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Fallopian tube serous adenocarcinoma with dizziness as the initial symptom: a case report

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Abstract

Paroxysmal vertigo as the presenting symptom of a fallopian tube tumor is rare among patients. We present a patient who was finally diagnosed with fallopian tube serous adenocarcinoma with subacute cerebellar degeneration. We analyzed the patients' clinical, pathological, and imaging data. We conclude that the possibility of paraneoplastic neurological syndrome should be considered when conventional treatment is ineffective for a fallopian tube tumor and other neurological diseases are excluded.

Keywords

Paraneoplastic neurological syndrome, subacute cerebellar degeneration, fallopian tube serous adenocarcinoma, cerebrospinal fluid, glucose metabolism, dizziness, laparoscopic resection

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Introduction

Gynecological tumors are common diseases among middle-aged and older women. A fallopian tube tumor is small relative to other gynecological tumors. The occurrence rate of fallopian tube tumors is approximately 0.14% to 1.80%.[1] Recent studies have shown that 40% to 60% of ovarian cancer or primary peritoneal cancer may originate from the fallopian tube.[2] Such tumors often

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have no typical symptoms in the early stage and are often missed clinically. We report a case of tubal serous adenocarcinoma with dizziness as the first symptom.

Case

A 53-year-old woman presented to our hospital because of subacute onset of paroxysmal vertigo, especially when standing up. Seven days after presenting to our hospital, her vertigo spontaneously resolved, but 3 days later, she complained of numbness in her left hand and difficulty controlling her right hand and leg. She was thought to have had a small stroke, and was treated with aspirin and statin in a primary hospital, but her symptoms failed to improve.

The patient denied a family history of genetic diseases and similar diseases. A physical examination showed horizontal nystagmus bilaterally and her speech was dysarthric. A cranial nerve examination was normal. There was moderate right arm and leg weakness (Medical Research Council grade of 4/5). Deep tendon reflexes were reduced in the upper extremities and absent in the lower extremities. The patient had marked ataxia on finger-to-nose and heel-to-shin testing with intention tremor. The Romberg test was positive with subtle sway with eye closure. A routine blood test and biochemistry examination were generally normal. A cerebrospinal fluid examination showed the following: cerebrospinal fluid protein level, 738 mg/L; and cerebrospinal fluid leukocyte count, 35×10^6/L. Magnetic resonance imaging showed small ischemic lesions scattered in the brain (Figure 1). A pelvic ultrasound examination showed a small amount of pelvic fluid (Figure 2). On the basis of the findings of ataxia, reduced tendon reflexes, nystagmus, and cerebrospinal fluid protein–cell separation signs and symptoms, the patient was diagnosed with variant Guillain–Barré syndrome.

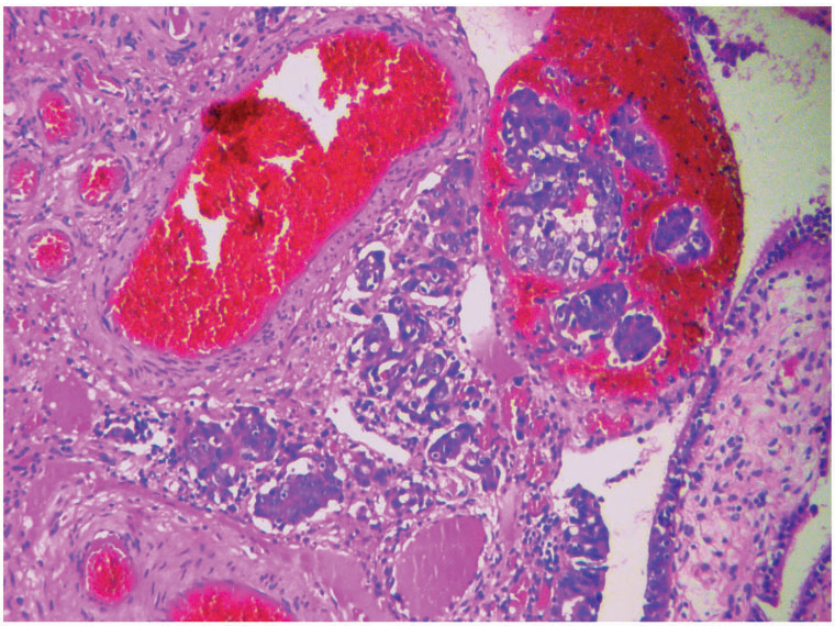

Figure 1. Skull magnetic resonance imaging shows cerebral ischemia.

