Objective: Although shown to be effective for their intended medical treatment, AAS have been shown to induce hypogonadotrophic hypogonadism in adult males. The medical literature is conflicting in the reports of spontaneous return and long-term suppression of gonadal suppression post AAS usage. This observational study documents the treatment protocol of HCG, clomiphene citrate, and tamoxifen in returning hormonal function to normal post AAS usage. Design: Five HIV-negative males age 27-49, weighing 77-100 kg, with serum total testosterone levels below 240 ng/dL and luteinizing hormone (LH) levels below 1.5 mIU/mL were considered for this observational study. All five patients were administered the treatment protocol.

Methods: Treatment consisted of combination therapy which included concurrent administration of (a) Human Chorionic Gonadotropin, (b) Clomiphene Citrate and (c) Tamoxifen Citrate for a standard duration of 45 days. This protocol was repeated with every patient until serum LH and total testosterone values reached normal ranges.

Results: All five patients were considered eugonadal by normal laboratory reference ranges by the conclusion of treatment. Average serum total testosterone rose from 98.2 to 692.8 ng/dL (p<.001) while the average serum LH rose from an average undetectable value of less than 1.0 to 7.92 mIU/mL (p<.0008).

Conclusions: Although the treatment protocol of HCG, clomiphene citrate, and tamoxifen proved beneficial in reversing AAS induced hypogonadotrophic hypogonadism, future controlled studies...
INTRODUCTION

While AAS have proven effective in cases of lean muscle wasting conditions (HIV/AIDS), this class of medicines is not without their inherent problems. AAS have been shown to induce hypogonadotropic hypogonadism (Alen et al, 1987; Bhasin et al, 1996; Bijlsma et al, 1982; Clerico et al, 1981; Jarow & Lipshultz, 1990; Strawford et al, 1999; Stromme et al, 1974). This condition typically results from an abnormality in the normal functioning of the hypothalamic-pituitary-gonadal axis (HPGA), usually from a negative feedback inhibition of one of the hormone secreting glands, causing a cascading unbalance in the rest of the axis. Possibly resulting from a physiological abnormality (i.e. mumps orchitis, Klinefelter’s syndrome, pituitary tumor) or as an acquired result of exogenous factors (i.e. androgen therapy, AAS administration). Clerico et al (1981) found a dramatic suppression of serum gonadotropin levels in athletes given methandrosteneolone, suggesting a direct action of AAS on the hypothalamus. Similar results of suppressed gonadotropins have been found in patients supplementing solely testosterone (Bhasin et al, 1996; Marynck et al, 1979; Strawford et al, 1999; Tenover, 1992). Case report studies discussed a 36-year old male competitive bodybuilder and a 39-year old father, each using various AAS regimens over extended periods of time, who showed a blunted response to GnRH stimulation tests (Jarow & Lipshultz, 1990). One particular study administered 600 mg of nandrolone decanoate to 30 HIV-positive males over twelve weeks (Sattler et al, 1999). The results made no reference to LH or testosterone levels. The lack of gonadotropin measurement is
puzzling as the data showed 12 of 30 subjects experienced testicular shrinkage, implying Leydig cell dysfunction and suppressed testosterone levels. Other studies using AAS have also shown no reference to LH or FSH levels but suppressed values are expected in each case (Bagatell et al, 1994; Behre et al, 1997; Sheffield-Moore et al, 1999; Tricker et al, 1996).

