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Metabolic Encephalopathy after Pancreaticoduodenectomy and Left Hemihepatectomy: A Case Analysis and Literature Review

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ABSTRACT

Metabolic Encephalopathy mainly refers to a group of diseases in which biochemical metabolic changes in the body cause changes in the environment of brain tissue, resulting in brain dysfunction. For the post-operative patients who underwent radical pancreaticod- uodenectomy combined with left hemihepatectomy in Hepatobiliary Pancreatic Surgery, such as severe trauma, many postoperative complications and long fasting time, the probability of metabolic encephalopathy is higher. A case of metabolic encephalopathy after radical pancreaticoduodenectomy combined with left hemihepatectomy in our hospital is analyzed as follows.

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Case Data

The patient, a 57-year-old female, went to our hospital in May 2020 because of "liver space occupying lesions found in examination". And after perfecting relevant examinations, she was diagnosed as "1. Malignant tumor of pancreatic head; 2.Duodenal malignancy; 3.Secondary malignant tumor of liver and intrahepatic bile duct". So, she underwent "1. Radical pancreatoduodenectomy; 2.Left hemihepatectomy; 3.Microwave ablation of liver lesions under the guidance of ultrasound" in June 2020. Postoperative pathology suggests: pancreatic endocrine tumor. In December, 2020, the patient returned to the hospital for reexamination, and CT examination showed: gall bladder-Intestinal anastomotic stenosis and multiple metastatic tumors in the liver were bigger and more than before (May, 2020). So, in December, 2020, "1. Partial hepatectomy; 2. Ultrasound-guided microwave ablation of liver lesions; 3. Bile duct plasty; 4. Bile duct stent implantation" was operated on her. After the operation, the patient recovered partially, and she was transferred to our hospital branch (New Kunhua Hospital) to continue symptomatic support treatment. In January, 2021, the patient developed physical activity disorder, slurred speech, unconsciousness and lethargy, and she was transferred to our hospital for further treatment. So, she was admitted to our hospital for "unconsciousness and lethargy for 5 days". Physical examination: T 36.5°C, P 104 times/min, R 20 times/min, BP 105/60mmHg, SO_2 99%. The general condition is poor, indifferent and frequently asked but unanswered. Physical examination is inadequate, pharyngeal reflex exists and bilateral frontal lines are symmetrical. Bilateral eyeballs move freely, bilateral pupils are round, and sensitive to light reflection, d:3.0mm, sclera without yellow dye. Bilateral nasolabial sulcus is symmetrical, limb pain stimulation can be seen to retract spontaneously limb muscle tension is normal and limb tendon reflex is symmetrical. Pathological sign is negative. Neck is soft, and Kerning sign is negative. Feeling, does not cooperate with mutual aid. Heart and lung are normal. The whole abdomen is flat and an old surgical scar of 18cm is seen in the upper abdomen, and the whole abdominal muscles are soft, without tenderness and rebound pain. Laboratory examination: leukocyte 12.9x10^9/L, neutrophil 80.4%, lymphocyte 13.6%, erythrocyte 2.92x10^12/L, hemoglobin 82g/L, hematocrit 0.234L/L, platelet 235x10^9/L; AST 16U/L, ALT 7U/L, direct bilirubin 7.6umol/L, indirect bilirubin 8.6umol/L, Albumin 33.2g/L, prealbumin 106mg/L, glucose 6.9mmol/L, creatinine 30umol/L, urea nitrogen 2.7mmol/L, sodium 129mmol/L, chlorine 98mmol/L, potassium 4.0mmol/L, lipase 111U/L and amylase 78U/L. CK-MB 0.3ng/ml, aTnI 0.0 ng/ml, MYO 33.0 ng/ml. Arterial blood gas analysis: pH 7.49, oxygen partial pressure 111.6mmHg, nitrogen dioxide partial pressure 28.0mmHg, actual residual alkali -0.3mmol/L, whole blood residual alkali -1.0mmol/L, standard bicarbonate 23.5mmol/L, bicarbonate concentration 20.9mmol/L, potassium ion concentration 3.75nmol/L, the sodium ion concentration is 131.2mmol/L, the calcium ion concentration is 1.123mmol/L, the chloride ion concentration is 96.7mmol/L and the anion gap is 17.3mmol/L. Blood ammonia 25umol/l, lactic acid 2.78mmol/L. APTT 39.5s, PT 14.2s, TT16.2s, FIB 3.73g/L, FDP 5.2ug/ml, DD2 3.00ug/ml, APIII 73.2%. Examination of cerebrospinal fluid: the total number of nucleated cells 4x10^6/L, neutrophils 69%, lymphocytes 19%, monocytes 9%, glucose 3.7mmol/L, chlorine 116mmol/L, TB DNA (-), Pro-CSF 324mg/L, IgG 37.10mg/L, IgA 5.80mg/L. Pandy test (-). blood culture: facultative anaerobic Escherichia coli was detected. Culture of biliary pus: Enterococcus faecalis and Candida albicans were detected. Paraneoplastic antibody (-). Anti-TORCH antibody tests

