

Reactions to Imported Fire Ant Stings

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ABSTRACT

Reactions to the sting of the imported fire ant are an increasing problem for residents of the Gulf South. Three recent evolutionary changes suggest that the habitat of the ants will continue to expand. This article gives a brief review of the problem, including unresolved questions related to management of sting reactions. (Allergy Proc 14:13-16, 1993)

HISTORICAL PERSPECTIVES AND EPIDEMIOLOGY

Fire ants appear to have been introduced into the United States early in this century through the port of Mobile, Alabama.¹ The insects were initially an agricultural problem as their mounds damaged agricultural equipment used for farming, and, occasionally, the ants attacked food crops. As colonies became more dense, reports of attacks on livestock occurred. This continues to be a problem. With further spread of the ants, concentrations as high as 200 colonies per acre and greater have been reported in some southern states.² By 1985, the ants had infested 250 million acres in much of Florida, South Carolina, Georgia, Alabama, Louisiana, and Mississippi and had been noted to be present in North Carolina, Tennessee, Arkansas, Oklahoma, Texas, and Puerto Rico. The previous estimate

that the spread of the ants would be limited by average minimum temperatures of -12.5°C (10°F)³ probably no longer holds true for several reasons. Hybrids of *Solenopsis invicta* and *S. richteri*, the two imported fire ant species, have been found in northern Mississippi and Alabama. The hybrid is apparently more cold tolerant than the original species. Other recent adaptations include the appearance of multiple queens in fire ant colonies and the use of streets and highways as heat sumps that facilitate overwintering of the ants (Fig. 1).

At present, 30 to 60% of the population in urban areas infested by fire ants are stung each year.⁴ In stinging, the ant attaches itself to the skin using its mandibles, arches its body, and injects up to $.11\ \mu\text{L}$ of venom through a stinger located in the distal abdomen (Fig. 2). The ant then rotates its body around the mandibles and stings repeatedly. The venom induces an immediate, severe burning sensation at the site of the sting, followed by itching that may last for days.⁵

FIRE ANT VENOM ALLERGENS

In a series of recent studies, Hoffman and colleagues⁶ have increased our limited knowledge of the constituents of imported fire ant venom. Ninety to 95% of the venom is composed of water insoluble piperidine alkaloids that are irritant in nature and probably cause the sterile pustule, which is a characteristic of most fire ant sting sites at 12 to 24 hours. Four *S. invicta* proteins, which make up only 0.1% of the venom by weight, have been isolated and characterized. *Sol i I* composes 2 to 5% of venom protein and appears to have a molecular weight of 37,000 d. *Sol i II* venom composes approximately 67% of venom, has an apparent molecular weight of 28,000 d, and has phospholipase activity as does *Sol i I*. *Sol i III* composes about 30% of venom protein with a molecular weight of 26,000 d and has neurotoxin activity. *Sol i IV* composes some 9% of

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Figure 1. Typical fire ant mound near curbing in a residential area in Mobile, Alabama.

venom protein with a molecular weight of 15,000 d and seems to be present only in *S invicta*.⁵⁻⁹ All four proteins appear to be important allergens, and all four are immunologically unrelated to each other. Venoms from the two imported fire ant species are highly cross-reactive in both RAST and RAST inhibition.

The production of monoclonal antibodies to these venom proteins has allowed development of immunoassays for their quantitation in venom preparations obtained using various techniques and in whole-body extracts.

VENOM VERSUS WHOLE-BODY EXTRACTS

Several studies have demonstrated the variable quantity of venom proteins in commercially available whole-body extracts of imported fire ants, which are the only reagents available for diagnosis and treatment.^{10,11} Using immunoassays for *Sol i* III, Hoffman has demonstrated 10 to 16 $\mu\text{g/mL}$ of that allergen in commercial whole-body extracts. Concentrations of *Sol i* II have been more variable and range from extracts



Figure 2. Fire ant in the process of stinging.

containing very little allergen to potent extracts containing 7 to 25 $\mu\text{g/mL}$. Present techniques for obtaining venom from fire ants involve obtaining venom sacks of ants by dissection and are extremely laborious. Commercial techniques to facilitate obtaining larger quantities of venom have been developed.