Declining, or suppressed, circulating testosterone levels as a result of either pathophysiological or induced hypogonadal conditions can have many negative consequences in males. Declining levels of testosterone have been directly linked to a progressive decrease in muscle mass (Mauras et al, 1998), loss of libido (Schiavi et al, 1991), decrease in muscular strength (Balagopal et al, 1997; Mauras et al, 1998) impotence (Rakic et al, 1997), oligospermia or azoospermia (Vermeulen & Kaufman, 1995), increase in adiposity (Mauras et al, 1998) and an increased risk of osteoporosis (Wishart et al, 1995). While some research suggests that the hormonal axis will spontaneously return to normal shortly after cessation of testosterone administration (Knuth et al, 1989), documented cases have taken up to 2 ½ years to return to normal (Jarow & Lipshultz, 1990). This case of a 39-year old male who previously used AAS was found to have low serum testosterone levels (6nmol/L, range 14 to 28 nmol/L) 2 ½ years after his last administration of the drugs (Jarow & Lipshultz, 1990). For most men, suffering with diminished libido, impotence, depression, fatigue, muscle atrophy, and infertility for 2 ½ years is not a pleasant option. Other androgen or anabolic steroid induced cases of hypogonadotropic hypogonadism have taken 6 months (Gazvani et al, 1997; Wu et al, 1996), 8 months (Gazvani et al, 1997), 10 months (Boyadjiev et al, 2000), 12 months (Schurmeyer et al, 1984), and 18 months (Gazvani et al, 1997) to finally return to eugonadal status.

The individual use of human chorionic gonadotropin (HCG), clomiphene citrate, and tamoxifen citrate in the treatment of testicular sub-function and gonadotropin suppression, respectively, is well documented. HCG has been shown to significantly improve gonadal function in hypogonadotropic hypogonadal adult males (Barrio et al, 1999; Burgess & Calderon, 1997; Cisternino et al, 1998; D’Agata et al, 1982; 1984; Dunkel et al, 1985; Kelly et al, 1982; Ley & Leonard, 1985; Liu et al, 1988; Martikainen et al, 1986; Okuyama et al, 1986; Ulloa-Aguirre et al, 1985; Vicari et al, 1992). Studies using clomiphene citrate to induce endogenous gonadotropin production in males found significant improvements in LH and FSH values after treatment (Bjork et al, 1977; Burge et al, 1997; Guay et al, 1995; Landefeld et al, 1983; Lim & Fang, 1976; Ross et al, 1980; Spijkstra et al, 1988). Tamoxifen citrate has also been found to produce a profound increase in serum LH levels as well as improved semen and sperm quality (Gazvani et al, 1997; Krause et al, 1985; Lewis-Jones et al, 1987; Wu et al, 1996). As HCG’s effect is centralized at the Leydig cells of the testicles, clomiphene citrate and tamoxifen citrate act upon the hypothalamic-pituitary region in stimulating gonadotropin production. Tamoxifen, a nonsteroidal antiestrogen, and clomiphene citrate, a nonsteroidal ovulatory stimulant, compete with estrogen for estrogen receptor binding sites, thus eliminating excess estrogen circulation at the level of the hypothalamus and pituitary and allowing gonadotropin production to resume normally. The normal operation of both the testicular and hypothalamic-pituitary regions is crucial in returning HPGA function to normal. Returning one component of the axis to normal without concurrently returning the other would sabotage and inhibit the operation of the entire HPG axis. It was with this understanding that HCG was eventually combined with clomiphene citrate and tamoxifen as attempted therapy to reverse gonadal
function in hypogonadotropic hypogonadal males. In accordance with previous studies, each medication was used individually, and along with HCG, in initial trials. The simultaneous use of clomiphene citrate and tamoxifen was determined through preliminary use of clomiphene citrate and tamoxifen individually. **It was discovered that although both clomiphene citrate and tamoxifen met with some success, when combined together they achieved a more significant increase in gonadotropin production. This clinical outcome resulted in the combination therapy of HCG, clomiphene citrate and tamoxifen.**

Following is a clinical evaluation of the combined, simultaneous use of HCG, clomiphene citrate, and tamoxifen citrate as a treatment option in suppressed testosterone and gonadotropin levels in hypogonadotropic hypogonadal adult males. This observational analysis of the aforementioned treatment protocol assessed the efficacy of these medicines under non-controlled conditions.

**METHODS**

An observational study was done on the medical records of 5 adult male patients presenting to a clinic with induced hypogonadotropic hypogonadism. Patients were monitored and treatment recorded for the purposes of this observational study.

