Forms of dementia, their effects on cognition and consequences for behaviour

Short introduction to module

Dementias are one of the most common psychiatric-neurological disorders of older age. Dementias are usually accompanied by a decline in so-called higher mental abilities such as memory, orientation, thinking and judgement or language. In most cases, they are chronic or progressive. In the course of the disease, behavior, emotional control and personality change, so that the usual social daily life is increasingly impaired.

According to ICD 10 (Dilling et al. 2004), these symptoms must have existed for at least 6 months; other clinical pictures such as depression or delirium must be excluded as differential diagnoses.

Alzheimer's dementia

Alzheimer's disease is an organic brain disease. It is named after the German neurologist Alois Alzheimer (1864 - 1915), who first described the disease scientifically in 1906. The most common clinical presentation of dementia is due to Alzheimer's disease. The disease is considered the main cause of all dementias, accounting for over 60 %.

Alzheimer's disease is a slowly progressive degenerative process with a presumed duration of 20-30 years. The diagnosis is only made during the course of the disease when the first symptoms appear. From this point on, the average duration of Alzheimer's disease is about 5-8 years until death. The greatest risk factor for the development of Alzheimer's disease is age. Only in rare cases are those affected younger than 60 years of age.

The time courses vary greatly from person to person and depend, among other things, on the age at which the diagnosis is made. For example, a 60-year-old can live with the disease for 20 years, while an 85-year-old has a much shorter life expectancy.

Typical changes in people with Alzheimer's dementia are:

- Impairment of new memory, episodic and semantic memory
- language is increasingly affected in fluency, word finding and information content
- Impairment of recognition
- Practical skills such as planning movements and recognition are lost
- Deterioration in coordination and visoconstruction (performing sequences of actions in the correct order).
- Changes in personality,
- Drive, interest and initiative decline
- Attention is lost, divided attention is limited
- Orientation in the various sub-areas is lost

Neurobiological foundations

Macroscopic changes

The clinical symptoms of Alzheimer's disease are caused by a progressive loss of nerve cells. The consequence of this is the shrinkage of the brain by up to 20%, and an associated deepening of the convoluted furrows on the surface of the brain as well as a widening of the cerebral ventricles. In intermediate and advanced stages of the disease, this shrinkage can be visualised by imaging techniques such as computer tomography (CT) or magnetic resonance imaging (MRI). These examinations can help delineate other diseases that present with a similar clinical appearance. These include cerebrovascular diseases, degenerations of the frontal brain, Lewy body disease and Parkinson's disease.

Microscopic changes

The loss of nerve cells occurs not only in the cerebral cortex, but also in deeper brain structures. The loss of nerve cells also destroys the transmission points between the nerve cells that serve to transmit and process information. At the same time, there is a proliferation of supporting cells. A deeper-lying brain structure that exhibits nerve cell death particularly early is the Meynert basal nucleus, whose nerve cells produce the transmitter acetylcholine. As a result of the death of cells in this nucleus, there is a considerable reduction of the transmitter substance in the cerebral cortex. This change causes disturbances in information processing and is causally involved in memory loss. The typical feature of Alzheimer's disease is that the death of nerve cells is accompanied by the formation of abnormally altered protein fragments which are deposited in the brain in the form of fibrils. These are, firstly, the neurofibrillary tangles described by Alois Alzheimer. These tangles, which can be detected within many nerve cells, consist of tau protein, a normal component of the cytoskeleton. In Alzheimer's disease, however, the tau protein becomes excessively loaded with phosphate groups. This causes disturbances of stabilisation and transport processes in the cell, which ultimately lead to the death of the cell. The second pathological protein deposit characteristic of Alzheimer's disease are the plaques found between the nerve cells. They consist of a central amyloid core surrounded by pathologically altered nerve cell processes and supporting cells. In many patients, the amyloid is also deposited in the wall of small blood vessels. This worsens their permeability and leads to disturbances in the supply of oxygen and energy to the brain. Amyloid is a cleavage product of a larger protein molecule, the function of which is not yet precisely known.

Core symptoms

Memory disorder

Memory impairment is the obligatory and dominant symptom in the pre- and early stages of Alzheimer's disease. If there is no significant memory impairment, the assumption of Alzheimer's disease must be doubted. The term "short-term memory" is often incorrectly used for the function of the new memory. In fact, short-term memory, i.e. the ability to hold content in consciousness for a few seconds, is undisturbed in the early stages of Alzheimer's disease (Kensinger et al. 2003).