REACTIONS TO IMPORTED FIRE ANT STINGS

Reactions to fire ant stings may be classified as local, systemic (anaphylactic), and other. At least three types of local reactions occur: a wheal-and-flare reaction, a sterile pustule, and a large local reaction.⁴

Immediate pain associated with a wheal-and-flare reaction followed by intense itching is the typical local reaction experienced by all who are stung by fire ants (Fig. 3). The wheal-and-flare reaction usually resolves in 30 minutes to an hour. A papule forms and evolves into a fully developed sterile pustule at the site of the sting within 24 hours. The histologic features of the pustule at 24 hours include necrotic tissue with cellular infiltration of polymorphonuclear cells, lymphocytes, and eosinophils.¹² This pustule is pathognomonic for imported fire ant (IFA) stings in the United States. The epidermis covering the pustule sloughs off over 48 to 72 hours, and healing takes place in 3 to 10 days, leaving a superficial scar or macule. Usually no treatment is indicated to prevent or resolve the pustules. If there are multiple lesions, and if they become excoriated and superinfected, pyoderma or even sepsis can develop. This is a special problem in diabetic patients stung on the lower extremity.

Between 17 and 56% of patients have large local reactions at the site of the IFA sting.¹² These lesions are erythematous, edematous, and indurated areas contiguous to the sting site (Fig. 4). They are extremely pruritic and last 24 to 74 hours. Large local reactions to fire ant stings have been shown to be mast-cell dependent, IgE-mediated, late-phase reactions.¹² Histologic examination of these reactions reveals cellular infiltrates of eosinophils and neutrophils with fibrin deposition in a gel structure. This gel structure facilitates the trapping of tissue fluids and edema formation, which in extreme cases may cause compression of nerves and blood vessels in affected extremities. These reactions are best treated symptomatically with oral antihistamines, topical corticosteroid ointments, and local anesthetic creams. If severe, they may require elevation of the extremity and systemic corticosteroid treatment for several days to relieve the discomfort and swelling and to prevent the need for decompressive surgery. These reactions do not require further evaluation or immunotherapy, inasmuch as they have not been shown to predispose patients to systemic allergic reactions.⁸

Systemic or anaphylactic reactions, on the other hand, are IgE-mediated reactions that involve symp-



Figure 3. Fully developed sterile pustules 24 hours after three fire ant stings in a normal person who did not experience a large local or systemic reaction to the stings.

toms and signs at sites remote from the sting site: generalized urticaria, cutaneous or laryngeal edema, bronchospasm, and/or cardiovascular collapse. Urticaria and angioedema are estimated to occur in 16% of stings and life-threatening anaphylaxis follows 0.6 to 4% of stings.⁸ Thirty-two documented deaths due to IFA stings have been reported in the southern United States.¹³ Emergency room physicians in areas infested with IFA know to check between the toes and elsewhere on the lower extremities for evidence of fire ant stings when patients present with anaphylaxis. Anaphylaxis usually occurs shortly after a sting. However, a case report described such a reaction hours after a sting.¹⁴

Other, unusual neurotoxic reactions to IFA stings have been reported,¹⁵ including focal or generalized seizures and peripheral neuropathy occurring minutes to 36 hours after urticaria, angioedema, or large local reactions. The pathogenetic mechanisms causing the

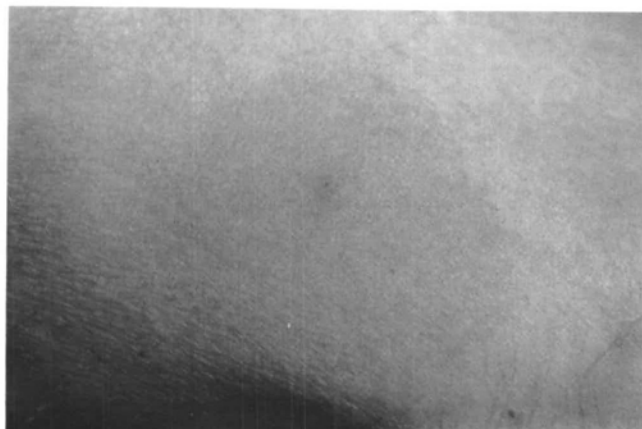


Figure 4. Developing late phase allergic reaction 6 hours after fire ant sting in a patient with a history of large local reactions. A developing sterile pustule is present in the center of the reaction.

