AMH-Breast Cancer
Karaçetin D1*, Hale A2, Didem CT3, Cigdem UA4, Canan K5, Cihan O6, Murat U7 and Selim A8

1Department of Radiation Oncology, Bakırköy Dr. Sadi Konuk Research and Training Hospital, Turkey
2Department of Medical Biochemistry, Istanbul Education and Research Hospital, Turkey
3Department of General Surgery, Istanbul Education and Research Hospital, Turkey
4Department of Medical Oncology, Istanbul Education and Research Hospital, Turkey
5Department of Pathology, Istanbul Education and Research Hospital, Turkey
6Department of Medical Biochemistry, Edirne Government Hospital, Turkey
7Department of Medical Biochemistry, Giresun University School of Medicine, Turkey
8Department of Gynecologic Oncology, Istanbul University Faculty of Medicine, Turkey

Letter to the Editor

Globally, Breast Cancer (BC) is the most frequently diagnosed malignancy, accounting for over a million cases each year [1]. It is also the leading cause of cancer death in women worldwide. Premenopausal (PM) women with BC can develop ovarian failure when treated with chemotherapy. To accurately predict ovarian function after chemotherapy is important for both adjuvant endocrine therapy decision making and future fertility planning [2]. Serum Anti-Müllerian Hormone (AMH) concentration has been shown to be the best biochemical marker for assessment of decline in reproductive capacity in healthy women [3].

We conducted a preliminary study evaluating the pretreatment levels of AMH in BC patients with the approval of our institution’s ethical committee. At the beginning, we designed the study including 35 patients, and 25 healthy controls. All BC patients were PM according to their menstrual cycles, FSH, LH and E2 levels and we excluded women having AMH levels below 0.16 ng/mL. Women with increased levels of anti-thyroperoxidase (>28 U/L) and thyroid stimulating hormone levels (>5.0 µIU/mL) were also excluded. Fourteen women (aged 24 to 46; median 40.5) who received no anti-cancer therapy made up our patient group for the statistical analysis. Nine healthy women (aged 34 to 48; median 39) without known breast cancer which was confirmed mammulo-sonographically, made up our control group. In the patients’ group, 8 of them had AMH levels below 0.8 ng/mL before any therapy. AMH was measured by enzyme-linked immunoassay method (Ref: A79765; Beckman Coulter, USA); blood samples were taken before Chemotherapy (CT) (first sample), just after CT-before radiotherapy (second sample), and after radiotherapy (third sample). Reference intervals for AMH were reported as follows; normal range: 1.0 ng/mL to 5.0 ng/mL; residual ovarian reserve: 0.8 ng/mL to 1.0 ng/mL; menopause <0.1 ng/mL. We classified AMH levels below 0.8 ng/mL as indicating ovarian dysfunction, and used chi-square in statistical analysis.

There was no difference in percentage of ovarian dysfunction between the patients and the controls before the anti-cancer therapy but there was a significant difference between the first, second and the third samples according to AMH levels (p=0.003). Decrease in AMH levels was noticed during intervals of therapy, nearly 3 months’ period of time between first, second and third blood sampling. But we also found that in PM patients with BC; some of them can have lower AMH levels which is a finding which shows us that their ovarian reserve is too low to bear a child.

This is a preliminary study with a very small number of individuals, encouraging further studies about long-term cellular effects of anti-cancer therapy on ovarian tissue in breast cancer; blood sampling for AMH in females below 40, besides questioning the menstrual cycles, smoking, and any other life style habits. Future studies will explore potential applications AMH levels that might help clinicians prescribe appropriate hormone agents for specific subgroups of patients.

References