Clinically, the new memory disorder manifests itself in the forgetting of new experiences, conversation content, facts and plans. Patients and relatives report, for example, that details of journeys or events are no longer remembered, that questions and statements are repeated, that the plot of films and books cannot be followed, agreements are forgotten, the parked car can no longer be found or no orientation can be gained in a strange place. Some patients develop an amnesic syndrome early on, i.e. they can barely retain new content for 1 minute, repetitively ask the same questions at short intervals, think they are still in their old place of residence after moving, forget that they recently ate.

Temporal and spatial disorientation

An early consequence of memory impairment is also temporal disorientation, as the duration and sequence of events are remembered blurrily. Frequent misplacement of objects such as keys, savings books and cash can lead to paranoid reactions.

Disturbance of the old memory

Old memory is the sum of all acquired knowledge about facts (semantic knowledge) and events (episodic knowledge). A pronounced disturbance of the old memory is called retrograde amnesia. With the spread of Alzheimer's pathology in the neocortex, the contents of the old memory are also increasingly lost; retrograde amnesia thus sets in. Content from the recent past is the first to be affected, while old knowledge from childhood and adolescence is highly consolidated and therefore relatively resistant. In the middle and late stages, however, patients with Alzheimer's dementia increasingly lose knowledge about their living environment and biography. With this process, Alzheimer's disease progresses from a disabling disease to a loss of identity and personality. Patients sometimes live in the past, think that people who died long ago are alive and search for them, no longer recognise their home and their elderly relatives, and may be frightened by their own reflection in the mirror. Possible consequences are alienation, fear and agitation.

Disturbance of the visual-spatial thinking

This affects the ability to understand spatial relationships, to grasp the topology of objects and to decipher the structure of signs and symbols. As a result, there are serious deficits in everyday activities such as calculating, writing, reading clocks, doing handicrafts, repairing, putting things away, reading maps and traffic signs, filling out forms, finding their way around buildings, etc. Naturally, more complex and less practised activities, such as drawing, are affected first.

Course of the disease

Early stage

However, patients with Alzheimer's disease in the early stages are often still alert, approachable, able to vibrate and hardly changed in their personality. In simple dialogue, their speech and thinking speed is not or only slightly reduced. The emotional sphere and drive can also remain relatively intact for a long time. Many patients therefore appear little conspicuous in superficial contact ("good facade") until the middle stage. In the early stage, Alzheimer's disease tends to have the status of a person with reduced abilities but a largely intact being.

On the somatic level, there are no deficits in the early stages. On the psychiatric level, depression may be present in the preliminary or early stages. Since it can occur even before the diagnosis is made or suspected and in patients who have never been depressed before, it is obvious that this depression is of organic origin, i.e. results from degenerative changes, e.g. in the serotonergic system. It is not uncommon for the first recognisable symptom of Alzheimer's disease to be delusional-paranoid symptoms.

Patients and their relatives often tend not to notice dementia symptoms by playing them down, denying them or bypassing requirements.

Middle stage

In the middle stage of the disease, the core symptoms of dementia, i.e. the cognitive deficits, become unmistakable. Patients now show the following symptoms:

- pronounced memory deficits with a resulting disturbance of temporal orientation
- clear disturbances in visual-spatial thinking, e.g. when writing and reading the clock
- Increasing disturbance of the ability to express oneself, with slurred, imprecise speech interspersed with word-finding disorders
- Deficits in manual dexterity (dyspraxia)

As a result, they suffer increasing limitations, even in simple activities of daily living, such as personal hygiene, dressing, orientation in nearby spaces, preparing meals and drinks. The continuity of experience and memory is gradually lost. Thinking is often directed towards the old past. In addition to cognitive tool performance, basal mental functions are also reduced, such as drive, activity, attention and speed in thinking, acting and speaking. The sick are dependent on care and can only be left alone for short periods of time. On a psychiatric level, paranoid delusional symptoms can develop, e.g. being stolen from, poisoned or cheated. They can partly be traced back to the cognitive deficits, e.g. when hiding places for money, passbook and keys are forgotten. Pre-existing problematic traits can become accentuated. Depression may persist or be new. Depression and cognitive deficits can cause listlessness and apathy. The loss of control over reality and the experience of one's own change can cause anxiety and restlessness. There is often an urge to move and a tendency to run away. Fear and not understanding the situation can lead to agitation, anger and aggressive behaviour. On a somatic and vegetative level, there is often a disturbance of the sleep-wake rhythm with nocturnal restlessness, getting up, walking around or moving about in the home. Control over urination decreases; incontinence may occur.