reactions. The pathogenetic mechanisms causing the neurologic reactions are uncertain. There is reason to think they could be toxic or immunologic in origin. Nephrotic syndrome also has been reported as a complication of ant stings.¹⁶ More recently, the attack of a neonate in bed by fire ants was reported.¹⁷ The infant experienced an almost fatal reaction of shock, coma, hemolytic anemia, and coagulopathy.¹⁷

IMMUNOTHERAPY FOR PREVENTION OF ANAPHYLAXIS

Studies of anaphylaxis in response to the stings of other Hymenoptera, such as honeybees and yellow jackets, have established that the production of IgE after stings is common and often transient. Furthermore, the clinical manifestations of anaphylaxis, the probability of serious reactions after subsequent stings, and the duration of sensitization tend to be higher in adults than in children. The natural history of sensitivity to IFA stings, however, is unknown, and the indications for immunotherapy to prevent the recurrence of IFA sting anaphylaxis are therefore unclear.

The experience with immunotherapy for honeybees, yellow jackets, and wasps has demonstrated that whole-body extracts are of no value, because they contain little or no venom. Venom immunotherapy, on the other hand, has shown to be safe and effective in patients with systemic reactions after honeybee and yellow jacket stings.^{18,19} Extracts of IFA venom are not yet commercially available and whole-body IFA extracts presently are used to diagnose and to treat thousands of affected patients who live in infested areas. Fortunately, there is a substantial, although variable, amount of venom in IFA whole-body extracts (IFA-WBE). Treatment consists of weekly subcutaneous injections of IFA-WBE, with the dose increasing until a maintenance dose, usually 0.5 mL of a 1:10 dilution, is reached. This maintenance dose is then given monthly or at longer intervals for at least 3 to 5 years. The effectiveness of IFA-WBE was first supported in a study by Hylander et al.²⁰ Findings of that study suggested that IFA-WBE contains relevant allergens and that immunotherapy with IFA-WBE is efficacious for most patients by 2 years, at which time patients tolerate stings without reaction. In additional studies, sting challenges in 30 patients with previous systemic reactions and subsequent immunotherapy with IFA-WBE induced no systemic reactions.²¹ Research is under way to develop IFA venom for diagnosis and therapy. In our own practice, each new lot of commercial IFA-WBE is tested before use in diagnosis and treatment by using skin testing in a panel of patients known to have IgE to IFA venom to assure that the relevant allergens are present.

Two native species of fire ants, *S. xyloni* and *S. aurea*, have been reported to cause systemic reactions as well, but less is known about the constituents of their venom.

CHEMICAL CONTROL OF IMPORTED FIRE ANTS

Earlier attempts to control fire ants led to the use of pesticides that were toxic to wildlife and were thus discontinued. The two approaches to control fire ants presently available include treatment of individual mounds and "broadcast" treatments. These techniques have recently been reviewed in detail.²² Treatment of individual mounds with pesticides including diazinon granules or drenches or injecting mounds with chlorpyrifos only temporarily clears small areas. Broadcast treatments use baits which attract ants. The baits consist of soybean oil, corn grits, and a chemical agent. The bait is picked up by a worker and taken deep into the mound and eventually fed to the queen. Baits have been impregnated with insecticides and synthetic versions of natural insect growth hormones. These agents appear to be capable of clearing large areas of ants for months at a time.

CONCLUSION

Fire ant stings are the source of increasing numbers of allergic reactions to their venom in residents of the Gulf south and southeastern United States. As the ants continue to adapt to their adopted environment, this problem can be anticipated to increase. A better understanding of the natural history of fire ant allergy, the allergens in fire ant venom, and the availability of venom for diagnosis and for treatment is needed.

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BRIEF SUMMARY

CONTRAINDICATIONS: Hypersensitivity to any of the ingredients of this preparation contraindicates its use.