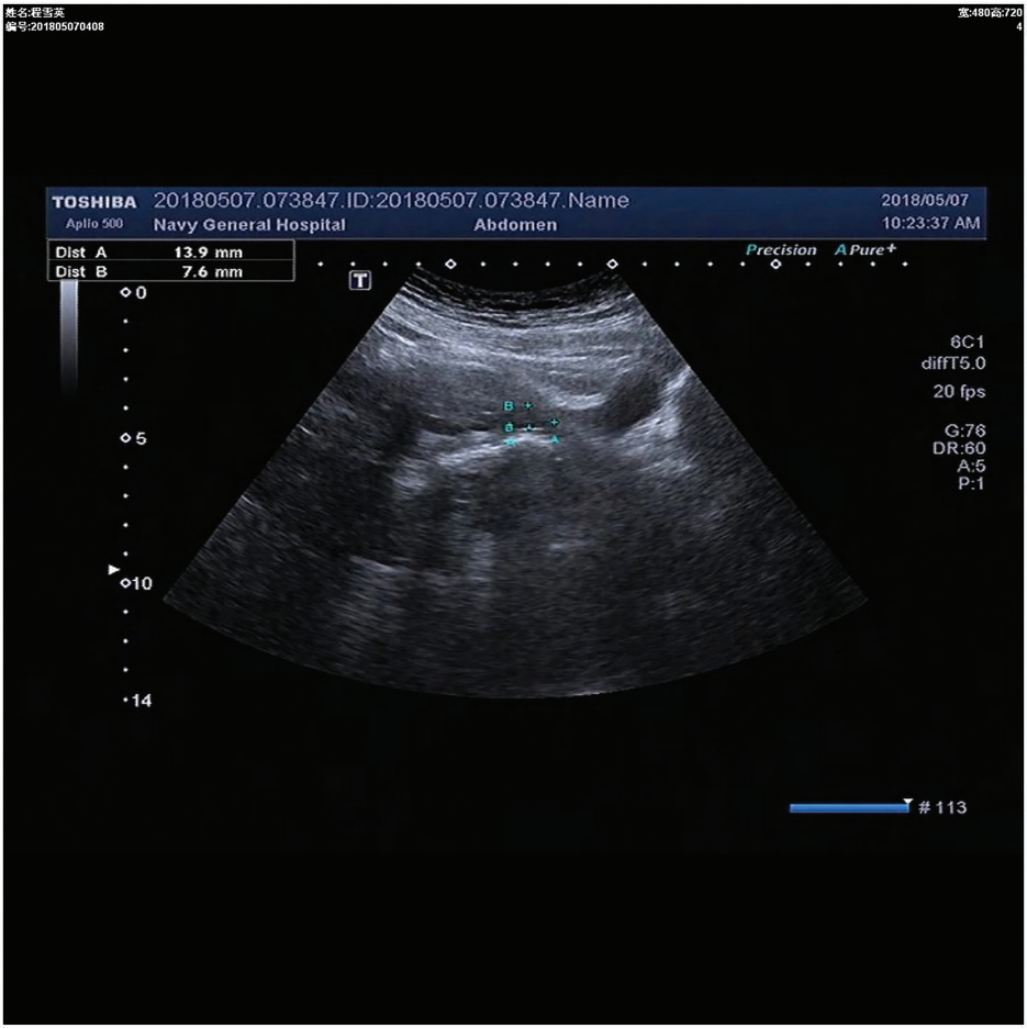

Figure 2. Transvaginal ultrasound shows a small amount of pelvic effusion.

