Anaphylaxis due to vaccination in the office

Howard Ovens, MD, CCFP (EM)

In their offices physicians may inject a variety of substances, most commonly vaccines, toxoids and desensitizing solutions of allergens, and occasionally medications. Any injected substance may provoke sudden, life-threatening anaphylaxis, which the physician must be prepared to deal with.

Case report

A 32-year-old woman attended her family doctor's office for vaccination against diphtheria, poliomyelitis and tetanus (DPT). She denied prior vaccination and allergic reactions.

Seconds after the intramuscular injection of 0.5 mL of adsorbed DPT vaccine the patient complained of shortness of breath. Within 5 minutes urticaria and hypotension were evident. When 0.2 mL of a 1:1000 dilution of epinephrine administered subcutaneously had little effect an ambulance was called.

The patient arrived at our hospital 17 minutes after the injection. She was conscious but unable to speak. There was stridor, bronchospasm, tachycardia and marked hypotension (systolic blood pressure 70 mm Hg). Oxygen inhalation by mask was begun, an intravenous line and fluid administration were established, and a cardiac monitor was placed. After 5 mL of a 1:10 000 dilution of epinephrine was given by slow intravenous infusion 50 mg of diphenhydramine hydrochloride (Benadryl), 500 mg of hydrocortisone sodium succinate (Solu-Cortef) and a crystalloid fluid bolus were administered intravenously. Simultaneously the patient inhaled salbutamol from a nebulizer. A cricothyroidotomy tray was kept at the bedside in case of complete airway obstruction.

For about 5 minutes the patient experienced brief rebound hypertension and ventricular arrhythmias (premature contractions in couplets and triplets), likely secondary to the intravenously administered epinephrine. In 20 minutes her vital signs were stable and she was able to speak. She was observed in the holding area of the Emergency Department for 24 hours and received corticosteroids orally for 48 hours. There were no long-term adverse effects.

From the Emergency Department, Mount Sinai Hospital, Toronto

Correspondence to: Dr. Howard Ovens, Emergency Department, Mount Sinai Hospital, 600 University Ave., Toronto, Ont. M5G 1X5 *(reprints not available)*

Discussion

Although the patient denied it, she might have been vaccinated in the past. Thus, sensitization to any component of DPT vaccine, including the adjuvant, cannot be ruled out. Obviously rechallenge to identify the responsible agent is contraindicated.

Severe, systemic reactions to DPT vaccine like the one I have described are rare, occurring perhaps once in 100 000 injections.[1] This was the first reported case in Canada due to adsorbed vaccine (Health Protection Branch, Department of National Health and Welfare: personal communication).

Adsorbed vaccines for intramuscular use were released in Ontario in the autumn of 1984.[2] Although these vaccines were reported to induce a superior antibody response and to less frequently have adverse effects compared with the older, fluid preparations,[2,3] practising physicians have remained sceptical.[4,5] The low frequency of serious reactions is reassuring, but it also renders safety comparisons before product release difficult and emphasizes the importance of physician compliance with voluntary reporting programs.

More important, physicians who even occasionally inject substances of any kind in their offices must remember that anaphylaxis can occur suddenly, without warning. Patients should therefore be observed for at least 20 minutes after any injection. If a nurse gives the injection the physician must be present in the office.

If urticaria, dyspnea, angioedema or hypotension develops after an injection 0.3 to 0.6 mL of a 1:1000 solution of epinephrine should be administered subcutaneously every 5 minutes as required. If vascular collapse occurs, slow intravenous administration of 5 to 10 mL of a 1:10 000 solution may be preferred[6] and should be followed by intravenous administration of an antihistamine (such as diphenhydramine, 25 to 50 mg) and a

corticosteroid. Transfer to hospital should be arranged immediately and oxygens and fluids given as soon as possible. Salbutamol may be helpful if bronchospasm is prominent. In severe cases pressor agents may be required to support the blood pressure, and surgical creation of an airway (via a cricothyroidotomy) may be necessary if laryngeal obstruction occurs. If the office is remote from any hospital the physician must be prepared to perform the entire resuscitation. The patient should be observed for 12 to 24 hours in case of delayed reactions,[6] but oral steroid therapy for 48 to 72 hours (e.g., 8 to 12 doses of prednisone, 10 mg, every 6 hours) should lessen the risk.

The physician's office supply of drugs and syringes should be kept up to date and readily available (as in a red emergency box). The staff should be educated on the need to observe patients for at least 20 minutes after all injections and on the early recognition of anaphylaxis. All adverse reactions to injections should be reported.

References

1. Pollock TM, Morris J: A 7-year survey of disorders attributed to vaccination in North West Thames region. *Lancet* 1983; 1: 753–757
2. Gold R: Adsorbed vaccines: a change for the better. *Ont Med Rev* 1984; 51: 268–269
3. Mathias RG: Reactogenicity of fluid compared with adsorbed diphtheria–pertussis–tetanus vaccine. *Can Med Assoc J* 1984; 130: 1561–1565
4. Lewin PK: Non-adsorbed vaccine as an option [C]. *Ont Med Rev* 1984; 51: 605
5. Dickson RC: Intra-muscular vaccines [C]. *Ont Med Rev* 1985; 52: 202
6. De Shazo RD, Salvaggio JE: Anaphylactic emergencies: update on a changing picture. *Mod Med Can* 1983; 38: 923–926

Acute hydrocephalus and eclampsia

J. Hugh Devitt, MD, MSc, FRCPC
Thomas W. Noseworthy, MD, MSc, FRCPC, FACP
Allan Shustack, MD, FRCPC
Christopher F. Hoskins, MD, FRCSC
Kenneth C. Petruk, MD, FRCSC

Most of our information on the neurologic complications of eclampsia comes from postmortem studies.[1] However, computed tomography (CT) has been reported to aid in the diagnosis of such neurologic sequelae of eclampsia as cerebral edema, intracerebral hemorrhage and multiple white matter infarctions.[2–6] We report a case of eclampsia with neurologic deterioration in which CT scans showed acute hydrocephalus resulting from brain edema.

Case report

A 20-year-old white primigravid woman was transferred to the obstetric service at 36 weeks' gestation for management of pre-eclampsia and intrauterine growth retardation. She had no history of neurologic or other medical problems or of operations.

She was alert and oriented. There was no hyperreflexia or peripheral edema. The blood pressure was 180/110 mm Hg at the time of admission and 170/100 mm Hg 6 hours later. Urinalysis showed a moderate amount of protein. The results of liver function tests and the platelet count were normal.

Initially bed rest and phenobarbital were prescribed. The fetal heart rate was judged to be reactive (i.e., to show normal variability) without stress testing.

Because of continued hypertension magnesium sulfate was administered in a loading dose of 4 g and then infused at a rate of 1 g/h, and labour was induced with synthetic oxytocin and artificial rupture of the membranes. The fetal heart rate was monitored. Eleven hours later a living infant weighing 1340 g with Apgar scores of 3 and 8 at 1 and 5 minutes was delivered per vaginam with the aid of local anesthesia. The mother's blood pressure was 160/100 mm Hg post partum. The magnesium sulfate infusion was continued and the intravenous administration of fluids restricted.

The patient complained of headache and be-

From the departments of Adult Intensive Care, Obstetrics and Neurosurgery, Royal Alexandra Hospital and the Faculty of Medicine, University of Alberta, Edmonton

Reprint requests to: Dr. Thomas W. Noseworthy, Intensive Care Unit, Royal Alexandra Hospital, 10240 Kingsway Ave., Edmonton, Alta. T5H 3V9