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Case Report

Retrospective Diagnosis of Fallopian Tube Cancer in a Case of Autoimmune Cerebellitis - PNS

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Abstract

Introduction: A remote underlying tumor causes neurologic impairments known as paraneoplastic neurological syndromes (PNS). PNSs arise from a tumor-elicited immune response against onconeural antigens that are shared by tissues of nervous system, muscle, and tumor cells. It has been revealed that PNS may precede the diagnosis of cancer in 50 - 80% of cases [1]. We describe a patient with subacute cerebellar degeneration whose fallopian tube serous adenocarcinoma was retrospectively diagnosed.

Case Report: A 51-year-old lady with bilateral lower limb weakness with imbalance and giddiness since 3 months; and was evaluated elsewhere diagnosed with right parafalcine meningioma - could not be operated due to bridging veins. On further evaluation the anti-Yo antibodies was detected in this case report which are almost exclusively associated with gynaecological tumours (Breast cancer and ovarian cancer) [3].

The discovery of onconeuronal antibodies, whose detection has been linked to certain tumor entities, was suggestive for the subsequent diagnosis of a paraneoplastic condition.

MRI brain showed cerebellar atrophy. The tumour markers CEA, CA 125 and AFP were in the normal ranges. Mammography and thorax CT were also unremarkable; on transvaginal sonography the internal genitals were inconspicuous except altered echotexture and increased vascularity in right ovary measuring 2.1 x 1 cm. On PET CT there was hypermetabolism in right ovary with metabolically active enlarged retroperitoneal lymph nodes, prominent left inguinal lymph nodes and marrow hyperplasia. Inguinal lymph node excision biopsy was negative for malignancy and bone marrow aspiration showed hemophagocytosis.

After consensual agreement a bilateral laparoscopic adnexectomy was performed but with unremarkable abdominal findings. The histological examination confirmed a high grade serous carcinoma of right fallopian tube. After a stage-related staging operation, the final classification was found to be the FIGO-IIIA1(ii) (2014) stage on account of positive retroperitoneal lymph nodes. Thus, adjuvant chemotherapy with 6 cycles of carboplatin and paclitaxel was planned and given.

Conclusion: The necessity of identifying the underlying malignancy in individuals with subacute neurological impairment is highlighted by this example, which also demonstrates that fallopian tube cancer can potentially cause PNS.

***Keywords**: Paraneoplastic Syndrome; Fallopian Tube Cancer; Anti Yo Antibodies; Onconeural Antibodies*

Introduction

A remote underlying tumor causes neurologic impairments known as paraneoplastic neurological syndromes (PNS). PNSs arise from a tumor-elicited immune response against onconeural antigens that are shared by tissues of nervous system, muscle, and tumor cells. It has been revealed that PNS may precede the diagnosis of cancer in 50 - 80% of cases [1]. Paraneoplastic sensory neuropathy (PSN) is probably the most common type of PNS (accounting for 3 - 7/1000 cancer diagnoses), followed closely by paraneoplastic encephalitis (PEM) (3/1000) and paraneoplastic cerebellar degeneration (PCD) (2/1000) [2].

Paraneoplastic neurological syndromes mostly manifest prior to the diagnosis of a tumour. These are rare and occur in less than 1% malignant tumor patients. In the past decades the number of and knowledge about antineuronal or, respectively, onconeuronal antibodies that are expressed by malignant tumours have increased markedly [3].

PNS begins as a peripheral immune response against autoantigens produced in malignancies. Onconeural immunity, a cancer-stimulated immune response that reacts with neural tissue, is thought to be the primary pathogenic cause underlying PNS. The oncoantigens that drive the immune response are normally restricted to the nervous system [4].

PCD is a rare but fatal neuronal syndrome associated with ovarian, breast and lung cancer patients. It is characterized by cerebellar atrophy with a diffuse loss of Purkinje cells, mediated by a cross-reaction of antibodies with tumor antigens and cerebellar tissue. PCD-related autoantibodies include: i) anti-Hu, ii) anti-Ri/Nova and iii) anti-Yo. Anti-Hu and anti-Ri/Nova are detected in patients with small cell lung and breast cancer, respectively. Anti-Yo, also called Purkinje cell cytoplasmic antibody type 1 (PCA-1), is usually associated with ovarian and other gynaecologic cancers [5]. The exact mechanism of Purkinje cell death in anti-Yo antibody-positive PCD is unknown and may be related to the activation of CD8+ T cells. Early pathological changes in anti-Yo antibody-positive PCD include infiltration of lymphocytes around the blood vessels, activation of microglia, and infiltration of CD8+ T lymphocytes into the cerebellar Purkinje cell layer. With disease progression, the pathological manifestation of PCD is mainly large and rapid loss of noninflammatory Purkinje cells [6].

Truncal and appendicular ataxia, dysarthria, vertigo, nystagmus, and diplopia are among the clinical signs of PCD that are often characterized by subacute onset but increasing pancerebellar degeneration [7].

The patient becomes severely incapacitated as a result of these symptoms, which worsen over weeks to months before stabilizing.