The patient then received immunoglobulin therapy, but her dizziness had worsened, and she dared not to turn over in bed. A cerebrospinal fluid test showed that anti-Yo antibody was positive. After an obstetrics and gynecology consultation, a ThinPrep cytological test showed no intraepithelial lesions or malignant tumor cells. A human papilloma virus examination showed positivity for HPV52. A positron emission computed tomography (PET-CT) examination showed that the left uterine appendage area and retroperitoneal multiple lymph nodes had abnormally increased glucose metabolism (Figure 3). Furthermore, right cerebellar glucose metabolism was diffusely reduced. On May 28, the patient was operated on with laparoscopic bilateral uterine appendage resection and exploratory curettage. The right ovarian suspension ligament, the right mesosalpinx, and the proper ligament of the right ovary were cut by electrocoagulation

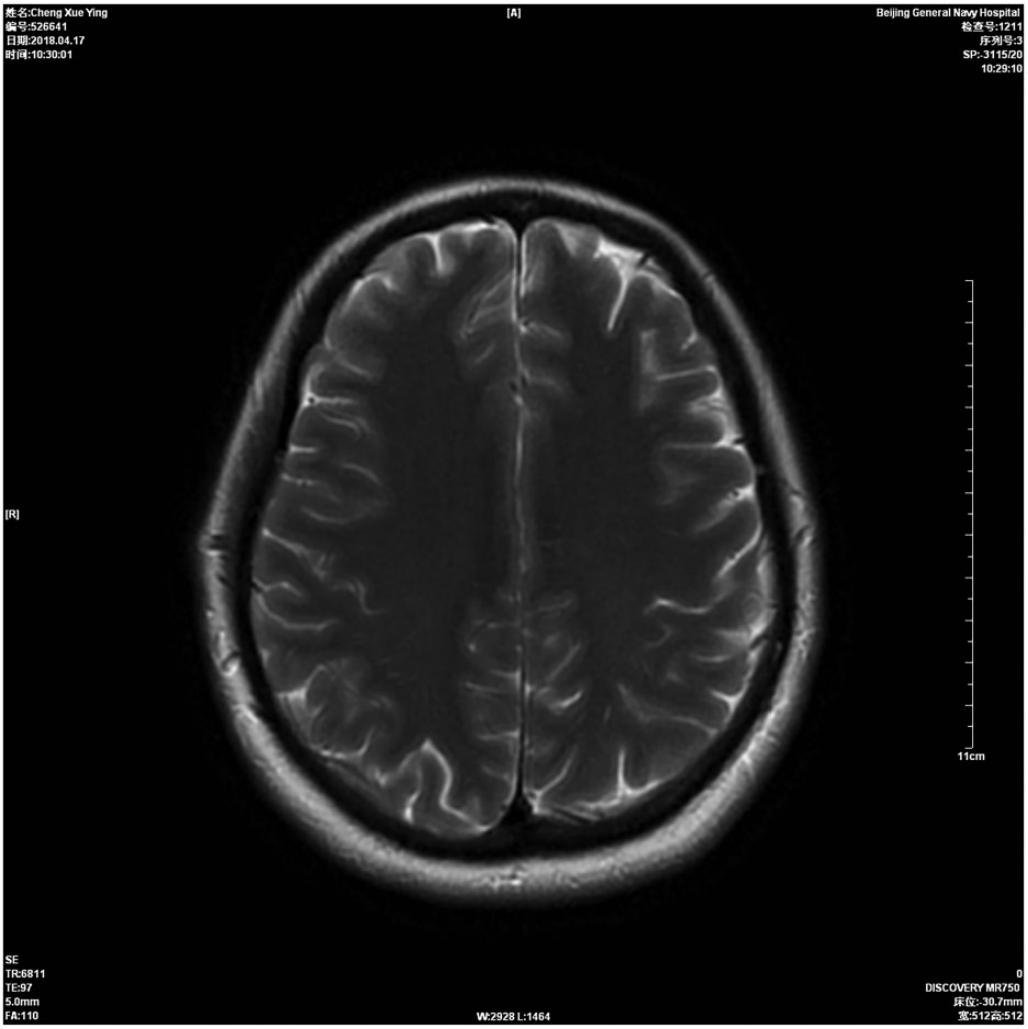

Figure 3. A positron emission computed tomography examination shows that glucose metabolism is increased in the left uterine appendage area and there is diffuse reduction of right cerebellar glucose metabolism.