Late stage

In the late stage of the disease, patients can no longer manage even simple activities of daily living without help and are completely dependent on care and nursing. The continuity of experience is lost; the loss of language progresses. The patient's own home and relatives can become unfamiliar. A regular sleep-wake rhythm often requires the administration of sedating medication. Formerly agitated patients may become calmer and depression may fade into the background. However, anxiety, restlessness, sleep disturbance and irritability can also increase, so that a normal daily routine and care at home are hardly possible any more. On a physical level, there is now usually urinary incontinence; gait unsteadiness and dysphagia may set in. Due to motor restlessness, dysphagia and lack of appetite, many patients progressively lose weight. Apathy, mutism, cachexia and immobility are characteristic of the final stage. Death occurs as a result of cachexia or intercurrent diseases.

Vascular dementia

Vascular dementia" is caused by a disturbed blood supply to the brain tissue. Depending on the mechanism of this circulatory disorder, doctors distinguish between different forms of vascular dementia. Accordingly, there is, for example, multi-infarct dementia, which is caused by several small

brain infarcts (strokes). Other forms are subcortical vascular dementia and mixed (cortical and subcortical) vascular dementia.

In Western populations, the various forms of vascular dementias together represent the second most common cause of dementia at around 10-30%. In Asia, vascular dementias seem to be the main cause of cognitive decline, accounting for about 50% of dementias.

Symptoms

People with vascular dementia find it difficult to speak coherently, listen attentively and orient themselves. They often appear confused as a result. Drive and concentration disorders as well as mood swings also occur. The latter can manifest themselves, for example, in the fact that those affected switch very quickly between laughing and crying (often without any corresponding emotion).

Vascular dementia is also associated with focal neurological deficits (caused by the cerebral infarctions): for example, hemiplegia and increased muscle reflexes may occur. Disorders of bladder emptying (micturition disorders) in the form of an imperative urge to urinate or incontinence are also possible.

Despite the diversity of the vascular disease, some features can be classified as characteristic. For example, gait disturbances with small-stepped, shuffling or even spastic gait patterns usually occur in the early stages of the disease. This results in frequent falls. Urge incontinence can occur in the early stages. Paralysis, akinesia (lack of movement), speech and swallowing disorders can also occur. But a pronounced emotional instability can also be the result, which manifests itself in uncontrolled laughing or crying (Haberl and Schreiber 2005).

Personality and social behaviour are not affected by vascular dementia. Memory skills are often only slightly affected by the disease.

Causes and risk factors

Vascular dementia results from reduced blood flow in the brain (cerebral ischaemia), which causes nerve cells to die. Various mechanisms can trigger such ischaemia:

The classic form of vascular dementia is multi-infarct dementia: it develops when several brain infarcts (ischaemic strokes) occurring simultaneously or staggered over time cause a critical mass of nerve tissue to die.

In other cases, vascular dementia is caused by a single, sometimes small, infarct in a strategic location (such as the thalamus) that leads to a disruption of pathways.

The circulatory disorder can also be caused by a thickening of the walls of small blood vessels that supply deeper areas of the brain with blood. This results in small infarcts (lacunae) and damage to nerve fibres (medullary layer damage). Doctors speak of subcortical vascular encephalopathy (SVE).

In some patients, vascular dementia is the result of minor or major brain haemorrhages. This is called "hemorrhagic dementia".

There are also other, rarer forms of vascular dementia.

Various factors favour vascular dementia. These include, for example, high blood pressure, heart disease, diabetes mellitus (diabetes), high cholesterol, obesity, lack of exercise and smoking.

