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Review Article

SECONDARY METABOLIC COMA; SYMPTOMATOLOGY AND PROGNOSIS

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Large number of metabolic abnormalities are responsible for the development of brain disease. Stupor and coma do arises from intrinsic diseases of the neurons, neuroglial cells or from diseases extrinsic to neurons and glia(1). Intrinsic disorders that results in primary metabolic brain disease encompasses the degenerative cerebral diseases, which usually develop insidiously and are mostly irreversible. A typical example is ALZHEIMER's disease.

Secondary metabolic coma results when extracerebral diseases interfere with brain metabolism, either by causing deficienies of nutrition or by producing electrolyte imbalances of intoxication. These metabolic comas are caused by endogenous or exogenous disturbances. Examples of endogenous disturbances are ischemia, anoxia, hypoglycemia, uremia and hepatic coma, postictal coma, coma due to meningitis or subarachnoid hemorrhage. A typical example for an Eaogenous disturbance is poisoning with soporific drugs(2).

The clinical picture of secondary metabolic coma, the depth of coma and the complications during the course of treatment depends on the causative illness.

Despite of individuailities, specific illnesses often produce characteristic clinical symptoms. On the other hand the clinical signs depend on the severity of the onset, the duration of the metabolic disorder and the secondary brain dysfunction.

According to the clinical picture and the course of the metabolic coma the following stage are to be distinguished :-

1- The acute course of metabolic coma (table 1).

1.1.A rapid decrease of consciousness caused by an acute breakdown of cortical and brainstem functions. This state is characterized by the symptomatology of the different phases of acute midbrain syndrome(3). It is impossible to distinguish exactly between the phases before patients are in the full stage of midbrain syndrome. An acute exogenous reaction type(4) or an organic peychosyndrome are rarely observed in these cases, because of the short lived initial mental changes. Although, almost any eye position or random movement can be seen transiently when brainstem function is changing rapidly. A maintained conjugate lateral deviation or dysconjugate positioning of the eyes at rest suggests structural rather than metabolic disease(1).

The phases of Medullary syndrome with the breakdown of all cerebral functions finally appear. Typical examples are acute hypoxia due to different disorders and acute intoxications with soporific drugs, alcohole or cyanides.

1.2. The appearence of brain edema is the cause of a less dramatic development of the phases of acute midbrain syndrome. Typical examples of metabolic disorder, inducing secondary brain edema are acute hepatic or uremic coma, electrolyte imbalances and intoxications with mushrooms or soporific drugs(5).

2. The subacute course of metabolic coma (table 2).

2.1. The "turbulent" course is one out of two possibilities of mental changes in the initial stages of subacute metabolic coma. This course shows signs of the neurasthenic or emotional-hyperesthetic syndrome(4) with irritatability, restlessness, fearfull depressive mood, forgetfulness, disturbance of concentration and lack of motivation are remarkable. In the later course, an acute exogenous psychosis with optic and acoustic hallucinations, the so called Amential phase may be observed.

2.2. The "silent" course of subacute metablic coma is characterized by the development of an organic psychosyndrome accompanied with disturbance of attentiveness, narrowing of thoughts, perseveration, disturbance of recent memory, emotional dullness and increasing primitive motor patterns. This clinical picture corresponds to the amnestic or organic psychosyndrome(6).

2.3. Symptoms of WERNICKE-KORSAKOFF syndrome occur in the majorty of patients in the later course of the metabolic disease.

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illness patients may show the clinical
f delirium. Recent memory is impair-
e severely than other mental functions.
nemory loss produces the KORSA-
syndrome.
metabolic disease continues a KLU-
3UCY syndrome may develop. The
f consciousness is slightly reduced.
imitive motor pattern changes to com-
ittern, like grasping objects and brin-
iem to mouth. Hypersexuality, init-
haraterized by verbalisation later on
s of masturbation.
futher stages of disintegration, cons-
ss declines to somnolence and final-
a. The complex motor patterns in-
and are replaced by tossing and turn-
ovements. Optical fixation but follow-
ovements are maintained. Occasion-
articulate utterances are given. There
ncrease in muscle tone and pyramidal
ppear. This stage is usually describ-
"precoma".
on equent stage is the full stage of me-
coma. Patients are unconscious.
logical examination reveals divergence
bulbs, decorticate position of the ex-
es, maximal nicrease of muscle tone,
son symptoms detailed primitive
pattern and diffuse abnormal motor
incluling tremor myoclonus and es-
y asterixis.
might pass into chronicity at this stage
l picture signifies the metabolic apallic
me according (7).
ical examples of the subacute course is
te hepatic or uremic coma chronic me-
oning, chronic intoxications with tran-
r bromides, deficiency of vitamines and
iseases.