Because a better cancer prognosis is obtained if the tumor is discovered in its initial stage without spread, an early diagnosis is preferred. Also, since cerebellar degeneration, once developed, does not seem to improve either by tumor removal or through immune-modifying therapies including chemotherapy [8].

Early resection of such tumors is a significant part of PNS management and improves the outcome.

Case Report

A 51-year-old lady with bilateral lower limb weakness with imbalance and giddiness since 3 months and was evaluated elsewhere diagnosed with right parafalcine meningioma - could not be operated due to bridging veins. On further evaluation the anti-Yo antibodies was detected in this case report which are almost exclusively associated with gynaecological tumours (Breast cancer and ovarian cancer) [3]. The discovery of onconeuronal antibodies, whose detection has been linked to certain tumor entities, was suggestive for the subsequent diagnosis of a paraneoplastic condition.

MRI brain showed cerebellar atrophy. The tumour markers CEA, CA 125 and AFP were in the normal ranges. Mammography and thorax CT were also unremarkable; on transvaginal sonography the internal genitals were inconspicuous except altered echotexture and increased vascularity in right ovary measuring 2.1 x 1 cm. On PET CT there was hypermetabolism in right ovary with metabolically

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After consensual agreement a bilateral laparoscopic adnexectomy was performed but with unremarkable abdominal findings. The histological examination confirmed a high grade serous carcinoma of right fallopian tube. After a stage-related staging operation, the final classification was found to be the FIGO-IIIA1(ii) (2014) stage on account of positive retroperitoneal lymph nodes. Thus, adjuvant chemotherapy with 6 cycles of carboplatin and paclitaxel was planned and given. After taking 3 cycles of carboplatin and paclitaxel, she experienced severe neurological symptoms, which settled down after 1 month. Subsequently the Medical Oncologist initiated 2nd line chemotherapy, completing 6 cycles. The patient's HRD report was positive, hence maintenance therapy with Olaparib and Bevacizumab was given. Currently as of in January 2025, patient is doing good with no mobility of lower limbs, and is undergoing physiotherapy with follow up every 3 months with scan and CA-125. Her recent ultrasound and CA 125 in January 2025 is normal.

Discussion

Paraneoplastic neurological syndromes mostly manifest prior to the diagnosis of a tumour. These are rare and occur in less than 1% malignant tumor patients.

Finally, it has been suggested that fallopian adenocarcinomas are indistinguishable from ovarian adenocarcinoma. Indeed, the majority of the serous tumors appear to originate from dysplastic lesions in the distal fallopian tube, and what has been traditionally considered ovarian cancer may in fact be tubal in origin [9].

Therefore, if a PCD had not been expressed, it is likely that a primary fallopian tube adenocarcinoma diagnosed at an early stage would be regarded as an ovarian adenocarcinoma detected later.

Anti-Yo represents the most frequent immune-mediated PCD, and is almost always associated to gynaecological (ovarian) cancer [10].

Only slight to moderate improvements in neurological function have been observed in ovarian cancer patients after a combination of chemotherapy, intravenous Ig, and plasmapheresis. There are no established protocols for the treatment of most paraneoplastic syndromes. The physician may employ either plasma exchange or a combination of intravenous Ig and immunosuppressive agents, including corticosteroids or cyclophosphamide. Since the antibodies remain intrathecal and unaffected by plasmapheresis or intravenous Ig, there is often no benefit from these therapy, despite the odd claim of improvement. In the treatment of PCD patients, symptom alleviation is crucial. To maximise functional recovery, intensive rehabilitation, speech therapy, and psychological support are also essential [11].

In our patient, the typical time between the cancer diagnosis and the onset of cerebellar symptoms was around five months, as reported by Rojas., *et al*. [12].

Our patient was also initiated with immunomodulatory therapy regimens involving high intravenous Ig dosages and corticosteroids, post-onset of PCD; unfortunately, this treatment proved to be ineffective. Before PCD was diagnosed, there was already a significant and permanent neuronal loss, which may have contributed to the therapy's failure.

Lastly, there has been evidence to imply that ovarian and fallopian adenocarcinomas are identical. In fact, dysplastic lesions in the distal fallopian tube seem to be the source of most serous tumors, and what has historically been thought to be ovarian cancer may really have tubal origins. Despite this hypothesis, there are few reports of PCD in association with fallopian tube cancer [13-16].

It has been expected that a small percentage of women with ovarian adenocarcinoma will also have PCD, albeit the exact amount may differ greatly.

Therefore, if a PCD had not been expressed, an early-stage primary fallopian tube adenocarcinoma may have been mistaken for an ovarian adenocarcinoma that was discovered later.

Conclusion

Paraneoplastic cerebellar degeneration associated with anti-Yo (Purkinje cell) antibodies is almost invariably related to ovarian epithelial adenocarcinoma.

The patient with fallopian tube cancer, identified months after PCD, is the subject of this article. PCD and anti-Yo antibodies, however uncommon, should raise the possibility of a primary ovarian adenocarcinoma, independent of whether the tumor originated in the fallopian tube or the ovary.

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