Neuropsychiatric complications of alcoholism

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INTRODUCTION

It is common knowledge that alcohol disrupts the normal functioning of the nervous system and that it is the cause of numerous automobile accidents: alcohol accounts for between 40 and 45% of road fatalities, and the risk of provoking a fatal accident is multiplied by a factor of 4.4 for a blood alcohol concentration of 0.80 g/l and by a factor of 16.2 for a blood alcohol level of 1.50 g/l (15).

The physiopathology of neuropsychiatric complications due to alcoholism is extremely complex, and experimental data are often contradictory.

In general, the clinician has to makedo with the current hypotheses.

NEUROPSYCHIATRIC COMPLICATIONS OF ALCOHOLISM (3)

The syndrome of severance

From a physiopathological point of view, the syndrome of severance consists of a hyperactivity of the central nervous system which was previously inhibited by alcohol. This may be expressed by excessive trembling upon awakening, by convulsive crises (21), by hallucination or by delirium tremens (17).

Animal experimentation shows that severance brings about the development of epileptic points in the periventricular, median and anterior regions.

Trembling also indicates a state of hyperactivity induced by severance (2); in fact, noradrenalin and adrenalin are abundantly excreted during the delirium tremens and an elevated rate of MHPG (3-methoxy 4-hydroxy phenylglycol) can be found in the LCR.

There is also an augmentation of MHPG found in urinary excretions and a diminution of VMA (vanyl-mandelic acid).
In addiction, a diminution of the rate of GABA (gamma-aminobutyric acid) can be noticed (14).

It is important to note that the metabolical modifications of the syndrome of severance lasts much longer than its apparent clinical effects: these rarely last longer than eight days, whereas a verifiable "subacute syndrom of severance" lasts at the very minimum three months, during which a state of hyperexcitability of the central nervous system can be remarked (for example from a biological point of view, of with the study of evoked potentials).

**Gayet Wernicke encephalopathy**

This is rather common disease of deficiency origin, where the avitaminosis of B1 plays a determining role. Its major cause is chronic alcoholism causing a deficiency in thiamin (vitamin B1), either by a deficiency in diet (anorexia, imbalanced diet rich in carbohydrates and poor in protein) or by improper absorption (gastritis, intestinal difficulties, or difficulties due to a gastrectomy) (5).

Surface lesions have a characteristic topography: bilateral and symmetric, found in the structures surrounding the third ventricle, the Sylvius duct, and the fourth ventricle. Mammillary regions are often affected.

Apparent lesions (neural alterations, glial and vascular proliferation) vary in aspect according to their shape: acute or prolonged; hemorrhagic suffusions are common in acute lesions.

With regards to the clinical aspect, everything starts by difficulties in alertness (somnolence, mental confusion, disorientation); followed by hypertonic phenomena of opposite pace, grasping, cerebellar signs, troubles with maintaining equilibrium while standing or walking, and in 20 to 50% of cases, the ocular nerves may be affected, thus bringing to this syndrome all its originality (inter-, supra- or nuclear paralysis, to which a nystagmus may be added).

In the absence of treatment, the fatal evolution of the disease was common in the acute lesions (hypertonic coma with signs of intense neurovegetation) (19).

**Korsakoff syndrome**

This is a particular variety of mnestic trouble, often related to a polyneuritis of the lower limbs, and for which the pronosis is quite serious.

Four cardinal signs characterize this syndrome: temporal-spacial disorientation, anterograde amnesia, fantasizing, false recognition (which furnishes the missing memory) (18, 20).
On the anatomical level, these disorders are related to lesions found in the mammillary tubercles, at the uncus of the hippocampus, at the thalamus, at the level of cerebral structures which constitute Papez's circuit (hippocampo-mammillothalamic) integrator of memory. Once Korsakoff's syndrome is diagnosed, treatment brings about a satisfactory recovery in only 25% of the cases. Nevertheless, a lacune mnesic of variable importance persists. In the other cases, the lack of recognition remains. If a partial recovery may make possible a slow recovery of autonomy, perhaps even the possibility of taking up a part-time job, severe deficits bring about a complete disability and require constant care, usually in a hospital (10).

**Cerebral atrophy**
Most researchers agree that physical dependency on alcohol is frequently accompanied by cerebral atrophy (in more than 50% of cases); in chronic alcoholics, cortical (sulcetasiess) and sub-cortical (ventricular dilation) atrophy is more marked the older the patients (4). It appears rather early, and far precedes the appearance of neurological signs, which, when these exist, are not as significant as signs of atrophy (11). Alcoholic dementia often appears rather early (around the age of 50) and affects women more than men.

**Cerebral atrophy of Pierre-Marie Foix-Alajouanine**
This is often associated with cortical and sub-cortical atrophy and predominates at the level of the vermis, although the cerebellar hemispheres are also affected. It is responsible for significant difficulties in the coordination of movements, predominantly in the lower limbs, with disruption of walking and rendering static equilibrium difficult, difficulties which can not easily be corrected by physical therapy.

