We report a 41-year-old woman who underwent a craniotomy and removal of a left clinoidal meningioma invading the cavernous sinus in May 1989. A graft of cadaveric dura mater (lyodura) was used as plastia, covering part of the left temporal lobe. A small tumoral fragment remaining was treated with radiotherapy. She stayed asymptomatic until October 1992 (41 months after surgery) when she began a rapidly progressive clinical deterioration (ataxia, dysarthria, pyramidalism, myoclonus and generalized slowing on the EEG). Two months later, a brain tissue frontal biopsy (fig. 1) led to the diagnosis of spongiform encephalopathy, thus suggesting Creutzfeldt-Jakob disease (CJD). The patient went into a progressive coma dying in February 1993.

The first description of a transmissible, person-to-person form of CJD was reported in 1974 in a patient who had received a corneal transplant [1]. Thadani et al. [2] published the first report of CJD transmitted by a cadaveric dura mater graft in 1988. Since then, 14 more cases [3–5] including ours have been reported (table 1). All of them, except 1, related to a commercial dura mater (lyodura) made by Brown Melsungen Company of Germany.

CJD can be transmitted through particles containing an abnormal form of a prion protein. These infectious particles are highly resistant to most of the common sterilization procedures. The Committee on Health Care Issues of the American Neurological Association [6] recommended 1-hour exposure to 1 molar NaOH, or steam autoclaving for 1 h at 132 °C as the standard sterilization procedures for CJD tissue or contaminated material. After May 1987, according
to Braun Melsungen AG, their procedures of collection and processing of dura mater were revised in order to reduce the risk of CJD transmission, including the manufacturing process of exposure for 1 h to 1 molar NaOH. However, some authors [7, 8] have emphasized that complete inactivation of the agent producing CJD cannot be obtained, and that such treatment merely prolongs the incubation period.

The epidemic proportion of this group of patients with CJD transmitted by dura mater graft could be larger if we think of the potential transmission of the disease to patients with severe head injuries and malignant brain tumors who, due to the short period of survival after surgery, do not surpass the theoretical incubation period of CJD. These groups of patients are the major working load of any neurosurgical department.

Brown et al. [9] have shown in a recent work that allelic homozygosity at polymorphic codon 129 was present in 92% of the patients with CJD studied. This supports the thesis that the genotype of codon 129 enhances the susceptibility to CJD iatrogenic infections.

We agree with others [10] that despite the assurance on the safety of the product treated with 1 molar NaOH, this homograft source should be abandoned, and we advocate preferential use of temporalis fascia, fascia lata, pericranium or synthetic substitutes.

References