Letter to the Editor

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Systemic Lupus erythematosus following Hepatitis B Vaccine

Systemic lupus erythematosus (SLE) is a connective-tissue disease the specific cause of which remains undetermined. An autoimmune physiopathologic process is undoubtedly involved, probably onto a genetic predisposition. Now, some chemical agents are able to produce a lupus-like syndrome, the features of which associate antinuclear antibodies directed to the DNA-histones complex, normal levels of complement, and absence of antibody to native DNA [1]. Recombinant hepatitis B vaccination is effective and well tolerated [2], and is more and more often offered to the general population in industrial countries. Serious adverse reactions are very rare [3], including immune-mediated diseases such as thrombocytopenic purpura [4], erythema nodosum [5], and glomerulonephritis [6]. To our knowledge, the first case of SLE identified as occurring following antihepatitis B vaccine was reported by Tudela et al. [7]. McMahon et al. [3] too noticed 1 case of cerebritis secondary to systemic lupus after hepatitis B vaccine, out of 43,618 vaccinated persons. However, they rejected it as an inappropriate diagnosis of adverse reaction to vaccination [3]. We report here another case of SLE following such an immunization.

A 26-year-old mixed-blood social worker woman was given a first recombinant antihepatitis vaccine dose (GenHevac-B) in September 1994. One week later, she experienced fever and chills, then a vaginal discharge, followed by cutaneous eruption of the face, arms and legs. She was referred to the local hospital where ocular and pulmonary involvements were noticed. Cutaneous biopsy showed lupus-like histopathologic changes but immunofluorescence was negative, as antinuclear antibody testing. In November, the diagnosis was assessed by antinuclear antibodies at 1/500 homogeneous, Farr test 92 IU, complement component C3 246 (normal range 550–1,200) amd C4 level 58 (200–500) mg/l. Like Tudela et al. [7], we believe that vaccination can induce an immune stimulation that may reveal or trigger a latent auto-immune genetic predisposition. The mechanism seems different from the chemical or drug-induced lupus-like syndrome, and closer to classical SLE. Whether thimerosal used as preservative, or aluminium hydroxide used as adjuvant, or Hb surface antigen protein [8] as in polyarteritis nodosa is the cause of the syndrome [2]. LED should now be added to the list of rare recombinant hepatitis B vaccine side-effects. When any immune lupus-like manifestation occurs, the patient should be checked up on systematically about a recent antihepatitis B vaccination, and the protocol be stopped, antinuclear antibody testing should be performed 3 months later. An early short corticosteroid course may be relevant.

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