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Crit Care Clin 21 (2005) 815–824

CRITICAL
CARE
CLINICS

Ricin

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Ricin, a potent toxin contained in the beans of the castor plant (*Ricinus communis*), has a colorful history. The plant originated in Asia and Africa and can now be found in all temperate and subtropical regions [1]. The innocuous appearing castor plant grows wild across the southwestern United States and is cultivated for decorative purposes [2]. Within its black and brown seeds is the glycoprotein ricin, which makes up 1% to 5% of the weight of the castor bean [3,4]. Another extract from the beans, castor oil, is non-toxic, contains no ricin, and has been used for centuries as a laxative, lubricating oil (eg, Castrol-R racing motor oil), and paint and varnish additive [1,2,4]. Castor oil was used by the military as an aircraft lubricant from World War I though the 1960s [2].

Although ricin was first perceived as a biological warfare agent during World War I, it has more recently been identified as an agent that may be used by terrorists against civilians [1,2,5]. Ricin is one of the most potent plant toxins known [2]. It is much easier to produce than other biological agents, such as anthrax or botulinum toxin [2,4]; production requires only basic techniques that are taught in undergraduate-level chemistry classes [6,7]. The potency of ricin, and the ease with which it can be produced, likely explain why it is frequently associated with extremist individuals and terrorist organizations [3].

In the 1920s, several nations, including the United States, began research on the use of ricin as a biological warfare agent [1,3]. In the United States, ricin was given the code name "Compound W" [1,3]. During World War II, the United States and Great Britain collaborated on the production of a bomb that contained ricin [2,3]; however, although this "W bomb" underwent testing, it

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0749-0704/05/$ – see front matter
doi:10.1016/j.ccc.2005.06.006 *criticalcare.theclinics.com*

was never used in combat [3]. After World War II, the United States and the Soviet Union continued to study ricin as a weapon, and in 1989, the Iraqi government allegedly tested the use of aerosolized ricin [2].

Ricin was also used as a homicidal agent by the Soviet Komitet Gosudarstvennoi Bezopasnosti (KGB). In 1978, a Bulgarian exile named Georgi Markov was assassinated in London. The assassin used a spring-loaded needle mounted within an umbrella to inject a small pellet of ricin into Mr. Markov's thigh [3]. He developed constitutional symptoms within a day and died 4 days later [8]. Another attempted homicide involving ricin occurred one month before this attack. Vladimir Kostov, a Bulgarian state radio and television correspondent, sustained a blow to his back in a Paris metro station. He was hospitalized for 12 days with a fever. After reading about the Markov case he was re-examined by physicians and the same type of ricin bullet was discovered in his skin. The bullet had not penetrated through the subcutaneous fat layer and so no ricin was absorbed systemically [1,8].

Since the 1990s, ricin has been found during seizure and examination of terrorist-related communications and documents [3,4]. In January 2003, authorities arrested six men suspected of producing ricin in their North London apartment [9,10]. In October 2003, a threatening letter along with a sealed container of ricin was processed at a mail facility in Greenville, South Carolina. The author threatened to poison the water supply if his demands were not met [11]. In January 2005, a 22-year-old man was arrested in Ocala, Florida, after a box of ricin was found in his home [12]. In February 2004, ricin powder was discovered on a mail-sorting machine in United States Senator Bill Frist's office building. Plans for ricin production were discovered in the possession of Chechen rebels and in Kabul, Afghanistan; both circumstances were thought to be related to planned terrorist activity [4].

Mechanism of toxicity

The toxicity of ricin is dependent on both the dose delivered and the route of the exposure [3,13]. The median lethal dose (LD_{50}) is the dose that would kill 50% of the people to whom it was applied; the smaller the LD_{50}, the less material is needed to kill the average person. For ricin, the LD_{50} is lowest for inhalation, and increases respectively for intravenous, intraperitoneal, subcutaneous, and intragastric administration [3]. Aerosolized ricin may lead to local pulmonary effects [14] as well as systemic effects following absorption. Ingestion of ricin is less likely to lead to toxicity, because of poor gastrointestinal absorption and potential enzymatic degradation of the protein within the gastrointestinal tract [3]. Contact allergies have been reported from dermal exposure (castor bean necklaces) [15]. Dermal absorption of ricin is poor [3,13], but systemic toxicity is possible if ricin is mixed in a solvent liquid, such as dimethyl sulfoxide [4], and is dispersed as an aerosol or liquid.