Frontotemporal lobar degenerations

Frontotemporal lobar degenerations (FTLD) represent a clinically, neuropathologically and genetically heterogeneous group of diseases characterised by atrophy preferentially affecting the frontal and/or temporal lobes. Three clinical syndromes are distinguished according to the localisation of the neurodegenerative process and thus according to the symptoms:

●Frontotemporal dementia: In this form of dementia, the most common clinical manifestation of FTLD, the neurodegenerative process is primarily focused on the frontal lobe.

●Semantic dementia. This is caused by a bilateral, often asymmetrical, left-focused atrophy of the anterior temporal lobes.

●Progressive non-fluent aphasia: This is characterised by neuronal cell death mainly of the frontal inferior gyrus, premotor cortex and island of the language dominant hemisphere.

Frontotemporal dementia

With an insidious onset of the disease and gradual deterioration, patients appear increasingly superficial and disinterested, accompanied by a loss of empathy and simultaneous flattening of affect. Social contacts are abandoned; drive is reduced. The otherwise occurring behavioural conspicuities, however, differ greatly in type and extent, both between patients and in the individual patient in the course. In some patients, disinhibition and aloofness are in the foreground and lead to the sick person behaving tactlessly and socially inadequately. Not infrequently, disinhibition also leads to conspicuous or dangerous behaviour in road traffic. Studies have shown that patients with behavioural variants of frontotemporal dementia and semantic dementia are more aggressive and risky drivers - in contrast to patients with Alzheimer's disease, whose driving style also changes, but who tend to drive slowly, over-cautiously and unsafely.

Some patients with behavioural variant of frontotemporal dementia are characterised by irritability and aggressiveness. Excessive food intake, including sweets, is not uncommon during the course of the disease; some patients also show increased alcohol and/or nicotine consumption. Changes in eating habits to the effect that certain foods are preferred may occur. Motor restlessness with an urge to move as well as ritualistic or stereotypically repetitive behaviours, which are usually more complex than the movement patterns described in patients with Alzheimer's dementia, sometimes occur in the early stages of the disease. For example, some patients insist on regular activities at a certain time of day. Sometimes patients collect more or less meaningful things up to rubbish. Some patients fixate on puzzles, crosswords, Sudoku or even games of chance. Some patients show hypochondriacal fixations; they complain frequently of physical complaints, some of which seem strange. Typically, the patients' insight into their illness is significantly reduced, often even completely lost.

The cognitive disorders of the behavioural variant of frontotemporal dementia primarily affect the areas of attention and executive functions. The latter is a collective term for all higher-order mental processes that enable planned, goal-oriented and effective action. The speech drive is usually reduced, although in individual cases a pronounced urge to talk can be observed. Temporal and spatial orientation as well as visual-spatial abilities are usually only slightly impaired for a long time in the course of the disease. Memory performance may be largely unimpaired in the early stages of the disease and is usually better than that of patients with Alzheimer's dementia in the course of the disease.

Movement disorders are not part of the typical picture of the purely behavioural variant of frontotemporal dementia; however, extrapyramidal motor symptoms can occur in the course of the

disease, especially bradykinesis and postural and gait disorders. Patients are sometimes urinary incontinent early in the course of the disease, and later also faecal incontinent.

Semantic dementia

Semantic dementia is characterised by a disturbance of semantic knowledge as a result of a gradual loss of semantic memory. Patients lose knowledge of the meaning of words, names, faces, objects, etc. Severe disorders of naming and language comprehension occur.

Semantic dementia is characterised by fluid aphasia; speech is inconspicuous in form, speed and quantity for a long time. The word-finding disorders that occur are bypassed or replaced by filler words and phrases. The language becomes increasingly empty of content despite preserved fluency. Semantic paraphasias occur rather discreetly in spontaneous speech, but are obvious in tests of naming early in the course. Gradually, the vocabulary (both active and passive) is reduced until finally only filler words and phrases are left, which the patients often perseverate stereotypically.

