore both circulatory competency.
and a satisfactory cardiac output. Control of the airway with delivery of 100 per cent oxygen and a vasopressor with alpha- as well as beta-stimulating properties is probably most useful under the conditions of massive peripheral vasodilation and bronchospasm which occur in anaphylaxis. In our case mephen- termine was used initially, with effect. In the face of developing bronchospasm and concern over possible tachyphylaxis to mephenetamine, an infusion of isoproterenol was started. Circulation continued to improve. It might be argued that an infusion of epinephrine would have been more appropriate at that time.

The role of a steroid in alleviating the anaphylactic reaction is not clear. The effect, if any, on the antigen–antibody reaction is not known. However, improvement is seen when steroids are administered to asthmatics, and some authors advocate their use in other allergic types of reactions. Antihistamines will not reverse the effects of an acute anaphylactic reaction, but may be useful in preventing further histamine binding. For this reason they are useful prophylactically if an allergic reaction is anticipated.

Desensitization methods have not proven practical in treating hydatid disease. In fact, an anaphylactic reaction may be precipitated by an intradermal injection of hydatid fluid. The physician should be aware of this complication and be prepared for treatment.

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REFERENCES


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RESPIRATORY DISTRESS SYNDROME Prenatal hypoxia and pulmonary hypoperfusion are thought to damage the alveolar epithelial cells and stop temporarily the production of surfactant. Since the surfactant stores are small in the lungs of a premature baby, the surface tension of the alveolar film increases and alveolar collapse occurs. In the author's hospital, the mortality from RDS was 52 per cent in 1964 and was decreased to 23 per cent in 1968. The improved results were probably due to maintenance of PaO₂ 70-90 mm Hg with avoidance of hypo- and hyperthermia and correction of any developing acidosis. Sodium bicarbonate or THAM was used to treat metabolic acidosis. Artificial respiration was used only when hypoxia or hypercarbia persisted. (Weisser, K: The Idiopathic Respiratory Distress Syndrome in Premature Neonates, Der Anaesthesist 19: 101 (March) 1970.)