Ricin is a Type II ribosome-inactivating protein [1,2], which consists of two polypeptide chains bridged by a disulfide bond [1,2]. These chains must be linked to cause toxicity [1,3]. The B-chain, a lectin, has galactose-binding sites on either end to facilitate hydrogen bonding with cell surface glycoproteins and glycolipids [1,2,16,17]. The A-chain is an N-glycosidase [18,19] that removes adenine from the 28 S ribosomal RNA subunit [19]. This halts the binding of elongation factors, which results in the failure of protein synthesis [18,20]. Ricin is internalized by way of multiple mechanisms, including receptor-mediated endocytosis (Fig. 1) [21]. It is initially transported to the Golgi apparatus, then

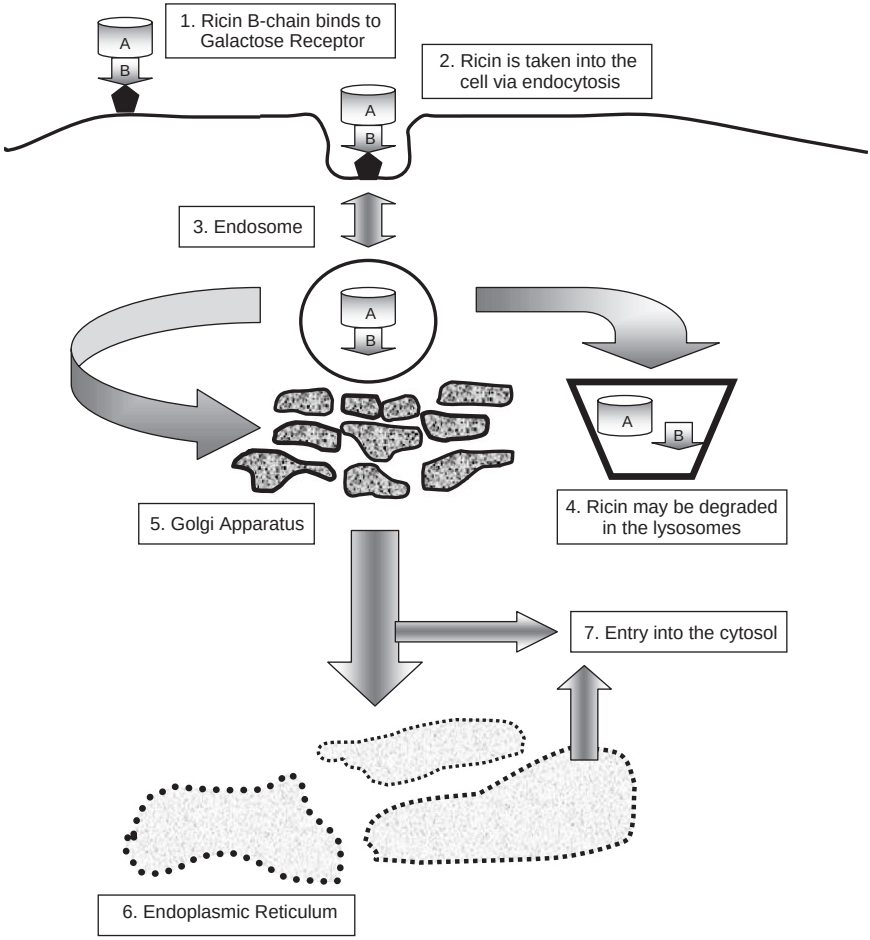

Fig. 1. Possible mechanisms of ricin entry into the cytosol. Steps in ricin internalization: The ricin B-chain binds to galactose receptors on the cell surface (1), and is endocytosed (2) into an endosome (3). Ricin is then either degraded by lysosomes (4) or transported into the Golgi apparatus (5), then either transported directly into the cytosol, or to the endoplasmic reticulum (6), and then into the cytosol (7).

the endoplasmic reticulum, and finally translocated to the cytosol [2,17,22]. Delivery to intracellular compartments is relatively slow (10% per hour at 37°C) [1,23]; however, only a small number of molecules present in the cytosol are necessary to produce cell death [2,18,24,25].