In terms of content, the language eventually becomes almost incomprehensible; however, the patients themselves do not seem to notice this. Word fluency is reduced, but phonematic word fluency is relatively better than semantic word fluency. The ability to repeat is good for shorter words; with longer words, it is noticeable that the repetition takes place without semantic knowledge. In reading, for the same reason, "surface dyslexia" occurs when patients are asked to read aloud a word pronounced differently than written (e.g. "level"); they read letter by letter. Another typical feature of semantic dementia is pronounced difficulties in understanding language. Above all, the understanding of individual words is disturbed, whether presented orally or in writing. As the language comprehension disorder increases, the patient's ability to participate in conversations is reduced. In semantic dementia, the loss of semantic knowledge is accompanied by a disturbance in object recognition. While objects that are in the patient's everyday use are still used without problems in the beginning, some patients show an inability to use less everyday objects (e.g. a stapler) correctly at an early stage. This disorder can progress until the patients can no longer do anything with scissors, a spoon or a brush. Due to the disturbance of conceptual knowledge, patients also have difficulties in recognising e.g. auditory, gustatory or otherwise presented material, whereas the recognition of numbers and colours is apparently largely intact for a long time. Episodic memory is well preserved, at least at the beginning of the disease. Orientation and visuo-constructive abilities are also largely normal for a long time. Sometimes already with the onset of the first linguistic symptoms, but at the latest in the further course, changes in social behaviour and affect also occur in semantic dementia, similar to the behavioural variant of frontotemporal dementia. Here, apathy, irritability and a loss of emotional warmth are the main symptoms. Patients seem increasingly constricted to themselves and certain interests; in some patients, excessive frugality becomes apparent. Sometimes a change in eating habits is noticeable, which manifests itself, for example, in the exclusive eating of certain foods. In the course of the disease, compulsive, perseverative behaviour (solving crossword puzzles, cutting out advertisements from the newspaper) often appears, together with increasing disinhibition, which can lead to tactless, dissocial and even delinquent behaviour.

Progressive non-fluent aphasia

In the foreground of the insidious onset of the speech disorder are pronounced word-finding disorders, often accompanied by pauses in which the patients search for the right word. Word finding is disturbed at the level of phonematic access; this is described by the patients as a "tip-of-the-tounge" phenomenon. They know what they want to say, but cannot get the word past their lips. Naming in writing is therefore easier for the patients. Naming verbs is more severely impaired than

naming objects. Accordingly, there are relatively fewer verbs in spontaneous speech. The overall pace of speech is slowed down. In tests of word fluency, patients with progressive non-fluent aphasia (PNFA) perform below average, with phonematic word fluency being relatively more reduced than semantic fluency. Furthermore, grammatical errors and syntactic disorders are found in PNFA. In the course of the disease, a strained telegram style is not uncommon. To varying degrees, there is apraxia of speech in which the programming of speech movements is disturbed, resulting in phonological errors, articulation disorders or phonematic paraphasias. It is not uncommon to also notice a pronounced dysarthria or dysarthrophonia with a disturbance of the normal speech rhythm and accentuation. While speech comprehension for individual words is intact, comprehension for whole sentences and their grammatical structure is limited. The important words in the conversation are recognised so that adequate answers can be given.

Slightly pronounced behavioural problems, most frequently apathy, occur during the course of the disease, but should not dominate the clinical picture over a longer period of time. In the early stages of the disease, patients with PNFA have full insight into their illness; they recognise their deficits and usually also suffer from not being able to communicate easily. In the course of the disease, however, this insight into the disease decreases. It is not uncommon for patients in the advanced stages to develop a marked change in their nature and behavioural problems, so that they can no longer be distinguished from patients with advanced frontotemporal dementia. The speech disorder often progresses in the course of the disease to complete mutism.

Lewy body dementia

Lewy body dementia (DLB) occurs both cortically and subcortically. Lewy bodies are inclusion bodies in the glial cells. This disease can occur together with Parkinson's disease, with Alzheimer's dementia or in a pure form. If Parkinson's symptoms are found early in the course of a dementia, this type of dementia should always be considered.

This has a high clinical relevance in particular because there is a significantly increased sensitivity to neuroleptics.

Frequency and course

Men are affected twice as often as women; overall, 15-36 % of all dementias are said to be of this type. In about 7-20 % of all patients with dementia, there are

Lewy bodies can also be detected in the cerebral cortex (Bürger et al. 2003). This disease progresses at different rates and shows a great fluctuation in symptoms.

Diagnostics and symptoms

A progressive cognitive decline is required for the clinical diagnosis; deficits in attention and in the visual-spatial imagination may be particularly conspicuous in cognitive tests.

