Essential acquired cold urticaria: Stimulated only by systemic as well as local cooling

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The background and differential diagnosis of essential acquired cold urticaria are discussed. The case report is of a 22-year old male who had previously been diagnosed as suffering from eczema. Working in a cold environment, with both systemic and local cooling stimulated development of an urticaria rash, diagnosed as being essential acquired cold urticaria.

INTRODUCTION

Urticaria can be produced by a wide range of stimuli. Recently, contact urticaria has been categorized as due to either chemically defined substances, or chemically undefined biological materials. Contact with water may produce an urticarial reaction either directly or as a feature of cold urticaria.

Essential acquired cold urticaria occurs rarely—usually in young adults and more commonly among women than men. It has been suggested that the stimulus is a drop in temperature, rather than the absolute temperature. The differential diagnosis includes cryoglobulinaemia, paroxysmal cold haemoglobinaemia, cold haemagglutination, and essential familial cold urticaria. In the first three of these it is more common to see purpura and necrosis as the main skin changes, possibly in association with Raynaud's phenomenon. Familial cold urticaria occurs as an autosomal dominant trait, often with a delayed response to cold exposure and, in some cases, local cold provocation does not produce wealing.

Cases of essential acquired cold urticaria have been reported in which systemic cold exposure has been necessary to produce the symptoms of urticaria. A number of pathological mechanisms for essential acquired cold urticaria have been suggested, including histamine and prostaglandin D2 as mediators. Consequently a combination of H1 and H2 blockers may be needed to control the condition, although avoidance of the precipitating environmental conditions should be regarded as the first line treatment.

CASE REPORT

The patient was a 22 year old male. He presented in the occupational health department soon after starting work as a management trainee in a food processing company. He declared a long history of eczema on both hands, for which he had been regularly using topical steroid cream. Apart from seasonal hayfever, there was no other history of ill health. There was no family history of skin disease.

On examination, there was a diffuse scaly rash on the dorsal aspect of both hands, with areas of telangiectasia and skin atrophy. There was also an excoriated red scaly rash on legs and trunk.

Before reviewing the occupational health aspects of an employee in a food factory having such a problem, it was decided that further control of the rash was likely to be possible. In view of the atrophic changes and telangiectasia, use of the steroid cream was stopped. Over the next three months he continued to work, although the rash persisted with only intermittent improvements. In particular the fingers and dorsa of his hands continued to show eczematous changes.

During a cold spell of weather, he was coincidentally transferred to work in a chilled part of the factory. Soon afterwards he presented with a clear acute urticarial rash across the exposed area of his face and forehead, although the rash on his hands had improved. At that stage he volunteered that the rash had always been worse in winter than summer.

A cold provocation test was undertaken by placing an icepack on the dorsum of his right hand. After 5 minutes a pronounced urticarial rash was present over the area corresponding to the position of the icepack. A diagnosis of essential acquired cold urticaria was made. He was treated with a combination of H1 and
H2 blockers with little immediate effect. Following occupational health advice, he was deployed to work in a warm office, avoiding work in the cold areas of the factory, and the urticaria settled, although it took several weeks to fully resolve.

Several weeks later the cold provocation test was repeated. At that stage the external ambient temperature was approximately 7° C warmer. There was no reaction to the cold provocation test. The facial rash had dramatically improved, although there was a persistent rash on his legs and trunk.

CONCLUSION

This is a case of previously undiagnosed essential acquired cold urticaria, another in which systemic cooling was necessary to produce a reaction to local cold provocation. One interesting feature of this case is that the diagnosis was made following general cooling and local cooling—to the face—both of which were of occupational origin. Previous reports of occupational exacerbation of cold urticaria emphasized the need for accurate diagnosis, including careful examination of the working conditions and the use of a simple cold provocation test, following which medical criteria for employment can be defined. Apart from occupational environmental exposure to cold, local cooling from an aerosol spray used at work has been described.

A programme of cold tolerance, involving repeated exposure to cold, may be useful in the short term by depleting mast cell stores of histamine; this is unlikely to be of benefit in the context of occupational health advice.

REFERENCES