TROUSSEAU SYNDROME AS AN INITIAL MANIFESTATION OF PANCREATIC ADENOCARCINOMA: CASE REPORT

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Abstract—Multiple ischemic cerebral strokes as the first manifestation of pancreatic carcinoma are infrequent. In recent times the term Trousseau’s syndrome must be reserved for unexplained thrombotic problems that precede or appear concomitantly with visceral malignancy. We report a case of 55-year-old female with malignancy–related thromboembolism, presented with multiple and recurrent ischemic strokes, from a recently diagnosed pancreatic adenocarcinoma in the tail.

We conclude that patients with multiple, repeated cerebral infarction, with minimal risk factors for stroke, must be further investigate for an underlying malignancy, possibly of pancreatic origin.

Keywords—Trousseau syndrome, stroke, malignancy, pancreas

I. Introduction

Multiple ischemic cerebral strokes as the first manifestation of pancreatic carcinoma are infrequent [1,7,9,10]. In recent times the term Trousseau’s syndrome must be reserved for unexplained thrombotic problems that precede or appear concomitantly with visceral malignancy [2,6,8,12]. Migratory thromboses, which occurred in about 10% of patients with pancreatic adenocarcinomas, may be associated with chronic disseminated intravascular coagulopathy, cancer–related hypercoagulability syndrome, or tumor embolism [5,13,15,16]. Pancreatic tumor of the body and the tail tended to grow relatively silently and metastasized before diagnosis [11].

We reported a case of 55-year-old female with malignancy–related thromboembolism with multiple and recurrent ischemic strokes, from a recently diagnosed pancreatic adenocarcinoma in the tail.

II. Case report

A 55-year-old female, previously healthy, presented with sudden-onset speech difficulties, dizziness, and right-sided leg weakness resulting in unsteady gait. Prior to admission to neurology clinic, a medical consult with gastroenterologist was done due to intermittent epigastric pain, noted after meal. She had normal vital signs and was fully awake, conversant with Glasgow Coma Scale (GCS) at 20. Neurological examination revealed partial sensory and motor aphasia, right-sided latent lower monoparesis, and hemi-hyperreflexia. The patient did not have any cranial nerves dysfunction, sensory deficit or coordination disorder. Laboratory data demonstrated anemia (Hb 116 g/l) and elevation of erythrocyte sedimentation rate 40 mm, Leucocytes 16.2 g/l, blood glucose 7.1 mmol/l, ASAT 49 U/l, ALAT 40 U/l, CRP 176 nmol/l, tumor markers: CEA - 328 ng/ml, CA 15-3 - 91.3 U/ml, CA 19-9 >1000 U/ml, CA 125(OV) >500 U/ml, proteinuria and hematouria. Hypercoagulability studies were negative, coagulation factors, and D – dimers were within normal.

Fig.1 Non-enhancing hypodense area (12.5x11 mm, 25 HU) in the left parietal and occipital lobes, and second one in the left parietal lobe.

Later on brain CT (25.09.2012) revealed one hypodense area (44x22 mm, 29 HU) in the left parietal lobe.
and second one located near the posterior corn of the left lateral ventricle (fig.2).

Fig.2. a hypodense zone left parietal 44/42mm, with mean density 29 HU, acute ischaemic insult; b small hypodense zone 9mm left parietal above the posterior horn of the left ventricle-subacute ischemic insult.

One week later the patient demonstrated progression of her neurologic deficit. Brain magnetic resonance imaging (MRI) (28.09.2012) revealed large infarction localized in the left temporal and parietal lobes, multiple punctiform areas in the right parietal lobe, both cerebellar hemispheres, and corpus callosum, as well as two encephalomalatic areas with perifocal gliosis in the right frontal and temporal lobes. MRI data confirmed multiple acute embolic ischemic strokes and two old ischemic cerebrovascular accidents (fig.3).

Fig.3. a AX T2 FLAIR- multiple hyperintense zones right parietal and left parieto-temporal, which present multiple acute ischemic insults; b DWI- multiple hyperintense zones right parietal, left parieto-temporal, with diffusion restriction, due to multiple ischemic insults; c ADC map, these lesions are correspondingly hypointense on ADC map - acute ischemic insults.

Fundoscopic examination discovered findings of hypertonic retinal angiopathy. Transcranial Doppler ultrasonography was normal. Trans-thoracic echocardiography was normal. Chest findings were normal. Enhanced abdominal CT (26.09.2012) demonstrated multiple, different sized oval-shaped metastatic lesions in the both liver lobes, the biggest one 60x50 mm, one hypodense area in the upper pole of the spleen, and necrotic tumor mass (50x39x25) located in the pancreatic tail (fig.4).

Fig. 4 Contrast-enhanced CT of abdomen shows hypodense zone in the pancreas tail – Tu – carcinoma; multiple hypodense zones in the liver, and one hypodense area in the spleen upper pole – meta.

Fine-needle aspiration liver biopsy (27.09.2012) detected cytological findings of metastases from low-differentiated adenocarcinoma. The consult with oncologist and abdominal surgeon confirmed data relevant to advanced stage IV adenocarcinoma of pancreatic tail with liver and spleen metastases.

We diagnosed Trousseau’s syndrome accompanying pancreatic cancer and performed anticoagulation (Heparin) and antiplatelet (Clopidogrel and Aspirin) treatment. Brain CT monitoring (18.10.2012) revealed hemorrhagic transformation in the previous ischemic areas (fig.5).

Fig.5. a hemorrhagic transformation, hyperdense linear foci, in the zones of the known insults; b hemorrhagic transformation, hyperdense linear foci right parietal in the new discovered insult.

The patient died of cerebral edema 1 month later.

III. Discussion

In 1865, Armand Trousseau (1801 – 1865) described that some patients presented with unexpected, unusual, or migratory thrombosis, later or concomitantly manifested a visceral malignancy [2,3,8,12,13,14]. In review of the findings – our patient suffered from multiple bihemispheric progressive ischemic stroke. Atherosclerosis, hypertension, diabetes mellitus, or nonneoplastic hypercoagulable states appear to be conventional risk factor for cerebrovascular disease, but she was not known hypertensive and/or diabetic. Ischemic stroke...
with no overt vascular risk factors should be considered for cancer screening [1,4,5,6]. For this patient CA19-9 was extremely high and a large silent necrotic mass in pancreatic tail were seen on abdominal CT. The combination of radiology with a best serum tumor marker for pancreaticobiliary cancer CA19-9, significantly increases the diagnosis sensitivity to 97.2% and the specificity to 88.7% [7]. The presented patient has necrotic stage IV adenocarcinoma of pancreatic tail with hepatic and splenic metastases and multiple acute cerebral ischemic stroke as the first symptom, which is very rare. Adenocarcinoma of pancreatic tail often grows silently and the most common clinical manifestation is glucose tolerance disorder [11]. The underlying pathophysiological mechanisms for the presence of the cerebrovascular diseases, due to the pancreatic cancer itself, are unclear [9,10,15,16]. For this patient, except the elevated serum pancreatic carcinoma antigen and mild hepatic pathology, the coagulation factors and all of the rest blood tests were within normal limits. Ultrasonographic duplex study, trans-thoracic echocardiography and chest findings were normal. We speculate that in our case the cause of the multiple cerebral infarctions may have been due to migratory arterial tumor emboli from necrotic pancreatic cancer.

IV. Conclusion

We conclude that patients with multiple, repeated cerebral infarction, with minimal risk factors for stroke, must be further investigate for an underlying malignancy, possibly of pancreatic origin.

REFERENCES