

Neurological Diseases caused by Lack of Vitamin B12

By Prof. Joachim Schmidt

Vitamin B12 is an essential water-soluble vitamin which is of crucial importance for the formation of blood, the function of the nervous system and the integrity of the mucous membrane of the gastrointestinal tract. For this reason, a lack of vitamin B12 in humans manifests itself as

- blood formation disorders, specifically the formation of erythrocytes,
- neurological/psychiatric disorders and
- epithelial changes to the mucosa of the digestive tract.

The blood formation disorders and the neurological consequences are particularly clinically relevant, as they can be associated with serious - and in some cases life-threatening - diseases. While blood formation changes are very characteristic and therefore mainly to the fore in the diagnosis of vitamin B12 deficiency, neurological disorders are much more varied and are not recognised to a sufficient extent in clinical practice as consequences of B12 deficiency. Neurological disorders, however, are often the earliest, and in some cases the only, clinical symptoms of functional vitamin B12 deficiency. The data on frequency vary. According to information provided by the IOM (Institute of Medicine, Washington DC), 75 to 90% of people with clinically relevant B12 deficiency have neurological disorders, and in approximately 25% of cases, these are the only clinical manifestations of B12 deficiency. The neurological disorders may occur together with haematological changes, but also independently of them. Overall, it can be assumed that approximately 60% of patients with pernicious anaemia also display symptoms of funicular myelosis. Of patients with confirmed vitamin B12 deficiency and neurological disorders, around one quarter had no haematological changes. Interestingly, there is also an inverse correlation to the strength of the haematological and neurological disorders. The greater the neurological disorders, the smaller are the haematological changes and vice versa. The causes of this are unknown.

The main aspect of the neurological symptoms is paraesthesia or feelings of numbness in the skin, hands or feet that have "gone to sleep" unsteady gait and coordination difficulties, or even paralysis. These symptoms are the expression of funicular myelosis (funicular spinal-cord disease, subacute combined degeneration (SCD) of the spinal-cord). This occurs on the basis of a combined degeneration of the lateral and posterior columns of the spinal-cord as a result of the defect in the myelin sheaths, and is one of the demyelinating diseases. Demyelination (destruction of the myelin sheaths) occurs in

- the posterior funiculus (which mediates tactile perception and proprioception),
- the lateral cerebellar pathways (which are also used for proprioception) and
- the pyramidal tract of the spinal-cord (which controls movement).

The resulting neuropathy is symmetrical and affects the legs more strongly than the arms. In the majority of cases the neuropathy is a peripheral sensorimotor polyneuropathy, although mononeuropathies (visual and olfactory), autonomic neuropathy (impotence, incontinence) and confined forms (myelopathy and neuropathy) are also possible.

Initially the patient experiences paraesthesia, which initially occurs on the feet - and possibly the hands - and spreads during the course of the disease to the respective limbs (as in neuropathy). The sensory disturbances are followed by motor disturbances (muscle weakness, symptoms of paralysis and impaired locomotor coordination). The nerve conduction velocity of motor and sensory nerves is reduced. If left untreated, the disease would result in paraplegia.

Furthermore, damage to the central neural pathways can also result in cerebral disorders and as a result (and additionally) psychiatric symptoms.

The symptoms of cerebral disorders vary and may manifest themselves as confusion, stupor, apathy, impaired memory and judgement, but also take the form of psychoses, depression and dementia. The cerebral disorders are less common than the peripheral disorders, but are underestimated in practice. Catatonia has also been described as a psychiatric form of vitamin B12 deficiency.

Particular attention is also required with respect to the clinical manifestation of B12 deficiency in infants and small children whose mothers have had vitamin B12 deficiency. This may result in serious haematological and neurological disorders in the infants, which may be associated with lasting consequences for the children's development.

The aforementioned diseases can - but do not necessarily - be a consequence of vitamin B12 deficiency. If such neurological disorders occur, the possibility of vitamin B12 deficiency should always be borne in mind. Treatment with vitamin B12 in these cases can bring about a very rapid improvement in the symptoms.

Prof. Dr. J. Schmidt
October 8, 2011