Very impressive are the symptoms of dementia with Lewy bodies:

- Attention deficits already at the beginning of the disease,
- visual hallucinations, which can be very detailed (are also experienced fearfully),
- non-visual hallucinations,
- motor symptoms of Parkinson's disease (if present, within one year before or after the onset of dementia),
- Falls already in the early phase of the disease,
- Agitation,
- comparatively early incontinence,
- Syncope,
- temporary loss of consciousness,
- high neuroleptic sensitivity,
- Sleep disorders,
- systematised delusion,
- Hallucinations in other sensory areas.

Changed behaviour

From a nursing perspective, patients with DLB are probably more likely to be perceived as having behavioural problems than others. In particular, anxiety, aggression and delusion can lead to major problems, especially from the perspective that this group of people is very sensitive to neuroleptics.

which are used precisely for psychotic behaviour.

However, the specific problems should also be known when referring people to sociotherapeutic services in order to avoid excessive demands and agitation.

and thus the varying cognitive performances are taken into account.

Dementia in Parkinson's disease

Until the 1970s, the following statement by James Parkinson was valid: "The senses and the mind are undamaged". The primary symptoms of Parkinson's disease are increased muscle tension, reduced movement and often a coarse resting tremor. More recent studies

However, studies show that a high proportion of dementia develops during the course of Parkinson's disease.

If dementia occurs, one third of it is caused by a pathology of the Lewy bodies, but in more than half of the cases it is caused by Alzheimer's disease (Stoppe 2003).

Frequency and course

The proportion of Parkinson's patients with dementia is between 30 and 40 %. Among Parkinson's patients older than 80 years, dementia syndromes are found in almost 70 % (Bürger et al. 2003).

Dementia with Parkinson's represents a significantly increased risk of moving into a nursing home and is associated with a drastic shortening of the otherwise almost normal life expectancy of Parkinson's patients treated with medication (Bartels 2005).

Diagnostics and symptoms

The diagnosis corresponds to that of Parkinson's disease, Alzheimer's dementia or Lewy body dementia. Risk factors or signs for the development of dementia in Parkinson's disease are: advanced age, speech disorders, early onset of L-dopa-induced psychoses and depression. For its part, dementia leads to a relatively early move into a nursing home.

For these Parkinson's patients, this course means being dependent on care at a very early stage.

The psychopathological symptoms observed in these patients vary greatly: depression, apathy, but also irritability, slowing down of thought processes (bradyphrenia) and disorders of attention can occur.

However, disturbances and changes in colour and smell perception can also occur. If anti-Parkinson's therapy is intensified in these patients because of the increase in Parkinson's symptoms, the dementia symptoms often worsen significantly

(Stoppe 2003).

Patients with Parkinson's dementia find it difficult to spontaneously reproduce thoughts, but also to recognise them. It is interesting that linguistic and apraxic disorders are less pronounced than in patients with Alzheimer's dementia.

A particular problem with this clinical picture becomes clear in the question: Are motor impairments functionally caused by Parkinson's syndrome?

explain or by a cognitive disorder? In the first case, the movement exercise would be in the foreground, in the other case the instruction.

Changed behaviour

Behaviour is characterised on the one hand by the cause of dementia, i.e. Lewy bodies or Alzheimer's dementia, but on the other hand by depressive symptoms, which are particularly common in Parkinson's, and mental abnormalities, which can occur as undesirable effects of Parkinson's therapy. For example, delirious states or hallucinations and delusions can develop, which are intensified under Parkinson's therapy.

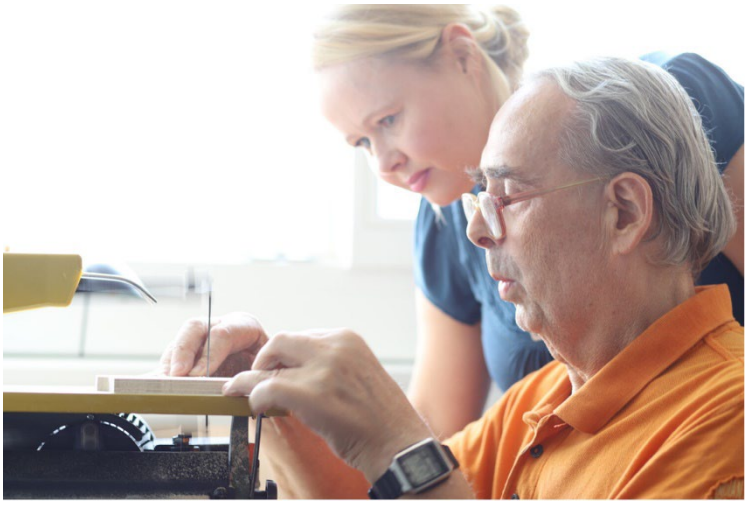
(Stoppe 2003).

