Contact Dermatitis

Dermatitis is a skin eruption which follows a set pattern of evolution. At first, it is confined only to the areas where the toxin, allergen or chemical has touched the skin of the victim. It starts with a reddening of the skin, accompanied by burning or itching. Next, there may be a roughening or blistering of the skin. This is followed by swelling, weeping and cracking. It finally resolves by drying, peeling, and reversal of all the proceeding symptoms.

There are two major types of contact dermatitis: Allergic Eczematous Contact Dermatitis and Primary Irritant Dermatitis. Both of these forms appear and are caused by something in the environment, e.g. toxic chemicals, dust, lichens, wood fiber, weeds etc.

ALLERGIC CONTACT DERMATITIS: More often than not, this is the more explosive of the two and tends to spread further and occur in an “all or none” fashion. It is due to substances which do not necessarily cause dermatitis on all people. People in the same occupation may react entirely different to exposure of the same toxin. The individual who usually reacts has probably had a history of other rashes e.g. Earrings, cosmetics, topical medications, dust or poison oak etc.

PRIMARY IRRITANT DERMATITIS: This is often the result of overexposure to a toxic chemical, detergent, solvent, resin, or other hydrocarbon. The dermatitis does not usually spread far from the site of exposure on the skin. Generally speaking, some sort of damage will result in the large percentage of the population if they are exposed to high concentrations of these materials.

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If contact with the offending agent continues over a prolonged period of time, many problems may begin to develop. The first symptom to arise after exposure is itching. This itching may become intolerable. The torment of this symptom often causes the victim to tear at their skin with great ferocity. This often leads to secondary infection, resulting in “blood poisoning”, also referred to as septicemia or lymphangitis-

This should be treated with antibiotics until the infection is controlled.

The second symptom to arise denoting severe contact dermatitis is a chronic worsening of the first symptom resulting in, skin thickening, pigmentation, drying, cracking and itching, which lasts long after the last exposure to the original incitant. Hypersensitivity or over-reactivity of the skin often develops next. Skin reacts overtly to rubbing, heat, sunlight or other physically induced excitants. Multiple allergic reactions may now show up which were not present or were subliminal before the major skin event.

Several diagnostic criteria must be met in order to establish contact dermatitis:

#1) A pattern of diability of the type described above often develops.

#2) A history of the appearance of dermatitis following exposure to a known toxic chemical, or allergen in the workplace, home or recreational environment.

#3) A history of lessening or disappearance of dermatitis when the individual is removed from his environment for a greater or lesser period of time. This is followed by a sudden reappearance of the dermatitis when re-exposed to the same conditions.

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One diagnostic test often used to diagnose dermatitis is patch testing. Patch testing can be quite difficult to employ technically, and to interpret, often requiring the services of someone with special skills in this field. Most qualified dermatologists can do screen tests which may be valuable in delineating the cause. Testing to toxic materials used in the workplace is not recommended. Besides causing a burn, a nasty flare-up of the dermatitis could be provoked.

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Protection usually is the most important factor in prevention of dermatitis. Individuals involved in occupations involving the use of irritating chemicals should wear protective clothing, use proper ventilation and always wear protective gloves. If this is not possible, then retraining or a job change may be necessary.

In summary: Contact dermatitis is a skin disease with many variables, it is hard to control and may have many things influencing its continuation besides the original toxin or allergen.

(It should be noted that to cover this subject in depth is not practical in this article, I have only laid out the basics of a subject on which many volumes and scientific journals have been written.)
minor or entrapment between the clavicle and first rib is suggested. Additional positive physical finding may included fullness above the clavicle, muscle wasting, atrophy, and weakness in the upper extremity. Abruit may be heard with the stethoscope placed over the supraclavicular fossa. There may be venous congestion in the arm one would expect.

While the Adson, Costoclavicular and Wright maneuvers may or may not be positive, various laboratory studies are often needed to pin down the diagnosis. X-ray films should include views of the cervical spine looking for cervical ribs, and evidence of arthritis. Electrodiagnostic studies (EMG and nerve conduction) may suggest a thoracic outlet syndrome but are more useful in excluding cervical radiculopathy, ulnar neuropathy at the elbow (cubital tunnel syndrome) or median nerve compression at the wrist (carpal tunnel syndrome).

Evaluation of the arterial component in thoracic outlet syndrome is assisted by the Doppler studies but relies on the arteriogram for definitive diagnosis. The arteriogram is considered the “gold standard” and it is important for both arterial and venous flow be observed.

The differential diagnosis would include conditions of the cervical spine such as cervical spondylosis or disc herniation, supraclavicular fossa tumor from lung cancer, brachial neuritis, cubital tunnel syndrome, carpal tunnel syndrome, reflex sympathetic dystrophy, and primary conditions of the shoulder.

“Treatment is usually conservative and relies heavily on exercises to correct postural abnormalities and to strengthen neck and shoulder girdle musculature, often under the direction of a physical therapist. Identification and treatment of underlying depression, if suspected, would be an integral part of the conservative treatment. A weight reduction program might be appropriate. In cases of severe thoracic outlet compression, exercises may increase symptoms and may not produce favorable results. Exercises should be performed for a period of two or three months before abandoning them.

With failure of conservative treatment, one may resort to surgical treatment, but this is generally withheld for the most refractory cases. There are several procedures described for treatment as there are several anatomic abnormalities. However, the common denominator for the successful surgical procedure involves removal of a portion of the first rib, often through a transaxillary exposure. Injury to the brachial plexus or veins are complications of the surgery. Surgical treatment of thoracic outlet syndrome in my community falls into the purview of the thoracic or vascular surgeon. In other communities, depending upon his interest and experience, the Orthopedic Surgeon may be involved with the surgical treatment.

REFERENCES

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