Chapter I

Introduction

Polycystic ovary syndrome (PCOS) is a common complex and heterogenous endocrine disorder that is characterized by oligomenorrhea or amenorrhea, hyperandrogenism, and multiple small subcapsular cystic follicles in the ovary on ultrasonography (Frank, 1995). It affects ≤10% of women of reproductive age (Vigil *et al*., 2007), with approximately 16%–80% of the affected women being obese (Dunaif, 1992).

Polycystic ovary syndrome frequently is associated with insulin resistance (IR) accompanied by compensatory hyperinsulinemia (Burghen *et al*., 1990), and IR is enhanced by the obesity and the syndrome (Dunaif, 1994). Whether hyperinsulinemia in PCOS is related to a defect in insulin action (Dunaif, 1989), to increased insulin secretion (Holte *et al*., 1994), to decreased hepatic clearance of the hormone (Ciampelli, 1997), and/or to a combination of these mechanisms is, however, under investigation.

Currently, accepted diagnostic criteria are based on a consensus developed at the 1990 National Institute of Health and Human Development Consensus Definition (Zawadzki and Dunaif, 1992). Such criteria require the presence of hyperandrogenism and chronic anovulation in the absence of specific diseases of the adrenal gland, ovary or hypophysis that may mimic PCOS, such as non-classical 21-hydroxylase deficiency, hyperprolactinaemia or androgen-secreting tumours (Zawadzki and Dunaif, 1992).

However, the recommendations arising from a conference sponsored by the European Society for Human Reproduction and Embryology and the American Society for Reproductive Medicine (ESHRE/ASRM) in 2003 suggested that evidence of polycystic ovaries in ultrasonographic scans could also serve as one of the diagnostic criteria for PCOS (Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004).

Ever since the beginning of the 1980s, there has been evidence of a significant correlation between the levels of androgens and insulin in patients with PCOS (Burghen *et al*., 1980). In this regard, these patients show a compensatory hyperinsulinaemia caused by the underlying IR.

Vitamin D is the "sunshine vitamin" that can play a very important role in dealing with PCOS and improving the overall health. Unfortunately, women with PCOS tend to have lower than adequate vitamin D levels. The role of vitamin D3 in insulin secretion and insulin sensitivity also has been well established by in vivo studies in both human and animal models. Vitamin D deficiency has been shown to be associated with impaired glucose clearance and insulin secretion. The exact mechanism, however, has not yet been elucidated fully and probably involves both direct and indirect actions of vitamin D3 (Kotsa et al., 2008).

Vitamin D deficiency continues to be a worldwide problem. The various factors that may contribute to vitamin D deficiency or insufficiency in relation to bone health among Saudi women are not known (Ardawi et al., 2010). Therefore, this study was designed to evaluate the vitamin D status in relation to insulin resistance, serum 25- hydroxyvitamin D [25(OH)D], serum intact-parathyroid hormone (PTH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), fasting serum insulin and fasting serum glucose in healthy Saudi women with or without PCOS. This can be investigated through studying the biochemical parameters in the serum of the subjects. Bone Mineral Density (BMD) was also demonestrated by dual energy X-ray absorptiometry (DXA), at the lumbar spine (L1-L4) and mean femur neck