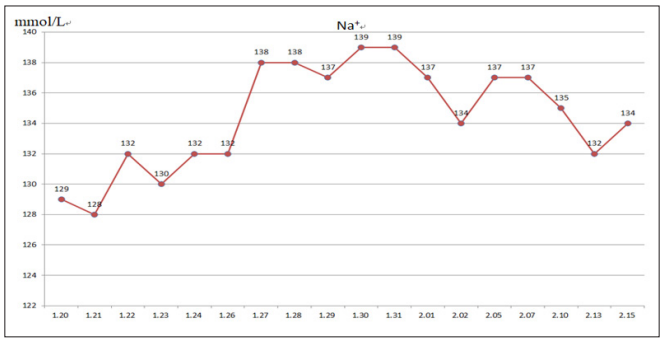
were all negative, interferon-gamma test (-) and fungus test (-) of T cells infected with tuberculosis. Imaging examination: CT examination showed: 1. Patch exudation, consolidation shadow and streak shadow were seen in the lower lobe of both lungs and the lateral segment of the right middle lobe, and inflammatory lesions were considered. 2. A small amount of effusion in the right thoracic cavity. 3. The heart shadow is not big; See the superior vena cava catheterization; Gastric tube indwelling. 4. The liver is scattered in flaky slightly low-density shadow, and the metastatic focus needs to be discharged. Stent shadow can be seen in portal area; a small amount of fluid in the liver area. EEG: Diffuse slow wave, moderately abnormal EEG. MRI examination of the brain shows that bilateral frontal cortex, bilateral hippocampus, bilateral thalamus, posterior pons and bilateral cerebellar hemispheres are symmetrical abnormal signal foci. Considering the metabolic possibility, please combine with clinical practice.

Discussion

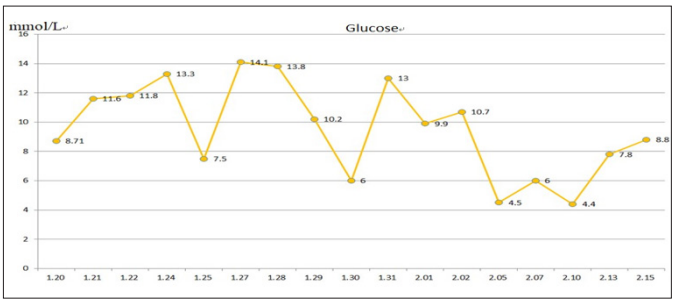
Metabolic Encephalopathy mainly refers to a group of diseases in which biochemical metabolic changes in the body cause changes in the environment of brain tissue, resulting in brain dysfunction [1, 2]. Its clinical manifestations are diverse, which can be divided into focal signs and comprehensive signs. The focal symptoms are mainly localization signs of cerebral hemisphere and brain stem, and the comprehensive signs are mainly diffuse brain dysfunction [3]. Mild cases have behavior disorder, insanity or local brain damage with epilepsy and hemiplegia, and severe cases have coma, brain ankylosis or corticated ankylosis [4, 5]. There are many causes of metabolic encephalopathy. In hepatobiliary and pancreatic surgical diseases, the main causes of metabolic encephalopathy are hepatic encephalopathy caused by cirrhosis, hyperammonemia encephalopathy, blood glucose disorder encephalopathy and electrolyte disorder encephalopathy, among which hepatic encephalopathy has the highest incidence.