Ricin's potential as an immunotoxin has been studied since 1951, when the A-chain was discovered to inhibit tumor growth [1]. It has been used in Phase I and II clinical trials alone or as a component of tumor cell-specific antibodies (immunotoxins) [26–28]. Vascular leak syndrome, the result of ricin-mediated endothelial cell destruction, is a dose-limiting side effect resulting in hypoalbuminemia and edema [28]. Other limitations of therapy include the antibody's lack of specificity, and the immunogenicity of the toxin, which creates a refractory immunity in the host [27]. This immunogenicity has been exploited in the hopes of developing a ricin vaccine [29].

Clinical presentation

Ricin inhibits protein synthesis diffusely, and its systemic manifestations are non-specific and widespread. Signs and symptoms from oral and parenteral exposures resemble diffuse endothelial damage or the Systemic Inflammatory Response Syndrome (SIRS), and include fever, tachycardia, tachypnea, hypotension, hepatitis, pancreatitis, nephritis, myocardial injury, cerebral edema, vomiting, diarrhea, and bone marrow suppression [2,3,13]. The onset of symptoms after an exposure to ricin may be delayed for several hours. In vivo, the latency period between exposure and the symptom onset ranges from 8 to 24 hours [3]. Reports of non-fatal ingestions have demonstrated symptoms manifesting within several hours after ingestion [30–33]. Documented fatal and non-fatal human exposures have reported nausea, vomiting, abdominal cramps, diarrhea, dehydration, gastrointestinal hemorrhage, anuria, mydriasis, fever, and hypotension [2,3,13,30–34]. Transaminitis is commonly seen in patients [13] and may develop several days after the exposure [34]. In severe cases, death may ensue within several days [3], or patients may require several weeks of intensive care.

The only human data available for inhalational ricin exposure involves workers exposed to castor bean dust in castor oil processing plants. These patients developed nasal and throat congestion, irritation of the conjunctivae, urticaria, chest tightness, and bronchospasm [35]. Removal from exposure and supportive care led to symptom resolution [36]. Immunohistochemical studies in rats exposed to aerosolized ricin show that ricin binds to ciliated bronchiolar lining cells, alveolar macrophages, and alveolar lining cells [3]. A diffuse, necrotizing pneumonia, with interstitial and alveolar inflammation and edema, was observed in rats 8 hours after inhaling lethal doses of ricin [3]. A primate study found a dose-dependent latency period of 8 to 24 hours following ricin inhalation, followed by the onset of anorexia, decreased physical activity, and death within 36–48 hours. The time to death was also dose-dependent [3]. Acute

tracheitis, airway inflammation and necrosis, diffuse alveolar flooding, fibrinopurulent pneumonia, and purulent mediastinal lymphadenitis were seen on necropsy [37].

Although orally ingested ricin may lead to nausea, vomiting, diarrhea, dehydration, gastrointestinal hemorrhage, and hypotension, ingestion of intact castor beans rarely results in systemic symptoms or death. In fact, from 1983 to 2002, no deaths from castor bean exposure were reported to the American Association of Poison Control Centers Toxic Exposure Surveillance System [38], and there have been no reported fatalities from castor bean ingestion since 1935 [2].

When orally ingested, ricin acts locally on the intestinal mucosa, and excess toxin may be enzymatically degraded [39]. Mastication of castor beans may facilitate ricin release and increase the risk of local or systemic toxicity [13]. Autopsy findings following ingestion include hemorrhage and ulceration within the gastric and small-intestinal mucosa, necrosis of mesenteric lymph nodes, hepatic necrosis, as well as nephritis and splenitis. Once absorbed, ricin concentrates within the liver and spleen [39,40], possibly because of the presence of mannose receptors on the cells of the reticuloendothelial system (Kupffer cells and macrophages), which render them vulnerable to binding of the B-chain [13].

