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We read with great interest the case presented by Czirják et al. [1] and should like to thank them for their attention to our article.

Different substances are able to elicit systemic scleroderma and sclerodermiform alterations. It has been reported that with the exception of silica, a cause of systemic sclerosis identical to the idiopathic form, the other substances produce sclerodermiform alterations and pictures of generalized morphea that can be partially reversed on interrupting exposure [2]. Recently, cases have been described [3-5] that suggest that other sclerosis-inducing substances, such as trichloroethylene, are able to induce clinical states that are undistinguishable from the classic 'idiopathic' systemic sclerosis.

Like vinyl chloride, trichloroethylene is an aliphatic hydrocarbon, and there is a clinical similarity between the disease caused by vinyl chloride and the alterations due to trichloroethylene exposure [6, 7]. The thrombopenia of the patient referred to by Czirják et al. [1] has been observed in patients exposed to vinyl chloride [8].

Reports have been made of genetic susceptibility among affected workers exposed to vinyl chloride, HLA-DR5 being a marker of susceptibility and HLA-B8 and -DR3 having been associated with progression of the disease [9]. It would have been interesting to have information on the histocompatibility antigens of the patient described by Czirják et al. [1] and, with further studies in other patients, to be able to confirm these findings since numerous factors have been implied in the etiopathogenesis of scleroderma, although its cause remains to be elucidated.

Since in many cases, once the clinical picture has begun, interruption of exposure to the suspected agent does not lead to remission of the disease, the only way to prevent new cases would be to take into account the occupational antecedents of workers and possible individual predispositions. This would further contribute to our insight into this disease.

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