SEPSIS-ASSOCIATED ENCEPHALOPATHY

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In January, 2012, a 53-year-old woman with a history of progressive polyposis coli and hypertension was admitted to the intensive care unit (ICU) of our hospital because of septic shock 4 days after laparoscopic subtotal colectomy.

She was intubated and mechanically ventilated, underwent emergent re-laparotomy for anastomotic leakage, and was treated with antibiotics. Blood cultures were positive for multiresistant *Enterococcus faecium*. After re-laparotomy the patient was sedated with midazolam for 2 days.

The neurologist was consulted on the fourth postoperative day because of the patient’s persistent low level of consciousness.

On neurological examination, we found no eye opening or motor response to a painful stimulus, intact brainstem reflexes, and generalised areflexia of arms and legs with indifferent plantar reflexes. We observed no signs indicative of minimally convulsive status epilepticus.
DEEP COMA AND TETRAPLEGIC?
During the period of septic shock and surgery, blood pressure had not been lower than 100/50 mm Hg and laboratory tests did not show abnormalities that could explain the patient’s coma.

Non-contrast brain CT showed abnormal hypodensity of the complete white matter with swelling of the entire brain. CT angiography was normal.
WHY COMA?
WHY TETRAPLEGIA?
BRAIN DAMAGE
SYSTEMIC HYPOTENSION
Infarto nelle zone di confine: infarto emorragico asimmetrico nella zona di confine tra a. cerebrale anteriore e media
BRAIN DAMAGE
HYPOXIA-ISCHEMIA
Hypoxia, hyperoxia, ischemia, and brain necrosis
Miyamoto O, Auer RN.

Nucleo caudato

Ippocampo
Hypoxia, hyperoxia, ischemia, and brain necrosis
Miyamoto O, Auer RN.
Electromyography showed absence of motor unit potentials in arm and leg muscles without evidence for accompanying neuropathy.

We made a diagnosis of ......

................................. critical illness myopathy.

Because of sepsis and the absence of severe structural brain alterations at brain CT we made a diagnosis of ..... 

................................. sepsis-associated encephalopathy.
1. BRAIN
2. SPINAL CORD
3. PNS
2. ISCHEMIC
3. HYPOXIC
4. HYPOXIC-ISCHEMIC
5. OTHER MECHANISMS
6. PROGNOSIS
Because of the deep coma and the diffuse and severely abnormal aspect of the white matter—both associated with poor outcome—discontinuation of treatment was discussed ......

......... but we recommended to continue treatment.
REASONS WHY

More time is needed
Condition is poorly known
Condition is reversible
Condition is very rare
Is the outcome of critically ill patients inherently unpredictable?

Yes, if early prediction of individual outcome is at stake.
On the 9th postoperative day the patient opened her eyes in response to painful stimuli, but there was still no motor response. Follow-up brain CT showed no improvement of the white matter abnormalities.

COMA?
A videotape showing that Ms. Schiavo was able to open and move her eyes ignited the public skepticism over her diagnosis of VS

James L. Bernat, Neurology 2008;71:964–965
In the following weeks the condition of the patient gradually improved and 4 weeks after the re-laparotomy she had regained full consciousness.

When the patient was discharged from the ICU her muscle strength had gradually improved with movements against gravity now possible.

At that time brain MRI was normal except for a small number of white matter lesions.
A disorder or disease of the brain.

_In modern usage_, encephalopathy does not refer to a single disease, but rather to a syndrome of global brain _dysfunction_; this syndrome can have many different organic and inorganic causes.
A term often used by electroencephalographers, referred to as delirium in psychiatry and as altered mental status or acute confusional states in neurology.
SEPSIS-ASSOCIATED ENCEPHALOPATHY (SAE)

SAE is commonly seen in systemically ill patients. The syndrome is defined by diffuse cerebral dysfunction that accompanies sepsis in the absence of direct CNS infection, structural abnormality or other types of encephalopathy (for example, hepatic or renal encephalopathy), as detected by clinical or standard laboratory tests.

Gofton TE, Young BG. Sepsis-associated encephalopathy. Nat Rev Neurol 2012; 8: 557-566
COMMENTS

Severe encephalopathy with extensive *white matter lesions* can be a reversible condition.

Clinicians should realise that the first *clinical* signs of improvement of the clinical condition could still be observed beyond 7 days after onset.

*Central and peripheral* nervous system and muscle complications often coexist in the septic patients.