A case of cutaneous necrosis during interferon-β1b (B-IFN) therapy in multiple sclerosis

Our patient, a thirty eight year old woman, had had relapsing-remitting multiple sclerosis since the age of 17. In September 1999 interferon-β1b (B-IFN, Betaferon, Schering) was introduced. After a training session the patient injected 5 million EU on alternate days. Injection sites were the thighs and the abdomen. The patient was seen 2, 4, 8, and 12 weeks after the beginning of the therapy. Some erythematous patches were noted at the injection sites. Otherwise there were no side effects. Blood tests performed after four weeks were normal. From the third month of treatment the patient complained of multiple painful scars at the injection sites. We examined her and found multiple severe necrotic skin ulcers with surrounding erythema at the injection sites (figure, A). After a review of the literature,1 we considered an interruption of the treatment. As the patient was eager to continue, an investigation on methods of self administration disclosed that the patient kept vials of B-IFN and saline in a refrigerator (+ 5°C). The vials were taken from the refrigerator just before application. After mixing, the substance was injected. We decided to heat the vials to body temperature for five minutes before mixing and to bring the suspension to body temperature by keeping it in a breast pocket for 10 minutes. After modifying the mixing procedure, no new scars appeared in the next six months of treatment (figure, B). The few cases previously reported2–6 with severe necrotic cutaneous lesions during B-IFN therapy have stressed a possible immunological basis for this serious side effect. We postulate that a local reaction to the improperly dissolved lyophilised substance has caused the necrotic ulcers.

CARLO ALBANI
Research Institute of Neurology, IRCCS, Casimiro Mondino, Bahnhofplatz 2, 8023 Zurich, Switzerland
GIOVANNI ALBANI
Institute of Neurology, IRCCS, Casimiro Mondino, Via Palestro 3, 27100 University of Pavia, Italy
Correspondence to: Dr Carlo Albani, Laboratory of Neurophysiology, Bahnhofplatz 2, 8023 Zurich, Switzerland


418
The hippocampi, essential for episodic memory function, are also rich in serotonin releasing neurons and are known to be targets of MDMA neurotoxicity in animals. Therefore we propose that in the present case MDMA ingestion led to alterations in the globus pallidus (seen in MRI but clini-
cially silent) and in the hippocampus (causing persistent memory problems). In post-
mortem studies of patients who died after MDMA ingestion, other mechanisms of damage were suspected. In the present case there was no hyperthermia and a water intoxication could be ruled out by normal serum sodium concentration. The sudden onset of a suspected generalised seizure could point to cerebral anaesthesia due to asphyxia as another possible reason for the patient’s memory disorder, but the fit was observed and no aspasia or even synkope was reported.

JOSEPH SPATT

ERIC GLAWAR

BRUNO MAMOLI

Ludwig Boltzmann Institut für Epilepsie und
Neuromuskulöse Erkrankungen
Neuropathologie, Vienna, Austria

Correspondence to: Dr Josef Spatt, Neurologisches Krankenhaus Rosenhügel, Riedholzweg 5, 1130 Vienna, Austria

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