tion syndrome has a poor prognosis, with a 50% mortality rate. The nervous system is seldom involved in the syndrome. If such involvement appears, it usually does so towards the end of the course of the disease. A patient with sensorimotor neuropathy related to axonopathy and occasional demyelination has been recently reported, but in the context of a fulminant illness.3

The distinctive feature of our finding is the occurrence of transient cranial nerve involvement as the probable first sign of macrophage activation syndrome. It could be claimed that the symptomatology is related to the lymphoma. However, very little is known about neurological complications in T-cell lymphoma, and their occurrence is probably rare.2 Kaufman et al.4 have reported an involvement of the nervous system in 14 patients out of 104 cases, eight being related to direct complications. In one only patient, palsy of the sixth cranial nerve was the first sign. Neurological signs occurred between 10 and 102 weeks after diagnosis of the lymphoma.

If polyneuropathy occurs in T cell lymphoma it is due to infiltration and the clinical evolution is usually stereotyped with slowly evolving sensorimotor signs.2 Because there is no invasion of cranial nerves, infiltration of peripheral nerves cannot be eliminated; but it is unlikely, considering the improvement in neurological signs. Meningoradiculitis could be evoked, but if that was so, there would have been a worsening of the initial signs.4 Moreover, CSF examination and cerebral MRI were normal. All these indications lead us to suggest that the neurological signs in our patient could be related to a remitting/relapsing neuropathy due to non-cutaneous T cell lymphoma infiltrating peripheral nerves, to vasculitis or, more likely, to the neurotoxic effects of corticosteroids. Cytokines, especially TNF, are secreted in large amounts in macrophage activation syndrome, and TNF can induce general side effects and cerebral damage.

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had a voracious appetite and indiscriminate eating habits which included paper towels, plants, styrofoam cups, and even faeces. At one point he even ate his own purse from a carry bag. The patient was no longer his usual assertive self and had become quite docile. He tended to wander about the ward touching objects or people and made inappropriate sexual advances. At the time of the examination, his speech was dysarthric but fluent, and he had difficulty with the comprehension of multiple step commands. He had muscular spatial orientation, and difficulty with constructions. The patient could not recognise colours, shapes, or objects by visual presentation; however, he could match the objects visually and name them by touch. He had normal visual acuity and visual fields, mild right hemiparesis, and right sided hyporeflexia. Brain CT showed a single hypolucency in the left temporo-parietal area.

The source of his behaviour change was considered to be a combination of postanoxic and epileptic injury involving both anterior temporal lobes. On the day of his death, the patient had a respiratory arrest after stuffing his mouth with surgical sponges. He had wandered about the ward picking up whatever he could find and putting it into his mouth. Neuropathological examination disclosed an infarction in the left posterior cerebral artery territory, virtual absence of the left anterior temporal lobe, and atrophy of the right parahippocampal gyrus, hippocampus, and amygdala.

Patient No 2 was a 54 year old man with dementia who developed aggressive food seeking behaviour. He would go from room to room, take food from others' trays, and rapidly eat it. His first symptom of dementia was at age 50 when he displayed uncharacteristically poor judgment by exchanging a brand new car for an old one. Over the next four years, he had a progressive deterioration of judgment and memory. In addition, the patient became placid, began manually exploring his surroundings and grabbing at objects, and occasionally exposed himself to others. On examination, he was passive and mute, except for the short phrase responses and echolalia. His memory was impaired, but calculation and construction abilities were preserved. He could visually recognise and name colours and objects. He had normal visual acuity, visual fields, motor testing, and reflexes.

The patient's CT showed disproportion-ate frontal lobe atrophy, and the patient was diagnosed with frontotemporal dementia complicated by the Klüver-Bucy syndrome. On the day of his death, he was seen to develop a breathing difficulty after a large meal. A Heimlich manoeuvre was performed, and he vomited a large amount of undigested food. There was massive aspiration of poorly masticated food in the pharynx, and the patient did not respond to resuscitation efforts. Neuropathological examination showed pronounced frontal lobe atrophy with Pick cells but no Pick bodies, and milder anterior temporal atrophy.

These two patients illustrate that hyperoral behaviour can be lethal. Both patients had the Klüver-Bucy syndrome with a tendency to engage in oral exploration of objects and hygienic, or an oral exploration of inanimate objects. In this syndrome, the hyperoral behaviour probably results from damage to the amygdalae in the anterior temporal lobes. Patients with frontotempo-ral dementia, as in patient No 2, are prone to this syndrome, but the Klüver-Bucy syn-drome has many aetiologies including trauma, strokes, ischaemia, and epilepsy, as in patient No 1.

