

The MR Centre

Trondheim Regional Hospital, N-7006 Trondheim Norway, tel. +47 73 99 76 70

Name: Tordis Klausen

Date of birth: 5 April 1942

Norwegian National ID No. 050442 47897

Address: N-3810 Gvarv Norway

Clinical issue:

See request

Request for evaluation in the outpatient clinic

Requesting dept./hospital: Doctor in private practice

Date: 19 December 1996

Code: 90-138.899

18 Nov. 1996 KÅT/tk

5 April 1942 Klausen, Tordis

13 Dec. 1996

CEREBRAL MRI:

1,5T. Sagittal FSE 2440/96 and transversal FSE 6780/105 and coronary FSE 3500/80. Transversal SE 2000/20/100.

Frontally, the patient has somewhat extended subarachnoid space, also laterally in the cerebellum. Furthermore one sees a slightly reduced signal in the putamina laterally, which may also be an expression of degenerative changes.

We have seen similar changes in patients exposed to solvents and also in patients with pronounced neurological symptoms which one has been able to correlate with amalgam intoxication, published

MRI: Suspicion of degenerative changes in the putamina. Slight cortical atrophy.

[Initials]

K.Å. Tuomas

Professor of Radiology

City of Oslo
Sunnaas Hospital
VRK/Poliklinikk

[Dept. of Evaluation, Counselling and Control, Outpatient Clinic]

NOTES FROM THE OUTPATIENT CLINIC

For: The patient herself,
Referring doctor [REDACTED]

Re patient: Ms Tordis Klausen, date of birth 5 April 1942, N-3810 Gvarv

Diagnosis: G 92. Toxic encephalopathy
[REDACTED]

Outpatient examination: 2 February 2000

Date: 2 February 2000
Ref.: JE/mv

General background:

Married, almost 58-year-old woman. Previously employed as a dentist's assistant with 23 years of job experience. Regularly exposed to copper amalgam. As of today, disabled.

Previous diseases:

On the whole well.

Present diseases and present situation:

At the end of the 1970s, the patient started developing general symptoms with increased fatigue, memory problems, neuromuscular pain, and sight disturbances which grew worse over a period of two to three years. She has tried to establish an occupational injury relationship through the legal system, but the matter has ended up with a psychiatric diagnosis with an emphasis on panic anxiety. Extensive neurological examinations have been carried out with CT, MR, cognitive evaluation with neuropsychology. The patient has also been to Sweden for an assessment in the field of occupational medicine, including loading tests, with clearly pathological findings, according to the patient. As of today, a picture with left-sided hemilateral symptoms is described, with numbness, problems with balance, reduced colour perception left eye, myokymia left foot. Rotary dizziness with tinnitus and radicular pain of variable intensity and localisation to C6/C7 dermatoma, left upper extremity. Imaging diagnostics give rise to a suspicion of central nervous atrophy, also changes in the basal ganglia, latent squint with convergent deficiency in eye motility, and the patient uses prismatic correction.

Having been asked to evaluate the contribution of occupational exposure at the municipal dental clinic of Boe in Telemark, Norway to the present state of illness shown by Tordis Klausen, I would like to make the following statement.

Chronic mercury poisoning can cause these clinical signs and the symptoms from the central nervous system. No other single cause can explain all clinical observations. I therefore conclude that chronic mercury poisoning is the most likely cause of Tordis Klausens brain injury and resulting functional insufficiency. I estimate the probability for this cause to be more than 75%. The way this case been handled may have hampered her rehabilitation and therefore also contributed to her functional disability.

Danderyd, November 5, 1998

Maths Berlin, MD
Professor Emeritus of Environmental Medicine, University of Lund.

Translated from Swedish to English by the author January,25, 2001.



Maths Berlin