lescribed stages are steps of disintegra-
re also in the reverse, steps of reintegra-
stepwise progressive disintegration of
um occurs with uniform central remis-
n in the levels of the cortex, limbic
idbrain, and lower brainstem. Certain-
ervation of all stages is not obligatory
e during the course of metabolic coma.
hortlived stages are withdrawn from
ion of the physician. Reintegration
r at any stage of the metabolic coma,
isly or induced by effective therapy. In
cases reintegration may be delayed.
which are not observed in the course
ration may occur in the later course of
on. These symptoms especially belong
to paranoidhallucinatory states. On the other hand phases of acute midbrain and medullary syndrome with patient's death in the later course may develop out of any stage of subacute metabolic coma. The fluctuation from a subacute course to an acute one and vice versa may recur during the whole development of metabolic coma.

There are two views about the mechanism of mental changes in the initial phase of metabocoma. The first, or quantitative view of brain function was supported by the study of Chapman and Wolf(8), who concluded that behavorial impairment was directly related to the total mass of inadequately functioning neurons. The other or focal, view of brain function was supported by correlations of specific defects in memory and orientation with specific anatomically varieified brain lesions(9,10). According to the suggestion of PLUM and POSNER(1) a combination of both pathological processes is probably the basis of clinical picture of most metabolic brain diseases.

A severe and repid onset of a secondary metabolic brain dysfunction causes an acute general loss of the highest integrative functions down to the midbrain level or lower brainstem. In these cases, stages of an acute midbrain or medullary syndrome appear in rapid succession. In other cases a slower development of acute midbrain or medullary syndrome is attributed to a secondary brain edema with impaction.

