INTRODUCTION

Anti-Müllerian hormone (AMH), also called Müllerian inhibiting substance (MIS), has been known for several decades to be responsible for establishing sexual dimorphism. Under the influence of AMH—produced by Sertoli cells in the emerging testes—the Müllerian ducts involute in male fetuses while androgens induce the Wolffian ducts to form epididymides, vasa deferentes, and seminal vesicles ***(Josso et al., 2006)***.

In the male, AMH level raises rapidly after birth, is highest during late infancy, then gradually declines until puberty and can therefore serve as a testis-specific indicator for diagnosis in newborn infants with ambiguous genitalia or nonpalpable gonads ***(Bergada et al., 2006)***. Blood concentrations of AMH decrease dramatically after puberty and remaining very low in adulthood ***(Fujisawa et al., 2002)***.

The regulation of AMH after birth is complex: basal levels of AMH are independent of gonadotropin regulation, for example, during childhood and in patients with hypogonadotropic hypogonadism. Throughout pubertal development, AMH correlates negatively with serum testosterone, this correlation persists if androgen levels are abnormally high (e.g., activating mutation of the luteinizing hormone (LH) receptor) but gonadotropins are low. These findings suggest that

AMH is down-regulated by androgens and not directly by gonadotropins ***(Wilhelm et al., 2007)***.

The decrease of AMH corresponds to the Sertoli cell maturation status: with the onset of puberty and spermatogenesis, AMH is expressed only by immature Sertoli cells and by immunohistochemistry found only in tubules with spermatogenic arrest or Sertoli-cell–only syndrome ***(Maymon et al., 2002)***.

However, its role in adults is not clear. It has been postulated that AMH controls Leydig cell proliferation and steroidogenic function ***(Racine et al., 1998)*** and may also be related to germinal cell proliferation ***(Cazorla et al., 1998)***.

AMH is secreted both into the seminiferous tubules and into the circulation. Since AMH is secreted predominantly by the apical pole toward the lumen of the seminiferous tubules, its concentration in seminal plasma is high. Although AMH is secreted predominantly into the seminiferous tubules, studying serum samples may be more advantageous than examining seminal plasma because the presence of seminal proteases may influence AMH levels in the latter ***(Isikoglu et al., 2006)***.

High AMH levels have been noted in patients with delayed puberty, whereas boys with precocious puberty had lower AMH levels than normal boys of the same age ***(Rey et al., 1993)***. Because of the thorough characterization of AMH levels, it is possible that serum AMH could be used to evaluate pubertal onset. In normal testes the switch off of AMH expression is usually associated with the terminal differentiation of Sertoli cells and the appearance of primary spermatocytes make it a good candidate for staging pubertal development in boys. AMH levels correlate better with developmental age than with chronological age ***(Rockett et al., 2004)***.

Very few studies are present concerning AMH levels in different age groups since delivery till senility. This hormone had not been studied in old age males. Its role in constitutional delayed puberty is obscure and the results are still controversial.

Aim of the work

The aim of this study is to evaluate the level of AMH in different aged groups of healthy men since delivery till senility, and to evaluate the AMH level in a group of boys complaining of constitutional delayed puberty.