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In North America, one out of every nine men will be diagnosed with prostate cancer at some point in their lives, and 10–20 percent of men diagnosed with PCa will develop castration-resistant prostate cancer within ve years of diagnosis. Although CRPC is classi ed as metastatic or non-metastatic, the value of early detection is still debatable, as roughly 33% of non-metastatic CRPC develops bone metastases within two years. In castrated patients, CRCP is de ned as a serum testosterone concentration of less than 50 mg/dl and one of the following: two or more new lesions in bone or so tissue, or three consecutive increases in PSA one week apart, resulting in two 50 percent elevations over the nadir and a PSA concentration of greater than 2 mg/ml. While there is a wide range of therapy options available, despite the fact that surgery, radiation, androgen deprivation, and chemotherapy have all been utilised for decades, metastatic and castration-resistant illness has few treatment choices [1].

e goal of GnRH agonists and antagonist's therapy is to stop testosterone from being produced in the testes. GnRH is produced in the brain and drives pituitary production of luteinizing hormone, which leads to testosterone synthesis in Leydig cells. Both medicines have been shown to reduce testosterone levels, but adrenal testosterone production, like androgen synthesis in cancer cells, is still possible. GnRH agonists disrupt this system by causing continuous GnRH ls lasting 1–2 weeks. It's to blame for some of the adverse e ects. e ' are phenomenon' is a condition that occurs in some people. To avoid side e ects, antiandrogens or estrogens must be used before and a er the procedure [2].

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