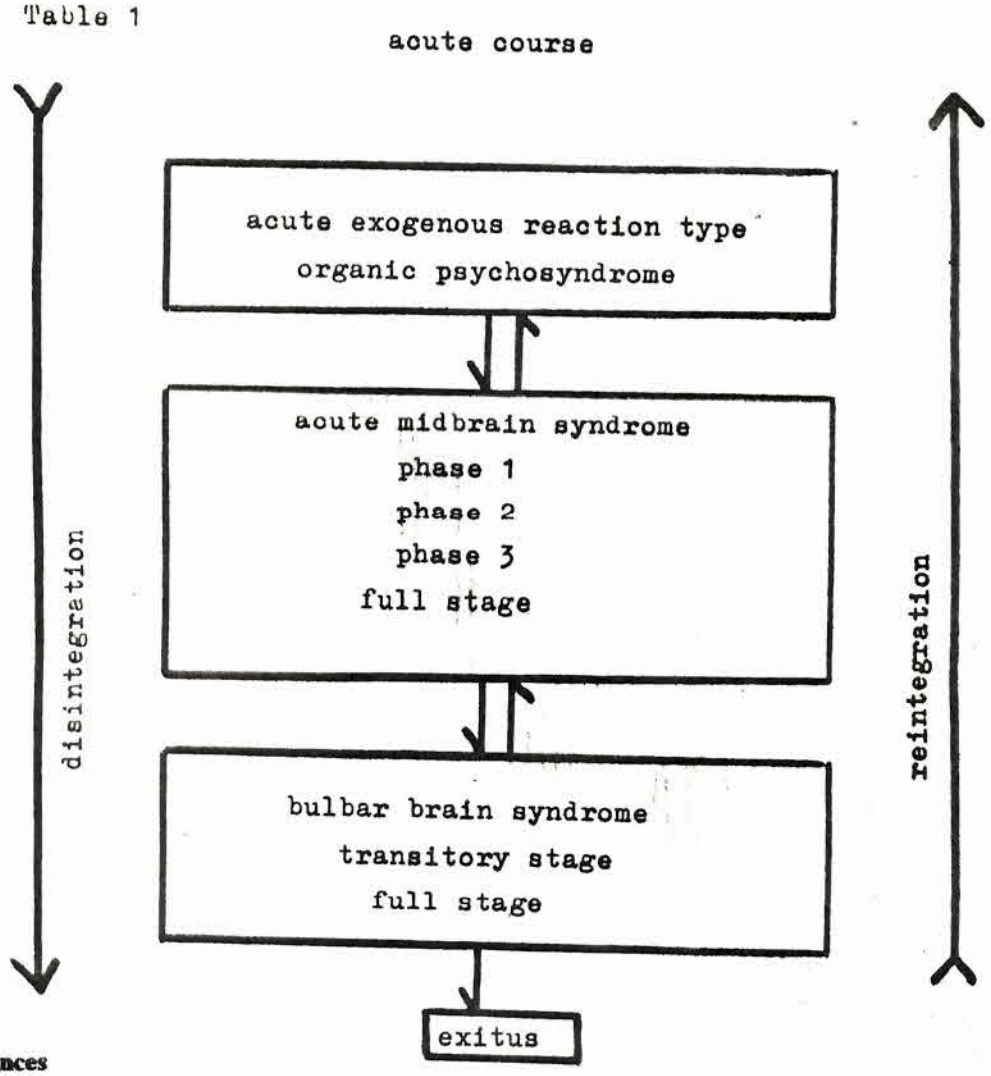
In subacute courses of secondary metabolic brain diseases causing coma, the development of the different stages is less rapid and dramatic depending on an interaction between the patient's premorbid personality and the gross amount of impaired cerebral tissue.

The neurological and psychiatric abnormalities are usually reversible if the metabolic disease is mild, brief or if therapy is sucessful. The clinical course of dis-and reintegration follows the described stages independent of the specific causative mechanism. Although some metabolic comas have a recognizable clinical stamp, it's generaly difficult to indentify the primary cause of the metabolic coma by clinical examinatio.n(1) Especially in final stages where different metabolic comas show rather identical clinical signs(12).

Therefore the value of an exact neurological and psychiatric examination is not a diagnostic but a prognostic one. Under normal circumstances the appearence of signs of reintergration give good prognosis to the outcome of a patient. On the other side a deterioration of the clinical picture indicates bad prognosis and may induce considerations to the therapeutic management.

METABOLIC COMA

Table 1



References

(1) Plum F., Posner J.B.: Diagnosis of Stupor and Coma. 2nd edition F. A. Davis Company, Philadelphia 142-216 (1972).

(2) Kubicki St., Rieger H., Busse G.: EEG in Fatal and Near-Fatal Poisoning with Soporific Drugs I. Typical EEG Patterns. Clinical Electroencephalography 1, 5-13 (1970).

(3) Gerstenbrand F., Lucking C.H.: Die akuten traumatischen Hirnstammschaden Arch. Psychiat. Nerven Kr. 213, 264-281 (1970).

(4) Bonhoeffer, K. (1917) Die Exegenen Reaktions Typen. Arch. Psychiat-Nervenkr. 58, 58-70.

(5) Hackl J.M., Kornberger E., Rumpl E., Geier W.: Intoxikationen und forcierte Diurese. Infusionstherapie 4, 280-284 (1977).

(6) Bleuler, M (1941) Organischen Psychosyndrom. Lehrbuch der psychiatrie. Leipzig.

(7) Gerstenbrand F., Avenarius H.J., Preissler H.P.: The Apallic Syndrome in Metabolic Disorders of Brain. In: The Apalsic Syndrome, Springer Verlag Berlin, Heidelberg, New York 29-36 (1977).