WARNINGS: The replacement of a systemic corticosteroid with a topical corticoid can be accompanied by signs of adrenal insufficiency and, in addition, some patients may experience symptoms of withdrawal, e.g., joint and/or muscular pain, lassitude and depression. Patients previously treated for prolonged periods with systemic corticosteroids and transferred to topical corticoids should be carefully monitored for acute adrenal insufficiency in response to stress. In those patients who have asthma or other clinical conditions requiring long-term systemic corticosteroid treatment, too rapid a decrease in systemic corticosteroids may cause a severe exacerbation of their symptoms.

Children who are on immunosuppressant drugs are more susceptible to infections than healthy children. Chickenpox and measles, for example, can have a more serious or even fatal course in children on immunosuppressant doses of corticosteroids. In such children, or in adults who have not had these diseases, particular care should be taken to avoid exposure. If exposed, therapy with varicella zoster immune globulin (VZIG) or pooled intravenous immunoglobulin (IVIG), as appropriate, may be indicated. If chickenpox develops, treatment with antiviral agents may be considered.

The use of Nasacort Nasal Inhaler with alternate-day systemic prednisone could increase the likelihood of hypothalamic-pituitary-adrenal (HPA) suppression compared to a therapeutic dose of either one alone. Therefore, Nasacort Nasal Inhaler should be used with caution in patients already receiving alternate-day prednisone treatment for any disease.

PRECAUTIONS

General: In clinical studies with triamcinolone acetonide administered intranasally, the development of localized infections of the nose and pharynx with *Candida albicans* has rarely occurred. When such an infection develops it may require treatment with appropriate local therapy and discontinuance of treatment with Nasacort Nasal Inhaler.

Triamcinolone acetonide administered intranasally has been shown to be absorbed into the systemic circulation in humans. Patients with active rhinitis showed absorption similar to that found in normal volunteers. Nasacort at 440 mcg/day for 42 days did not measurably affect adrenal response to a six-hour cosyntropin test. In the same study prednisone 10 mg/day significantly reduced adrenal response to ACTH over the same period (see CLINICAL TRIALS section).

Nasacort Nasal Inhaler should be used with caution, if at all, in patients with active or quiescent tuberculous infections of the respiratory tract or in patients with untreated fungal, bacterial, or systemic viral infections or ocular herpes simplex.

Because of the inhibitory effect of corticosteroids on wound healing in patients who have experienced recent nasal septal ulcers, nasal surgery or trauma, a corticosteroid should be used with caution until healing has occurred.

When used at excessive doses, systemic corticosteroid effects such as hypercorticism and adrenal suppression may appear. If such changes occur, Nasacort Nasal Inhaler should be discontinued slowly, consistent with accepted procedures for discontinuing oral steroid therapy.

Information for Patients: Patients being treated with Nasacort Nasal Inhaler should receive the following information and instructions.

Patients who are on immunosuppressant doses of corticosteroids should be warned to avoid exposure to chickenpox or measles and, if exposed, to obtain medical advice.

Patients should use Nasacort Nasal Inhaler at regular intervals since its effectiveness depends on its regular use. A decrease in symptoms may occur as soon as 12 hours after starting steroid therapy and generally can be expected to occur within a few days of initiating therapy in allergic rhinitis. The patient should take the medication as directed and should not exceed the prescribed dosage. The patient should contact the physician if symptoms do not improve after three weeks, or if the condition worsens. Nasal irritation and/or burning or stinging after use of the spray occur only rarely with this product. The patient should contact the physician if they occur.

For the proper use of this unit and to attain maximum improvement, the patient should read and follow the accompanying patient instructions carefully. Because the amount dispensed per puff may not be consistent, it is important to shake the canister well. Also, the canister should be discarded after 100 actuations.

Carcinogenesis, Mutagenesis: Animal studies of triamcinolone acetonide to test its carcinogenic potential are underway.

Impairment of Fertility: Male and female rats which were administered oral triamcinolone acetonide at doses as high as 15 mcg/kg/day (110 mcg/m²/day, as calculated on a surface area basis) exhibited no evidence of impaired fertility. The maximum human dose, for comparison, is 6.3 mcg/kg/day (240 mcg/m²/day). However, a few female rats which received maternally toxic doses of 8 or 15 mcg/kg/day (60 mcg/m²/day or 110 mcg/m²/day, respectively, as calculated on a surface area basis) exhibited dystocia and prolonged delivery.

