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SEDE DEL CORSO:
Fondazione Policlinico Universitario Agostino Gemelli IRCSS,
Largo Agostino Gemelli, 8 Roma
aula 617, VI piano ala A

DESTINATARI DELL'ATTIVITÀ FORMATIVA: Medico Chirurgo (specializzazioni: anatomopatologi, ginecologi, oncologi, senologi, radioterapisti) e Infermieri.

CREDITI ECM:
Il corso ha ottenuto 5 crediti ECM

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Uptodate cyclin inhibitors: how increase the bar

4Marzo2019

UNIVERSITÀ CATTOLICA del Sacro Cuore
OB&GYN JOURNAL CLUB

Presidente del Corso:
Prof. Giovanni Scambia

Responsabile Scientifico:
Dott.ssa Ida Paris

Fondazione Policlinico Universitario Agostino Gemelli IRCSS, ROMA

PROGRAMMA

09.30 Registrazione partecipanti
10.00 Saluto delle autorità ed introduzione al corso – G. Scambia, R. Masetti

Sessione 1: Moderatori – E. Bria / P. Vici
10.30 Come è cambiato lo scenario terapeutico nel management del tumore della mammella luminale HER2-. T. Gamucci
10.50 Differenze farmacologiche tra CDKi - R. Danesi

Sessione 2: Moderatori – A. Astone / G. Zampa
11.10 Differenze cliniche tra inibitori delle cicline (CDKi) - A. Fabi
11.30 Recenti acquisizioni in tema di CDKi e prospettive future - I. Paris

11.50 Coffee break

LETTURA MAGISTRALE - E. D. Capoluongo
12.10 Liquid Biopsies in metastatic breast cancer: what is the impact on patient outcome and prediction of early progressive disease. Talk about a management change

Debate: P. Marchetti – D. Santini – G. Naso – E. Capoluongo
13.15 Clinical case presentation - G. D'Auria, L. Carbognin

13.45 Light Lunch

Sessione 3: Moderatori – M. Mauri / G. Zampa
14.45 Pros & Cons about CDKi for each clinical case.
Clinical case 1: Cons B. Di Cocco
Pros I. Portarena
Discussant: Opinion Leader
Pros & Cons about CDKi for each clinical case
15.45 **Clinical case 2:** Cons L. Pizzuti
Pros N. Salesi
Discussant: Opinion Leader
16.45 Conclusions – I. Paris
17.00 Test ECM e chiusura lavori

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how increase the bar

RAZIONALE

CDK4 and CDK6 are cyclin-dependent kinases that control the transition between the G1 and S phases of the cell cycle. The S phase is the period during which the cell synthesizes new DNA and prepares itself to divide during the process of mitosis. CDK4/6 activity is typically deregulated and overactive in cancer cells. There can be amplification or overexpression of the genes encoding cyclins or of the genes encoding the CDKs themselves. Additionally, loss of endogenous INK4 inhibitors, by gene deletion, mutation, or promoter hypermethylation, can also lead to overactivity of CDK4 and CDK6.

A major target of CDK4 and CDK6 during cell-cycle progression is the retinoblastoma protein (Rb). When Rb is phosphorylated, its growth-suppressive properties are inactivated. Selective CDK4/6 inhibitors "turn off" these kinases and dephosphorylate Rb, resulting in a block of cell-cycle progression in mid-G1. This causes cell-cycle arrest and prevents the proliferation of cancer cells. Although the initial response to a selective CDK4/6 inhibitor is typically cell-cycle arrest, in some cases arrested cells enter a state of senescence. Understanding the determinants of whether a cell undergoes reversible G1 arrest or enters a senescent state is an important research area. Cancer cells entering senescence may undergo gradual regression over time; it is in such cancers that CDK4/6 inhibitors may produce the greatest clinical benefit.

This mechanism of action is known in estrogen receptor–positive breast cancer and seems to be dependent upon CDK4 for proliferation. To date, estrogen receptor–positive breast cancer is the malignancy for which this class of drugs has proven most effective and for which we have the most mature data from randomized trials comparing endocrine therapy alone with endocrine therapy combined with CDK4/6 inhibition.

This meeting would like to review the mechanisms of action and efficacy of these drugs in order to evidence the best choice in clinical management of estrogen receptor–positive metastatic breast cancer with special point of view in their future development.