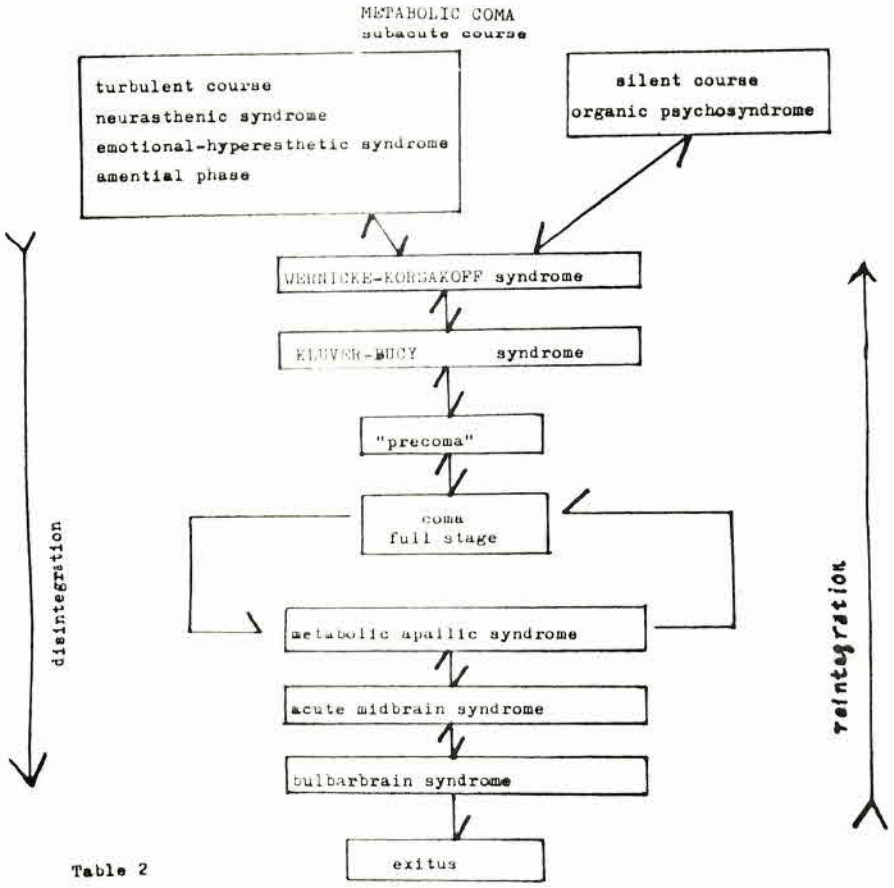


Table 2

(8) Chapman L.F. Wolff H.G.: The cerebral hemispheres and the highest integrative functions of man.
Arch. Neurol, 1, 357-424 (1959)

(9) Scoville W.B., Milner B.: Loss of recent memory after bilateral hippocampal lesions.
J. Neurol. Neurosurg. Psychiat. 20, 11-21 (1957).

(10) Penfield W., Milner B.: Memory deficit produced by bilateral lesions in the hippocampal zone.
Arch. Neurol. Psychiat. 79, 475-497 1958).

(11) Plum F.: Metabolic Encephalopathy. In the Nervous System.
Vol. 2: The Clinical Neurosciences, Raven Press, New York 193-202 (1975).

(12) Binder H., Gerstenbrand F.: Das metabolische Coma Symptomatologie und Verlauf.
Kop.klinik 1, 186-192 (1976).

الخلاصة

هنالك امراض أيضية تعكس على عمل الدماغ فتصيبه عن العمل مسببة اختلالا في الوعي ، منها من ينبع من الحجيرات الدماغية كمرض الزهايمر ومنها امراض خارج الدماغ تنعكس عليه ثانويا مسببة اضطرابات واختلال في العمل الدماغي كما يحدث في نقص السكر في الدم او عند خذلان الكبد .

ان هذه الحالات قد تظهر باشكال سريرية مختلفة منها الحاد ومنها المتدرج ولكنها جميعا تتبع نمطا خاصا سواء في التدهور او في التحسن كما مبين في المخططين المرفقين .

ان معرفة الاعراض العقلية والعصبية لا يعتبران مهمين فقط من حيث التشخيص بل ان لها الاهمية الخاصة من حيث انذار المرض واسلوب معالجته .