The pathogenesis of metabolic encephalopathy is complex, and there are many systemic diseases that produce metabolic encephalopathy. Different diseases have different pathogenesis [5]. The possible mechanisms are summarized as follows: changes of cerebral blood flow, changes of fluid, changes of interfering neurotransmitters, free radical damage and apoptosis [1, 2].

The patient's initial symptoms were limb dyskinesia and slurred speech. At the beginning of the symptoms, the blood Na+ was 131.9mmol/L. After sodium supplementation (10ml:90mg NaCl with 10% 1g:10ml NaCl*3)*3 groups, the patient became unconscious and sleepy. Changes in internal environment can affect the metabolic changes of water and electrolytes in brain tissue, leading to metabolic disorder in intracranial brain tissue. Hyponatremia is the most common type of water and electrolyte disorder in the central nervous system, but excessive and rapid correction of hyponatremia can cause severe demyelinating lesions of permeable brain tissue [6]. In the sequential therapy, the patient's blood Na+ was as low as 128.0mmol/L. After the standard sodium supplement therapy, the patient's blood Na+ gradually returned to the normal value (135-155mmol/L), and the patient's consciousness gradually recovered. Therefore, hyponatremia and excessive and rapid sodium supplementation may be one of the causes of metabolic encephalopathy.



During the treatment of metabolic encephalopathy, parenteral nutrition combined with enteral nutrition was used for nutritional support, and the blood sugar fluctuated between 4.4mmol/L and 14.1mmol/L. After radical pancreaticoduodenectomy combined with left hemihepatectomy, the function of regulating blood sugar was inadequate. Moreover, when enteral nutrition is combined with parenteral nutrition for nutritional support, the blood sugar itself fluctuates greatly, which easily leads to unstable blood sugar of patients. When blood sugar is low, it is manifested as paralysis of peripheral nerve or cranial nerve, convulsion, coma, wet and cold skin, etc.; when blood sugar is high, it is manifested as insanity, unconsciousness, coma, hemichorea and throwing action, etc. [7, 8]. In hepatobiliary and pancreatic surgery, radical pancreatoduodenectomy is one of the operations with the highest reexamination degree. Pancreas resection can cause blood sugar fluctuation and even blood sugar disorder. Require a certain fasting time after operation. In order to ensure the nutritional needs of patients, parenteral nutrition support can only be used to maintain the vital needs of patients. In this process, in the process of giving parenteral nutrition, patients will jointly give too much or too little exogenous insulin, jointly apply enteral nutrition with high sugar content or infuse high-concentration glucose to promote the body to release insulin. These will lead to high or low blood sugar [9-11]. During the follow-up treatment, after the patients gradually reduced enteral nutrition, their blood sugar tended to be normal and their consciousness recovered to some extent. Therefore, the result-oriented results showed that excessive fluctuation of blood sugar might also be one of the original causes of metabolic encephalopathy.



During the treatment of metabolic encephalopathy, the patient showed severe systemic inflammatory reaction, so lumbar puncture was performed for cerebrospinal fluid examination, and the central nervous system infection was excluded. Facultative anaerobic Escherichia coli was detected in blood culture. Enterococcus faecalis and Candida albicans were detected in the pus culture of biliary anastomoses. The change of the patient's consciousness may be related to systemic inflammatory reaction, that is, sepsis encephalopathy. Sepsis encephalopathy refers to a disease state in which patients lack clinical or laboratory evidence of central nervous system infection, exclude organic diseases, diffuse brain

dysfunction caused by systemic inflammatory reaction caused by sepsis, and seriously affect nervous system function [12, 13]. The clinical manifestations of the patients were mental consciousness and movement changes, even coma [14].

In the course of treatment, cefoperazone sodium sulbactam sodium and imipenem were used for anti-infection treatment, while the patients' systemic inflammatory reaction was controlled and their consciousness was recovered. Therefore, sepsis encephalopathy cannot be completely ruled out as the cause of consciousness change in patients [15].

The etiology of metabolic encephalopathy is complex and diverse, which can involve various diseases of many systems. The diagnostic criteria and pathogenesis are not clear, and its treatment lacks specificity. Most of them are to treat primary diseases while supporting symptomatic treatment. Early diagnosis and early treatment have a good prognosis and are the key steps to reduce the mortality of metabolic encephalopathy.

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