Parenteral ricin, when used in clinical trials in doses up to 23 mcg/m^2, has been well tolerated [26]. Fatigue and muscle pain were reported within 4 to 6 hours of the injections and these symptoms lasted for 24–48 hours [41]. Rats demonstrated Kupffer cell damage within 4 hours of being given intravenous ricin. Subsequent hepatic thrombi with resultant hepatocellular necrosis occurred after damage to the sinusoidal cells [42,43]. A transient leukocytosis in humans has been seen with both oral and intravenous administration of ricin [3]. There is a twofold to fivefold increase in the leukocyte count observed in cancer patients receiving ricin therapy [3,8]. Disseminated intravascular coagulation is seen in experimental animals that have received high-dose intravenous ricin [3]. Resultant hepatic and renal injuries may be secondary to toxin-mediated vascular damage and not direct toxin-mediated effects [44].

Intramuscular and subcutaneous injections of high doses of ricin in humans result in localized pain within the soft tissues and regional lymphadenitis [8]. Georgi Markov, the assassinated Bulgarian exile, received an estimated 500 μg of ricin injected into his right thigh. He developed immediate pain at the injection site, and complained of weakness within 5 hours of the injection [3]. He became febrile within 24 hours, with nausea and vomiting. He was not admitted to a hospital until 36 hours later, at which time he experienced fever, tachycardia, a stable blood pressure, and inguinal lymphadenopathy ipsilateral to the injection site. He developed induration around the puncture site. Approximately 48 hours after the injection, he became hypotensive and tachycardic (160 beats per minute). His white blood cell count was 26,300/mm^3. By the third day he had developed hematemesis and anuria. On the fourth day after the injection, he developed atrioventricular dissociation, cardiac arrest, and died. His final white blood cell count was 33,200/mm^3 [8]. A 36-year-old man injected the contents of a single castor bean (approximately 150 mg of ricin) intramuscularly and

developed headache, rigors, nausea, and tachycardia, with erythema and lymphadenopathy local to the injection site [45]. A 20-year-old man injected the extracted liquid of castor beans subcutaneously and developed abdominal pain, myalgias, vomiting, hypotension, metabolic acidosis, gastrointestinal bleeding, anuria, and death within several days of the injection [46].

Impact on critical care

Exposure to ricin may produce victims that require critical care in several ways. The contamination of communal resources, such as the food and water supply, could lead to a large number of exposures across widespread geographic areas with simultaneous presentations. Although it is likely that only a small percentage of these victims would require critical care, this may equate to a relatively large absolute number of critically ill patients. Alternatively, a small number of people may be targeted and poisoned by way of food or water supplies. This scenario may be more difficult to detect as the symptomatology is typically non-specific and may be confused with several other terrorist agents and naturally occurring diseases (Box 1). Finally, aerosolized ricin has the potential to expose a large number of people to a significant quantity of ricin at the same time. Patients may require critical care because of pulmonary edema and necrosis as well as systemic toxicity.

Box 1. Differential diagnosis

Staphylococcal enterotoxin B [3,5]
Tricothecene mycotoxin
Pyrolysis byproducts of Teflon, Kevlar (DuPont) [3]
Phosgene [3,5]
Paraquat [3]
Biologic toxins (S*taphylococcus aureus*, salmonella, shigella)
Viruses (Influenza, Norwalk virus, Rotavirus, Adenovirus)
Systemic Inflammatory Response Syndrome or Sepsis
Tularemia [5]
Plague [5]
Acute arsenic toxicity
Acute inorganic mercury, thallium, or iron ingestion
Acute radiation sickness
Chemotherapeutic agents
Capillary leak syndromes–autoimmune vasculitis, SJS

Decontamination

Patients who are exposed to ricin by way of liquid, powder, or aerosol must have their clothing removed and their skin thoroughly washed for 5–6 minutes with water before admission to the hospital [47]. Ricin does not require any unique external decontamination techniques [5].