and an ultrasound scalpel, and the right appendages were completely removed. A bag was inserted and removed from the left puncture hole. The left appendages were removed by the same method and a frozen pathological examination was carried out. Curettage of a small amount of endometrium was used for a pathological examination. The pathological diagnosis was that the left fallopian tube showed serous adenocarcinoma (Figure 4). The pathological examination also showed chronic salpingitis of the right fallopian tube, local epithelial hyperplasia of the fallopian tube, and no lesions in the right ovary. The final diagnosis was subacute cerebellar degeneration secondary to left fallopian tube serous adenocarcinoma (stage IC).

Surgery is the main treatment principle of this condition. The scope of surgery should include total hysterectomy, bilateral

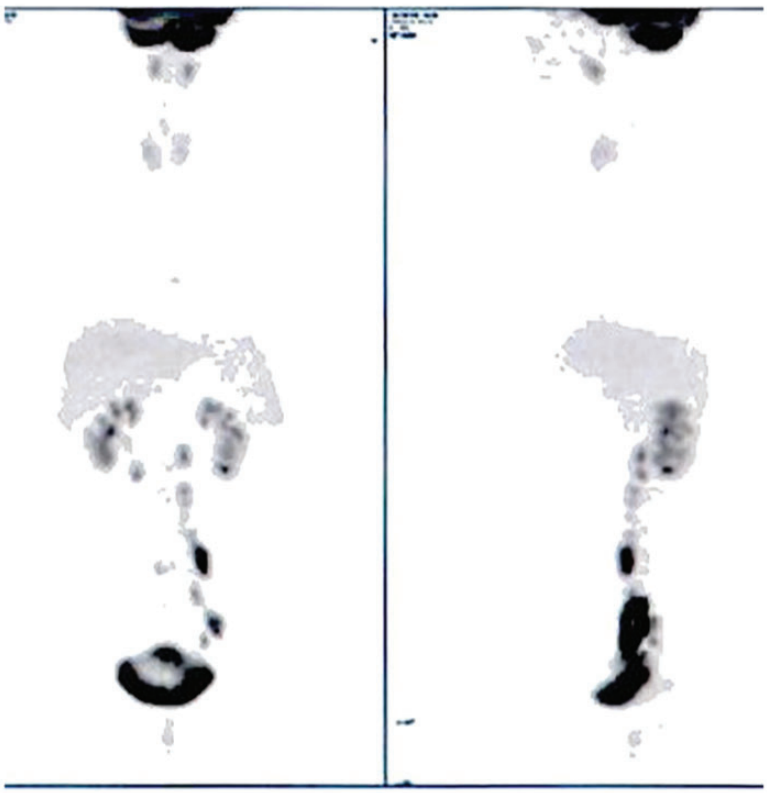

Figure 4. Pathological findings show fallopian tube serous adenocarcinoma.

appendage and omentum excision, and tumor reduction. Furthermore, pelvic lymphadenectomy, chemotherapy, and radiotherapy should be provided to the patient after the operation. However, our patient's general condition was poor, and she could not tolerate a staging operation. According to the National Comprehensive Cancer Network Guidelines for Ovarian Cancer, after aggressive surgical debulking of newly diagnosed ovarian cancer, most patients with stage IC should receive chemotherapy with paclitaxel and carboplatin for three to six cycles. Therefore, intravenous chemotherapy with paclitaxel 270 mg + carboplatin 450 mg was performed.

The study was approved by the institutional Ethics Review Board at The First Affiliated Hospital of Nanchang University, China. Informed consent was obtained from the patient for recording and publishing the disease-related information.