Korsakow's syndrome

Korsakow's syndrome (also: amnesic syndrome caused by alcohol toxic influences) appears in particular as a pronounced memory impairment. Those affected have lost the ability to store new information (so-called anterograde amnesia) and show losses of short-term and ultra-short-term memory. At the same time, they develop a tendency to fill the resulting memory gaps and orientation disorders with sometimes freely invented stories (so-called confabulation). However,

many patients are not aware of this, so that the confabulations are not conscious deception or even lies.

Rather, Korsakow's syndrome is an expression of severe, chronic damage to the brain, which primarily affects brain regions responsible for memory formation and the regulation of emotions.



The primary cause of Korsakow's syndrome is years of alcohol dependence or abuse. A metabolic disorder due to a vitamin B-1 deficiency (thiamine) is assumed. Thiamine is needed in nerve tissue for nerve excitability.

Alcoholics have a particularly high vitamin B requirement. On the one hand, because less vitamins can be absorbed through malnutrition as a result of alcohol consumption, and on the other hand, because alcoholics need more B vitamins to metabolise the alcohol. If the brain is insufficiently supplied with vitamin B1, parts of it are damaged and the result is Korsakow's syndrome.

If nerve cell damage persists, alcohol toxic polyneuropathy occurs, which initially manifests itself in one of the legs. However, this can also become noticeable in other areas of the extremities. It leads to a weakening or extinction of the muscle reflexes, to disturbances in sensitivity and to tingling sensations. The polyneuropathy can go so far that the patient is unable to walk. In the central nervous system, the toxic effect of alcohol causes atrophy of the cerebellum. This becomes noticeable through considerable walking disorders and coordination problems.

Dementia symptoms in people with a migration history

It can be assumed that dementia-related illnesses occur with about the same frequency in the population as in the autochthonous population. It is possible that they begin somewhat earlier, as the ageing processes also begin five to ten years earlier than, for example, in parts of the German population of the same generation. In addition to physical stress, there are also psychological stresses caused by living in a foreign country and the stress caused by the often low social and economic status. (Sütterlin, S., Hoßmann, I., & Klingholz, R. (2011)).

People of different countries of origin/ethnicities do not necessarily have the same ideas about symptoms of dementia.

The "10/66 Dementia Research Group" has compared the prevalence of behavioural and psychological symptoms in dementia patients in developing and industrialised countries. The prevalence of behavioural symptoms was comparable. However, there were large differences between their prevalence in India and China, for example - China was the least likely to report them. However, the authors of this study suggest that this lower rate in Asia could also be explained by

cultural barriers to disclosing to strangers. In addition, regional differences in the occurrence of specific symptoms were also found - for example, high rates of agitation, wandering and sleep disturbance were reported among Indian respondents, while high rates of vocalisation were reported in Latin America. The mental syndromes (depression, anxiety neurosis and schizophreniform/paranoid psychosis) were most frequently reported in Latin America and least frequently in China. (10/66 Dementia Research Group, 2004: 454f)

In a survey by Piechotta and Matter, clinic staff stated that dementia was often seen by Turkish migrants as an "inevitable sign of advancing age" (Piechotta & Matter, 2008: 226f). The staff of the specialist centre for migrants suffering from dementia report that in the Turkish migrant community in Germany there is also the idea of dementia as a "mental illness" (Jonas & Helck, 2007: 10). Similarly, staff state that some hodshas (Islamic scholars) in the Turkish migrant community would claim that dementia is a punishment from Allah for (immoral) lifestyles or other misbehaviour (Jonas & Helck, 2007: 10).

A symptom that specifically affects migrants is the loss of second language in the course of dementia. Knowledge of the language of the country of residence, which is usually learned later, is lost in the early stages of dementia. (Hirsch, 2006: 1; Piechotta & Matter, 2008).