**Centro-pontic myelinosis**
This is remarked in undernourished alcoholics as well as in other disorders which have metabolic difficulties (13). The clinical table is that of a flaccid quadraplegic with pesudo-bulbaire syndrome.

**Marchiafava-Bignami disorder**
A patient in an advanced state of alcoholism may experience progressively severe bouts of dementia with hypertonics, dysarthia, epilepsy, and astasia-abasia (6).
Anatomic control shows an intense demyelisation, and then a necrose of the axial region of the corpus callosum with a more limited extension to the cerebral hemispheres, to the antierieur white commissure, to the optic formations and to the average cerebellar peduncle.

**Chronic hepatic encephalopathy**
This is remarked mainly among subjects having undergone a porto-cave anastomosis, whether or not they have repeated episodes of acute encephalopathy. The progressive deterioration of the intellect (bradypsychia, lack of interest) is accompanied by an attack on the neurological system (ataxis, choreo-athetosic movements, trembling, dysarthria), this may progress to the stage of profound dementia.

Anatomically, the lesions resemble those produced by Wilson's disease: astrocytaric hyperplasia and degenerative neural lesions in the cerebral and cerebellar cortex, the basic nuclei and the serrated nuclei.

**The terminal hepatic coma**
This indicates complete hepatic degeneration, which follows terminal cytolysis of the rare hepatic cellular pockets which until then were still active.

The clinical table is that of a serious icterus, with digestive or cutaneomucus hemorrhaging, fetid breath (fœtor hepaticus) and significant biological disruptions (hypoglycem ia, hypocholesterolemia, hyperammoniemia, hypovolemia). The subject falls into a progressive coma, fatal in despite of all efforts one may make.

**Polyneuritis of the lower limbs**
From a physiopathological point of view, a nutritional difficulty intervenes with vitamin deficiency, and a unbalanced diet in favor of carbohydrates to the detriment of proteins.

Metabolical difficulties brings about a primitive axonal neuropathy, which produces a retrograde distal degeneration (7).

**The retro-bulbaire optic nevrite**
This is normally the consequence of the mixed intoxication of alcohol and tobacco. It is initially characterized by a progressive bilateral decline of visual acuteness, then by a genuine central scotoma.
Later, when an examination of the rear of the eye is conducted, a discoloration of the papilla in its temporal sector is remarked. Optic atrophy then begins, and risks to end in definite blindness if abstinence from alcohol or vitamin therapy is not undertaken.

**Alcohol neuro-acropathy**
Called the Bureau and Barrière syndrome (2), it concerns tropic symptoms in the extremities, both on the cutaneous level (tégumentary hypertrophy) and the osteo-articular level; these lesion have a noted radiological tendency (metatarsal osteolysis), for it is often accompanied by analgesia (16). Men are the most often affected.

**Attacks on the medulla**
Although rare, several cases of combined subacute degenerence of the medulla have been published, and have been attributed to a folic acid deficiency (8).

**Effects on the autonomous nervous system**
In chronic alcoholism, disruptions of sudation and arterial tension (hypertension, or orthostatic arterial hypotension) can be remarked (9). Vagal effects may even be the cause of gastro-intestinal difficulties, and a lower attack may result in impotence.

**Chronic deliriums**
The chronic deliriums of alcoholics are manifested in two different ways: chronic hallucinatory psychosis and interpretive passionate delirium or jealous delirium (12):
- **The chronic hallucinatory psychosis of alcoholics**:
The beginnings may be brutal, appearing after a confused dream-like state, even immediate symptoms of a mental automatism, or even more insidious, with behavior and attitudinal difficulties. It is a auditory and visual hallucinatory mania, with symptoms of persecution and jealousy, badly systematized, where the subject is unanxious and exhibits a certain indifference to hallucinatory remarks.
- **The delirium of jealousy**:
This delirium occurs in a subject predisposed to paranoiacal tendencies or following a change in attitude and personality induced by chronic alcoholism. An extensive systematized interpretative mechanism mania is present, with jealous tendencies; the clinician must
constantly take into consideration the dangerous character of the subject (aggressive and violent reactions towards the spouse or imaginary rival).

CONCLUSION

Several tendencies can be remarked in alcoholic behaviour: one may be unthinkingly brought to drink when placed in a social situation; one may also feel the need to compensate for a weakness, a suffering which disturbs social relationships and which finds a transient relief by resorting to a tranquilizing pastime, most often alcohol.

In each of these cases, the same goal is expected: to meet others and to socialize in situations where alcohol is present (1).

Alcohol may also be abused for its intrinsic hedonistic effects, in this case it becomes an object of social transgression, of egoistic pleasure where socializing with others does not occur.

For whatever reasons one begins abusing alcohol, the clinician must constantly remember that alcohol may be a source of its own neuropsychiatric pathologies, and that an attentive surveillance of the patient, the first stage in prevention, may at times permit the avoidance of the most serious after-effects.

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