Discussion

Paraneoplastic neurological syndrome (PNS) is a group of neurological symptoms and signs caused by distant tumors. PNS is rare, and its occurrence rate is less than 1% among patients with malignant tumors. PNS can occur before, after, or at the same time as a tumor is discovered, and some cases of PNS even involve the first symptoms of malignant tumors.[3] The mechanism of PNS may be a cross-immunization reaction (i.e., anti-tumor antibodies cross

the neural antigen).[4] If a patient is found with characteristic antibodies, such as anti-Hu, Yo, and Ri, the incidence of tumors is >70%.[5] The above-mentioned characteristic antibodies are important indicators of primary tumor-related sites. An example of this indication is that anti-Yo antibodies are more common in most women with breast cancer and ovarian cancer.[6] According to the diagnostic PNS standard published by Graus et al.[7] in 2004, patients with PNS are characterized by a classical or non-classical clinical syndrome and characteristic tumor neurological antibodies (e.g., anti-Hu, Yo, Ri, Ma2, and amphiphysin). Furthermore, even if no tumors are found, patients are also diagnosed with PNS.

Our patient had subacute ataxia with a positive Romberg test result, dysarthria, and a decrease in tendon reflexes during a physical examination. Laboratory tests showed that the cerebrospinal fluid protein level was increased and the number of white blood cells did not exceed 50×10^6/L. No other positive test results were found. The diagnostic criteria of Miller Fisher Syndrome[8] are as follows: (1) at acute onset, the disease peaks within a few days or weeks; (2) clinically, the main symptoms are paralysis of the lateral rectus, ataxia, and decreased tendon reflexes, and the muscle strength of the limb is normal or mildly decreased; (3) protein–cell separation occurs in cerebrospinal fluid; and (4) the course of disease is self-limiting. Therefore, our patient received immunoglobulin therapy, but the symptoms of dizziness did not improve. In a review of the patient's cerebrospinal fluid test, anti-Yo antibody was found to be positive, which indicated the direction for our diagnosis. Although PET-CT showed that the patient had increased glucose metabolism in the left uterine appendage area, pelvic ultrasound only indicated a small amount of pelvic fluid, and no clear space-occupying lesions were found. After we consulted with the patient's family members and they agreed with our regimen, we conducted laparoscopic bilateral uterine appendage resection and exploratory curettage. The pathological diagnosis was left fallopian tube serous adenocarcinoma (stage IC). The patient's final clinical diagnosis was subacute cerebellar degeneration secondary to left fallopian tube serous adenocarcinoma (stage IC).[9]

We found six patients with paraneoplastic cerebellar degeneration with positive anti-YO antibody among 11,000 serum and cerebrospinal fluid samples in the literature.[9] Of the six patients, two had dizziness as the first symptom and all of them had ovarian cancer. In a study of small cell carcinoma of the lungs and paraneoplastic syndrome of the nervous system, 91.2% (31/34) of patients were treated on the basis of findings in the nervous system, and 20.6% (7/34) had subacute cerebellar degeneration. Therefore, our case is not an isolated case, but similar cases are relatively rare, and the number of these cases is small.[10]

Conclusions

The findings of our patient suggest that attention should be paid regarding the following aspects. (1) The pace of onset and progression of cerebellar ataxia in older people and those with ineffective conventional treatment provides a major clue to the etiology, as well as the absence of structural defects. Acute to subacute onset of ataxia in older people has limited causes and once stroke, hemorrhage, and a tumor are excluded by imaging, differential diagnosis is limited to infectious, inflammatory, toxic, nutritional, and paraneoplastic causes. A cerebrospinal fluid examination will further narrow the differential diagnosis. (2) Special attention should be paid to the patient's clinical syndrome performance to search for potential tumors. The presence of specific syndromes may suggest the most

likely type of tumor. If no tumor is found, follow-up after 6 months is recommended. (3) A PET-CT examination can help to confirm the diagnosis and early detection of small lesions of metabolic abnormalities to achieve the result of early detection, early diagnosis, and early treatment.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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