Systemic polyarteritis nodosa following hepatitis B vaccination.

de Carvalho JF, Pereira RM, Shoenfeld Y.

Source

Rheumatology Division, São Paulo University School of Medicine, São Paulo, Brazil.

Abstract

The authors report a patient who developed systemic polyarteritis nodosa two months after hepatitis B vaccination and review the literature concerning this vaccination and the development of autoimmune conditions, mainly vasculitis. A 14-year-old boy who had no relevant previous history and who was not taking any drugs presented with a livedo reticularis, fever, loss of weight, testicular pain, and paresthesias two months after receiving the third dose of a hepatitis B vaccination. Inflammatory parameters (ESR and CRP) were high. The patient met the ACR diagnostic criteria for polyarteritis nodosa. He received corticosteroids and immunosuppressants and showed improvement. After reviewing the 27 cases of vasculitis after hepatitis B vaccination reported in the current literature, the authors suggest that, in some cases, vaccination may be the triggering factor for vasculitis in individuals with a genetic predisposition. Physicians should be aware of this possible association.

Status epilepticus and lymphocytic pneumonitis following hepatitis B vaccination.
Source

Rheumatology Division, São Paulo University School of Medicine, São Paulo, Brazil.

Abstract

The case reported refers to a patient who developed status epilepticus in the day of her third dose of hepatitis B vaccination and we review the literature on this subject. A 12 year-old girl, without a relevant previous history, taking no drugs, developed a seizure attack followed by unconsciousness, and eventually died after three days of her third dose of hepatitis B (HB) vaccination. Autopsy study revealed cerebral edema with congestion and herniation and diffuse interstitial type pneumonitis. There seem to be a straight forward time relationship between the third HB vaccine, the event of convulsion and the sudden death of the patient. We suggest that, in some cases, vaccination may be the triggering factor for autoimmune and neurological disturbances in genetically predisposed individuals and physicians should be aware of this possible association.


Acquired autoimmunity after viral vaccination is caused by molecular mimicry and antigen complimentarity in the presence of an immunologic adjuvant and specific HLA patterns.

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Source

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Abstract

Acquired autoimmunity syndromes occur after viral vaccinations. Molecular mimicry is involved in these phenomena as is the necessity for the presence of two chemically complimentary antigens and an immunologic adjuvant. The HLA pattern of the host is
also an important factor. The example used to explain these phenomena is
demyelinating disease that follows hepatitis B vaccination. The somatic antigen of the
hepatitis B virus in the vaccine has chemical complimentarity with the Epstein-Barr
virus antigen in the vaccine recipient. The Epstein-Barr virus shows molecular
mimicry with human myelin. The immunologic adjuvant is either present in the
vaccine or muramyl peptides in the individual who is vaccinated. Why more than one
type of autoimmune disease occurs is explained by the fact that specific autoimmune
T-cells have been shown to develop clones that attack multiple human tissues.


A study of molecular mimicry and
immunological cross-reactivity
between hepatitis B surface antigen
and myelin mimics.

Bogdanos DP, Smith H, Ma Y, Baum H, Mieli-Vergani G, Vergani D.

Source

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Abstract

On the basis of the reported association between hepatitis B vaccination (HBvacc)
and autoimmune demyelinating complications such as multiple sclerosis (MS), we
have looked for aminoacid similarities between the small hepatitis B virus surface
antigen (SHBsAg), and the MS-autoantigens myelin basic protein (MBP) and myelin
oligodendrocyte glycoprotein (MOG) that could serve as targets of immunological
cross-reactivity. Twenty-mer peptides spanning 4 SHBsAg/MOG and 1 SHBsAg/MBP
mimicking pairs, were constructed and tested by ELISA as targets of cross-reactive
responses. A total of 147 samples from 58 adults were collected before HBvacc
(58/58), and post-HBvacc (48/58 before the second and 41/58 before the third
boost). Eighty-seven sera from anti-SHBsAg antibody negative patients with various
diseases were tested as pathological controls. Reactivity to at least one of the
SHBsAg peptides was found in 8 (14%) pre-HBvacc subjects; amongst the remaining
50, reactivity to at least one of the SHBsAg peptides appeared in 47 (94%) post-
HBvacc. Reactivity to at least one of the MOG mimics was present in 4 (8%) pre-
HBvacc and in 30 (60%) post-HBvacc (p < 0.001). Overall 30/50 (60%) vaccinees
had SHBsAg/MOG double reactivity on at least one occasion compared to none
before-vaccination and in 2 (2%) of the pathological controls (p < 0.001 for both).
SHBsAg/MOG double reactivity was cross-reactive as confirmed by inhibition
studies. At 6 months post-vaccination, 3 of the 4 anti-MOG reactive cases before vaccination and 7 of the 24 (29%) of the anti-MOG reactive cases at 3 months post-vaccination had lost their reactivity to MOG5-24. There was no reactivity to the SHBsAg/MBP mimics. None of the vaccinees reported symptoms of demyelinating disorders. In view of the observed SHBsAg/MOG cross-reactivity, the vaccine's possible role as an immunomodulator of viral/self cross-reactivity must be further investigated.

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