LETTERS TO THE EDITOR

A case of cutaneous necrosis during interferon- β 1b (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 1995 interferon- β 1b (B-IFN, Betaferon, Schering) was introduced. After a training session the patient injected 8 million IU 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 literature¹⁻⁵ 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 saline 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 reported¹⁻⁵ 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.

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A

B

Severe necrotising cutaneous lesions in a woman with multiple sclerosis undergoing treatment with interferon β -1b. (A) Multiple necrotic skin ulcers on the sites of injection, with surrounding erythema. (B) Two months after modifying the mixing procedure. The old lesions have healed and there are no new lesions.

A pure amnesic syndrome after MDMA ("ecstasy") ingestion

Because of the widespread use of 3,4-methylenedioxymethamphetamine (MDMA) as a recreational drug (ecstasy), incidences of MDMA intoxication are of high clinical relevance. Whereas acute and even chronic psychosis,¹ hyperthermia with sometimes lethal outcome,² and hepatic, cardiac, and nephrological complications after ingestion of MDMA are well documented, persistent effects on memory function in humans have not been reported so far. This is especially remarkable, because of the known potential of MDMA to interfere with serotonin and dopamine brain metabolism and its established neurotoxicity in animals.³

We present the data from a 26 year old woman who developed a pure amnesic syndrome after exposure to MDMA. Other than MDMA the patient had taken no other drugs during the months before onset of her symptoms. In the past she had had occasionally taken cocaine and heroin, but she had stopped heroin misuse one year and cocaine some months before onset. She did well at night school, worked part time in an

office, and her organic and psychological state was inconspicuous according to her and her parents' reports. There was no indication of an unusual nutritive behaviour. Her own and her family's histories were unremarkable concerning seizures and psychiatric diseases. Three days after having taken half a tablet of ecstasy while taking part in a "rave", she was brought to an emergency ward in a fearful state, which was interpreted as an MDMA induced psychotic episode. Her mother reported that the episode had started with loss of consciousness and—as far as she could remember—clonic movements of her arms and legs. Afterwards the patient was disoriented in time and place, and showed regressive infantile behaviour and fear. Routine blood examinations, in particular serum sodium, done immediately after admission, were normal. Blood and urine concentrations of drugs three days after onset were negative, as were the results of EEG, ECG, CT, and HMPAO-SPECT. Neuroleptic treatment with clozapine was started. The psychotic episode soon resolved and neuroleptic therapy could be tapered but the patient complained about ongoing memory problems,