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Case Report

Sequential Response of a Platinum-Sensitive Ovarian Cancer Recurrence to Olaparib after Previous Response to Niraparib - A Case Report

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Background

PARP (Poly (ADP) Ribose Polymerase)-inhibitors have recently been introduced in the treatment of patients with recurrent platinum-sensitive ovarian cancer and a BRCA-mutation. Niraparib resulted in a significantly prolonged Progression-Free Survival (PFS) in both BRCA-wild type as well as BRCA-mutated ovarian cancer recurrence in the ENGOT-OV16/NOVA Studie [1]. Similarly, olaparib revealed a significant PFS benefit in BRCA-mutated patients (Study 19 [2], SOLO2-Studie). The clinical course of a patient who received successfully subsequent treatment with the two PARP-inhibitors is described.

Case Report

A 56 years-old patient with hereditary ovarian cancer and a BRCA2-mutation was diagnosed with a FIGO stage IIIc high-grade serous ovarian cancer. Initially, she received 3 cycles of a platinum-based chemotherapy. After interval-debulking with no residual disease she received three additional cycles of chemotherapy plus trebananib within the TRINOVA 3 study. Due to pleural tumor progression, platinum-based chemotherapy was started again. After a partial remission to second-line chemotherapy, the patient received the PARP-inhibitor niraparib for a total of eight months within the NOVA-study. A complete remission of the pleural effusion was stated.

Due to further tumor progression, Niraparib was discontinued. The patient now received 6 cycles of carboplatin and gemcitabine. After achievement of a partial remission following third line chemotherapy, treatment with the second PARP-inhibitor olaparib was initiated. The latter agent was administered orally at 800 mg/Tag divided in two doses. The patient is currently alive and well with no evidence of disease. Toxicity was minimal. No nausea, vomiting, or diarrhea was observed (Table-1).

Date	Diagnosis/Event	Important Therapeutic Steps
Oct-12	Serous high-grade FIGO stage IIIc ovarian cancer	Explorative laparotomy
10/2012-12/2012	Neoadjuvant chemotherapy	3x Carboplatin + Paclitaxel + 1x Trebananib (angiogenesis inhibitor)
Jan-13	Interval-debulking surgery (no residual diseases)	Total abdominal hysterectomy, bilateral salpingo-oophorectomy hysterectomy, sigmoid resection, omentectomy, splenectomy, pelvic and paraaortic lymphadenectomy
2/2013-4/2013	Adjuvant chemotherapy + trebananib	Additional three cycles
5/2013-12/2013	Consolidation therapy	Trebananib
Dec-13	Development of bilateral glaucoma	Probable association with study medication. End of study treatment
Mar-14	Cytologically verified bilateral pleural effusion (right 6 cm, left 1.8 cm)	Karnofsky 80
3/2014-7/2014	Partial remission	6x Carboplatin/PEG liposomal doxorubicin
9/2014-7/2015	Left pleural effusion and lung metastasis 1.3 cm	Niraparib study medication (NOVA study): Partial remission
7/2015-9/2015	Progression of left pleural effusion + peritoneal carcinomatosis	3 cycles of carboplatin and gemcitabine

Sep-15	Carboplatin hypersensitivity reaction at cycle 3	Stop of carboplatin therapy
9/2015-12/2015	Change to 3 cycles of oxaliplatin monotherapy	
Dec-15	Partial remission of peritoneal carcinomatosis	Start with olaparib therapy
12/2015 ongoing (6/2017)	Continuous therapy with olaparib	Maximum toxicity: Grade 1 anemia, grade 1 peripheral sensory neuropathy
Jun-17	Radiologically, biochemically and clinically no evidence of disease	Karnofsky score 90

Table 1: Overview of the clinical course of disease in the 56 years-old patient with FIGO IIIc recurrent ovarian cancer.

Conclusion

Targeted therapy with PARP-inhibitors in patients with BRCA-mutated ovarian cancer represents a relatively new therapeutic option. Our patient is now in complete remission 4.5 years after primary diagnosis of an advanced high-grade serous ovarian cancer deveoping its first recurrence as early as 15 months after primary diagnosis. After several platinum-based chemotherapies including angiogenesis inhibitors and treatment with two different PARP-inhibitors the patient is now still in remission. This case illustrated that there may exist no cross-resistance between niraparib and olaparib.

References

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