Another neurological causes of hyperoral behaviour may pose a danger to patients. Lesions of the ventromedial hypothalamic "satiation centre", such as hamartomas and germinomas, can lead to hyperphagia. The Kleine-Levin syndrome presents with periodic hyperphagia and hypersomnia, and there is one report of a patient with this syndrome who asphyxiated on a sausage. Additional causes of hyperphagia include bilateral thalamic infarcts and congenital disorders such as the Frader-Willi syndrome and the Laurence-Moon-Biedl syndrome.

Neurologists, psychiatrists, and others who manage these patients need to be aware of the danger of death from asphyxiation or aspiration. Close supervision and other preventive measures are indicated to avoid this complication in patients with the Klüver-Bucy syndrome and related neurological dis-orders.

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Anxiety disorders in non-demented and demented elderly patients: prevalence and correlates

Anxiety disorders account for only a fraction of admissions to psychiatric hospital of patients over the age of 65 and a decline in anxiety disorders has been reported in outpatient settings. The rate in which patients have used community based samples have reported mixed results, although a vast majority found a decrease in prevalence in the elderly population. This presents an interesting problem, as factors reported to be highly associated with anxiety disorders such as decreased physical health, bereave-ment, isolation, and decreased autonomy are more likely to increase than decrease. A possible explanation for this anomaly might be that age related biological factors reduce the sen-sitivity of older people to these risk factors.

Altogether depression is a common disorder in elderly people which is often accompa-nied by psychiatric symptoms there are few studies examining the association between dementia and anxiety. To our knowledge the only published study has reported an increased rate of anxious mood in demented persons. However, this study made no diag-noses of anxiety disorders and the patients were all at an early stage of dementia.

The aim of the present study was to esti-mate the prevalence of anxiety disorders in non-demented and demented subjects and to identify some of the psychosocial factors associated with this in an elderly population. The participants came from a population based study in Stockholm, Sweden.

We used data from the follow up phase of a longitudinal investigation of adults aged 78 years and over residing in the Kungsholmen parish of Stockholm, Sweden. A total of 1101 persons comprised the study popula-tion and a more detailed description of the popula-tion and the methods used has been reported elsewhere.

Information regarding psychiatric symp-toms was derived from psychiatric examina-tions conducted by the psychiatrists using the comprehensive psychopathological rating scale (CPRS). The psychiatrists were trained and attended regular meetings to ensure that the CPRS was administered in a consistent manner. Information on psychiatric his-tory and physical health was obtained by direct examination of the participants, inter-view with informants, and previous medical records. Disabilities in daily activities were assessed using the Katz index, which is a hierarchical scale (0–6) measuring independ-ence in six activities of daily living. Impairment in activities of daily living (ADL) was determined by both the psychiatrist using the DRS. The Swedish version of the mini mental state examination (MMSE), a global measure of cognitive functioning, was administered to participants, with a maximal score of 30. The ADL was made using the DSM-III-R criteria to maintain accuracy with previous phases of the study. The severity of dementia was classified according to the Washington clinical dementia rating scale (CDR). The psychi-atriac diagnoses were combined in three groups: depressive, psychotic, and anxiety disorders. DSM-IV criteria were used with the categorisation that every patient was passive—dementia—the diagnosis was made. In addition to dementia only one axis I diagnosis was made. If the person had more than one diagnosis on axis I only the most clinically significant was regis-tered.

To analyse differences in the prevalence of depressive, psychotic, and anxiety disor-ders, a one way analysis of variance was performed with sex and severity of dementia as the between subjects factor. Odds ratios (ORs) and 95% confidence intervals (95% CIs) were computed to analyse differences between subjects with and without anxiety disorder.

Psychiatric information was available for 966 of the 1101 persons. Missing data were mostly due to severe cognitive impairment. Of the 966 participants 740 were women and 226 were men. The mean age of the sample was 84±4 (SD 4±3) years and the mean MMSE score was 25±5 (SD 4±5). There were 786 non-demented participants, and 180 were diagnosed with dementia. Of the 180 demented persons, 58 were diag-nosed as questionable, 84 as mild, 31 as moderate, and seven as severely demented.

The table shows the prevalence of anxiety, psychotic, and depressive disorders with the population divided according to severity of dementia. Due to small numbers moderate (n = 31) and severe (n = 7) dementia


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