Advanced Oncology Certified Nurse Practitioner

REVIEW COURSE 2024

October 10-12, 2024 | Houston, TX

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

1. Which statement **<u>is not true</u>** regarding the use of hormone therapy in the treatment of breast cancer?

Answer is: B

A. Hormone therapy begins after local treatments for HR+ breast cancer are completed and continues for 5 -10 years depending on risk for recurrence

B. Hormone therapy is considered as curative intent in the locally advanced HR+ breast cancer patient

C. Status of estrogen/ progesterone receptor is needed before hormone therapy is prescribed

D. If aromatase inhibitors are prescribed in premenopausal women, ovarian suppression must be addressed with either oophorectomy or concurrent use of GnHR agonist

A, C, and D are true statements.
Before starting hormone therapy for breast cancer, the ER/PR status and the menopausal status of patient must be identified. Only patients who are ER/PR + are eligible to receive hormone therapy. Selection of agent selected also depends on the pretreatment menopausal status of the patient. For early stage disease, hormone therapy for HR+ disease is started after completion of local treatments i.e. after surgery and/or after completion of radiation therapy. In early stage disease, the local treatment of surgery or radiation are considered curative, but hormone therapy is to reduce risk of recurrence. Reference NCCN, 2024

2. Which androgen deprivation therapy agent has to be administered with a corticosteroid to decrease side effects of mineralocorticoid excess?

A. Luteinizing hormone-releasing hormone (LHRH) agonists such as Leuprolide, Goserelin

B. LHRH antagonists such as Degarelix or Relugolix

Answer is: C

C. Abiraterone, the androgen synthesis Inhibitor (CYP17 Inhibitor)

D. Antiandrogens such as the 2nd generation agents Enzalutamide, Apalutamide, Darolutamide

Besides androgens, the adrenals also produces cortisol (glucocorticoid) and aldosterone (mineralocorticoid).
CY17A1 is an enzyme crucial for the production of for both androgen and cortisol.
Administration of the CY17A1 inhibitor, Abiraterone, results not only in stopping androgen production (the desired effect), but also decreases production of cortisol and mineralcorticoid (an undesired effect). A low cortisol level triggers excessive adrenocorticotropic hormone (ACTH) release via a negative feedback mechanism resulting in
excess mineralocorticoid production with the subsequent fluid retention, hypokalemia and hypertension
By taking prednisone alongside abiraterone, the body is provided with a sufficient level of cortisol to minimize the compensatory increase in adrenocorticotropic hormone (ACTH) thus avoiding mineralcorticoid excess.

LHRN analogues (both agonists and antagonists) nor antiandrogens have any effect on cortisol production thus would not create an excess of ACTH production due to low cortisol. Reference Shaffi et al., 2024; NCCN, 2024

3. Which statement **is true** regarding use of androgen deprivation therapy (ADT) in the treatment of prostate cancer?

A. ADT therapy begins after local treatments for prostate cancer are completed and continues for 5 years or until disease progression

B. ADT therapy is used as curative intent in early stage prostate cancer

Answer is: D

C. Status of androgen/testosterone receptor is needed before ADT is prescribed

D. ADT therapy is indicated for treatment of locally advanced or metastatic prostate cancer and/or when localized disease has progressed after other treatment

ADT therapy is indicated when patient is diagnosed with locally advanced or metastatic prostate cancer and/or when localized disease has progressed after other treatment, usually evidenced by imaging and/or by rising PSA.

ADT has no role in early stage disease where local treatments are utilized. ADT is not curative intent, rather used to slow growth, reduces symptoms and prolong life

Androgen receptor status is not a biomarker in prostate cancer (unlike the evaluation of ER/PR status in breast cancer).

Ref: NCCN, 2024

4. What is the most lethal acute side effect of the biological agent, interleukin 2?

A. Capillary leak syndrome

B. Skin toxicity

C. Fever, chills

D. Prolonged cytopenia

Answer is: A

- Although not problematic, the skin toxicity and fever chills are not lethal side effect.
- Prolonged cytopenia can become lifethreatening, but are not acute side effects, happens over time
- The capillary leak syndrome is an acute occurring side effect and is definitely life-threatening with the hypotension, hypoperfusion of the kidneys, and the tissue overload picture especially if the tissue overload is manifested by the lungs i.e. pulmonary edema with hypoxia

Ref: Jeong, et al., 2019

5. A 35 year old Female diagnosed with early stage ER/PR + breast cancer has completed her surgery and radiation treatments. She remains premenopausal after these treatments and is to begin her endocrine therapy with an aromatase inhibitor. What must be included in her regimen?

A. A selective estrogen receptor down regulator (SERD)
B. A luteinizing hormone-releasing hormone (LHRH) agonist
C. A corticosteroid
D. A selective estrogen receptor modulator (SERM)

Answer is: B

- Aromatase inhibitors target the enzyme aromatase which converts androgen (produced by adrenals, fat cells and other tissues in women) to estrogen. Since the ovaries are still actively producing estrogen in premenopausal women, an aromatase inhibitor alone won't significantly lower estrogen levels without additional ovarian suppression.
- An LHRH antagonist signals the body to stop ovarian production of estrogen. These antagonists suppress the production of FSH which is necessary for ovaries to produce estrogen.
- A SERM (tamoxifen) can be used in pre or post menopausal. Corticosteroids have no role in endocrine therapy for breast cancer. A SERD would also need to have additional ovarian suppression if given to a premenopausal women, but is not in this patient's regimen. Ref Bradley et al., 2022; NCCN, 2024