If the patient was exposed by way of the gastrointestinal tract, or the exposure is unknown, gastrointestinal decontamination with gastric lavage and activated charcoal may be appropriate; however, gastric lavage is not likely to remove a significant amount of ricin unless it is performed within 1 hour of ingestion [48]. Ricin is poorly adsorbed by activated charcoal because of its large size [5]; however, activated charcoal may adsorb a small fraction of available ricin in the gastrointestinal tract and prevent absorption. Once absorbed, elimination techniques, including hemodialysis, are not effective. Once the patient has been externally decontaminated, they do not require isolation, negative pressure, or any personal protective equipment other than universal precautions [5,14]. Vomitus and diarrhea should be handled with barrier and splash precautions (eg, gown, eyeshield, mask, gloves), but is unlikely to produce a vapor or to expose health care workers to contamination [5].

Diagnostic studies

Laboratory evaluation should include renal and liver function tests, and electrolytes, which may need repletion secondary to volume loss. Critically ill and hypotensive patients should be evaluated with laboratory tests as if they have SIRS or sepsis, including blood gas, lactate, electrolyte panel including renal function tests, complete blood count, blood cultures, and cortisol testing.

Ricin may be detected via radioimmunoassay, ELISA, polymerase chain reaction (PCR), ricinine detection protocol, and time-resolved fluorescence immunoassay (TRF) [2]. Ricin has been measured in very low concentrations in both serum (1.5 μg/L) and urine (0.3 μg/L) after poisoning [32]. ELISA can detect ricin in human urine and serum at concentrations as low as 100 pg/mL [49]. This method is safe and accurate for use in the clinical setting [2]. Clinical testing for ricin may be performed by PCR at a regional public health laboratory by collecting 25 mL of urine [50,51]. The urine should be packaged as is instructed by the Centers for Disease Control and Prevention (www.bt.cdc.gov/labissues/pdf/shipping-samples.pdf and www.bt.cdc.gov/labissues/pdf/chemspecimencollection.pdf) [50,51]. TRF is a qualitative assay involving ricin-binding antibodies. It takes approximately 4 hours to complete. Faster assays will be required to detect aerosolized ricin [2]. To date, lab findings in animals exposed to airborne ricin are non-specific [3]. Antibodies are not detectable on initial evaluation; however, they may be present 2 weeks after exposure. [3] Early studies in rabbits and mice showed that these antibodies cross the placenta and are excreted in breast milk [3].

Antidotes, treatment, and supportive care

Supportive care is the mainstay of treatment. Hypotension should be treated with aggressive fluid replacement and direct-acting vasopressors, including phenylephrine, epinephrine, norepinephrine, or vasopressin. There is no evidence that any individual pressor is superior to another. However, there is evidence of increased endogenous norepinephrine release following ricin administration in rabbits, as well as decreased vascular responsiveness to norepinephrine [52,53]. Given this, vasopressin may be a reasonable alternative if catecholamines are ineffective.

Patients with non-cardiogenic pulmonary edema or airway edema may require mechanical ventilation. Although there is no research available to guide ventilator strategies, the pathophysiology of ricin pulmonary toxicity is a diffuse, inflammatory cellular toxicity. Therefore, ventilation strategies should likely mimic those for chemical pneumonitis or Acute Respiratory Distress Syndrome, with low tidal volume ventilation as a reasonable starting point [54].

Summary

Although ricin's history as a biologic weapon consists only of discrete episodes of poisoning, its widespread availability and ease of manufacture make it a viable terrorist threat. Clinicians must maintain a low threshold of suspicion for patients presenting with non-specific systemic illnesses, and be especially vigilant regarding large numbers of patients exhibiting gastrointestinal or pulmonary symptoms. The authors have outlined a plan of supportive care and diagnostic testing, that, along with a comprehensive differential diagnosis, should aid physicians in diagnosing and treating terrorist incidents involving ricin.

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