Thus, the institutions interviewed by Piechotta & Matter assumed that due to language problems and the widespread perception of dementia as a normal sign of advancing age, diagnostic procedures usually do not even occur (Piechotta & Matter, 2008: 227). Therefore, migrants are often diagnosed at a later stage of the disease than the majority population (Hinton & Levkoff, 1999: 463).

References:

Bartels C. Demenz bei degenerativen Systemerkrankungen. In: Wallesch CW, Förstl H. Hrsg. Demenzen. Stuttgart: Thieme; 2005: 195

Bürger K et al. Neurodegenerative Demenzen. In: Hampel H et al. Hrsg. Alzheimer-Demenz. Stuttgart: Wissenschaftliche Verlagsgesellschaft; 2003a: 235

Dilling H. Internationale Klassifikation psychischer Störungen ICD 10. Bern: Huber; 2004

Haberl RL, Schreiber AK. Vaskuläre Demenzen. In: Wallesch CW, Förstl H. Hrsg. Demenzen. Stuttgart: Thieme; 2005: 221

Hinton, L. & Levkoff, S. (1999). Constructing Alzheimer´s: Narratives of Lost Identities, Confusion and Loneliness in Old Age. Culture, Medicine and Psychiatry, Bd. 23 , S. 453-475

Jonas, I. & Helck, S. (2007). „Alzheimer ist eine Strafe Allahs". Pro Alter, Bd. 39-2 , S. 9-13

Kensinger, E. A., Shearer, D. K., Locascio, J. J., Growdon, J. H., & Corkin, S. (2003). Working memory in mild Alzheimer's disease and early Parkinson's disease. Neuropsychology, 17(2), 230–239

Neppert, D. (2011). „MigrantInnen und Demenz - Ressourcen, Belastungen und Krankheitskonzepte. Eine Rekonstruktion anhand von Interviews mit VertreterInnen von MigrantInnenselbstorganisationen“

Piechotta, G. & Matter. (2008). Die Lebenssituation demenziell erkrankter türkischer Migranten/innen und ihrer Angehörigen. Fragen, Vermutungen, Annahmen. Zeitschrift für Gerontopsychologie & psychiatrie, Bd. 21-4 , S. 221-230.

Sütterlin, S., Hoßmann, I., & Klingholz, R. (2011). Demenz-Report: wie sich die Regionen in Deutschland, Österreich und der Schweiz auf die Alterung der Gesellschaft vorbereiten können.. Berlin: Berlin-Institut für Bevölkerung und Entwicklung.

Stoppe G. Diagnose und Differenzialdiagnose der Demenzerkrankungen. In: Wächtler C. Hrsg. Demenzen. 2. Aufl. Stuttgart: Thieme; 2003: 24

The 10/66 Dementia Research Group. (2004). Behavioral and Psychological Symptoms of Dementia in Developing Countries. International Psychogeriatrics, Bd. 16-4 , S. 441-459.

Short summary of module

Dementias are usually accompanied by a decline in so-called higher mental abilities such as memory, orientation, thinking and judgement or language (Fig. 8.1). They are usually chronic or progressive. In the course of the disease, behaviour, emotional control and personality change, so that the usual social daily life is increasingly impaired.

Around 80 percent of all dementias are caused by diseases of the brain in which nerve cells are gradually lost. These are called neurodegenerative diseases and their causes are only partially known.

Alzheimer's disease is the most common disease, accounting for 60-70 percent of all cases. In addition, vascular dementias, Lewy body disease, Parkinson's disease dementia and frontotemporal dementia are the most common.

According to current knowledge, dementia and its symptoms occur to the same extent in people with a migration history. The diagnosis is often made much later due, among other things, to the perception and acceptance of the symptoms in their respective ethnic background.

Questions for reflection

1. Define the term "dementia
2. Define "vascular dementia
3. Describe psychological changes in a person with vascular dementia
4. Which form of dementia is characterized by aphasia at the beginning?
5. Which form of dementia is particularly susceptible to the side effects of neuroleptics?

6. Summarise the stages of Alzheimer's dementia

7. Which forms of dementia entail an increased risk of falling?

8. Which form of dementia progresses without memory disorders at the beginning?

Imparted competencies

- The carer knows different forms of dementia
- The carer knows different causes of dementia
- The carer knows the symptoms of different forms of dementia and can classify them
- The carer can assign specific behaviours to the different forms of dementia.