Developmental toxicity, which included increases in fetal resorptions and stillbirths and decreases in pup body weight and survival, also occurred at the maternally toxic doses (2.5 - 15.0 mcg/kg/day or 20 - 110 mcg/m²/day, as calculated on a surface area basis). Reproductive performance of female rats and effects on fetuses and offspring were comparable between groups that received placebo and non-toxic or marginally toxic doses (0.5 and 1.0 mcg/kg/day or 3.8 mcg/m²/day and 7.0 mcg/m²/day).

Pregnancy: Pregnancy Category C. Like other corticoids, triamcinolone acetonide has been shown to be teratogenic in rats and rabbits. Teratogenic effects, which occurred in both species at 0.02, 0.04 and 0.08 mcg/kg/day (approximately 135, 270 and 540 mcg/m²/day in the rat and 320, 640 and 1280 mcg/m²/day in the rabbit, as calculated on a surface area basis), included a low incidence of cleft palate and/or internal hydrocephaly and axial skeletal defects. Teratogenic effects, including CNS and cranial malformations, have also been observed in non-human primates at 0.5 mcg/kg/day (approximately 6.7 mcg/m²/day). The doses of 0.02, 0.04, 0.08, and 0.5 mcg/kg/day used in these toxicology studies are approximately 12.8, 25.5, 51, and 318.7 times the minimum recommended dose of 110 mcg of Nasacort per day and 3.2, 6.4, 12.7, and 80 times the maximum recommended dose of 440 mcg of Nasacort per day based on a patient body weight of 70 kg. Administration of aerosol by inhalation to pregnant rats and rabbits produced embryotoxic and fetotoxic effects which were comparable to those produced by administration by other routes. There are no adequate and well-controlled studies in pregnant women. Triamcinolone acetonide should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Experience with oral corticoids since their introduction in pharmacologic as opposed to physiologic doses suggests that rodents are more prone to teratogenic effects from corticoids than humans. In addition, because there is a natural increase in glucocorticoid production during pregnancy, most women will require a lower exogenous steroid dose and many will not need corticoid treatment during pregnancy.

Nonteratogenic Effects: Hypoadrenalism may occur in infants born of mothers receiving corticosteroids during pregnancy. Such infants should be carefully observed.

Nursing Mothers: It is not known whether triamcinolone acetonide is excreted in human milk. Because other corticosteroids are excreted in human milk, caution should be exercised when Nasacort Nasal Inhaler is administered to nursing women.

Pediatric Use: Safety and effectiveness have not been established in children below the age of 12. Oral corticoids have been shown to cause growth suppression in children and teenagers, particularly with higher doses over extended periods. If a child or teenager on any corticoid appears to have growth suppression, the possibility that they are particularly sensitive to this effect of steroids should be considered.

ADVERSE REACTIONS: In controlled and uncontrolled studies, 1257 patients received treatment with intranasal triamcinolone acetonide. Adverse reactions are based on the 567 patients who received a product similar to the marketed Nasacort canister. These patients were treated for an average of 48 days (range 1 to 117 days). The 145 patients enrolled in uncontrolled studies received treatment from 1 to 820 days (average 332 days).

The most prevalent adverse experience was headache, being reported by approximately 18% of the patients who received Nasacort. Nasal irritation was reported by 2.8% of the patients receiving Nasacort. Other nasopharyngeal side effects were reported by fewer than 5% of the patients who received Nasacort and included: dry mucous membranes, naso-sinus congestion, throat discomfort, sneezing, and epistaxis. The complaints do not usually interfere with treatment and in the controlled and uncontrolled studies approximately 1% of patients have discontinued because of these nasal adverse effects.

In the event of accidental overdose, an increased potential for these adverse experiences may be expected, but systemic adverse experiences are unlikely (see OVERDOSAGE section).

OVERDOSAGE: Acute overdose with this dosage form is unlikely. The acute topical application of the entire 15 mg of the canister would most likely cause nasal irritation and headache. It would be unlikely to see acute systemic adverse effects if the nasal application of the 15 mg of triamcinolone acetonide was administered all at once.

Caution: Federal (U.S.A.) law prohibits dispensing without prescription. Please see product circular for full prescribing information.

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