**SUBJECTS**

The medical records of five males age 27-49, mean 35.2, weighing 77-100 kg, mean 89.8 kg, with serum total testosterone levels below 240 ng/dL and serum luteinizing hormone (LH) levels below 1.5 mIU/mL were examined. Average presenting testosterone level was 98.2 ng/dL (normal= 240-827 ng/dL) while average LH level was undetectable at <1.0 mIU/mL (normal= 1.5-9.3 mIU/mL). The 5 patients had a history of AAS usage ranging from 9-60 months prior to presentation. All patients had ceased any testosterone therapy or AAS usage prior to initiation of treatment. Initial laboratory values confirmed that all patients had discontinued AAS long enough for endogenous lab values to fall below normal reference ranges. All patients were muscular in nature with an average BMI less than 27 at presentation. Table 1 presents the patient characteristics, anabolic history, and side effects upon presentation of the 5 patients.

**LABORATORY STUDIES**

Initial blood screening consisted of: AST, ALT, GGT, TOTAL CHOLESTEROL, LH, FSH, TESTOSTERONE, GLUCOSE, PROLACTIN, PSA TOTAL, TSH, T3 UPTAKE, T4 TOTAL, T4 FREE, HEMOGLOBIN, HEMATOCRIT

Table 2 shows all baseline serum blood levels at presentation. Baseline blood screening excluded any form of hyperprolactinemia or hypothyroidism as causes of hypogonadism in most patients. After physician examination and history and physical evaluation, it was determined that a history of AAS usage was present and most likely the cause of the patients’ hypogonadotropic hypogonadal lab values; not hyperprolactinemia or hypothyroidism. Laboratory testing was performed by Quest Diagnostics Inc., (Houston, TX) and SmithKline Beecham Clinical
Laboratories, (Houston, TX). Repeat serum LH & testosterone samples were measured by immunoassay using chiron reagent kits on an ACS-180 instrument.

METHODS

A review of patients’ medical records showed a treatment intervention of (a) human chorionic gonadotropin (HCG) (Ferring Pharmaceuticals), (b) clomiphene citrate (Teva Pharmaceuticals), and (c) tamoxifen (AstraZeneca). Typical dosage of HCG consisted of 2500 units every other day for 16 days. All HCG injections were self-administered intramuscularly. Starting dosages of clomiphene citrate and tamoxifen were 50mg and 20 mg daily, respectively. Patients started all three medications simultaneously and reported for the first follow-up blood work after completion of HCG, 16 days later. The post HCG blood analysis assessed testosterone-total response only. If testicular stimulation, i.e. testosterone production, was inadequate, additional HCG was administered at this stage of therapy rather than waiting an additional 30-45 days before the protocol completion. If the testicular response to the HCG demonstrated sufficient testicular stimulation (typically a blood serum level of >300 ng/dL), clomiphene citrate and tamoxifen were continued for 15 and 30 days, respectively. The arbitrary cut-off level of 300 ng/dL was used as a general assessment where sufficient Leydig cell stimulation was taking place even in light of artificial stimulation from HCG. A repeat blood sample was then taken at day 45 to assess hypothalamic-pituitary-gonadal axis status via luteinizing hormone and total testosterone levels. Because of the varying cessation times of the medications, the concluding blood sample was taken after a 30 and 15-day washout period of HCG and clomiphene citrate, respectively. For HPGA function to be considered normal, both LH and testosterone values had to fall within the normal reference ranges. For the purposes of patient treatment, if LH and testosterone values were still below normal limits at the conclusion of 45 days of treatment, a repeat protocol administration of HCG, clomiphene citrate, and tamoxifen was given. This protocol was repeated with every patient until LH and testosterone values reached normal ranges.

RESULTS

All five patients were considered eugonadal by normal laboratory reference ranges by the conclusion of treatment. Average serum total testosterone rose from 98.2 to 692.8 ng/dL. Average serum LH rose from <1.0 to 7.92 mIU/mL. An average of 48,974 U of HCG (five 10,000 Unit boxes), 3412.5 mg of clomiphene citrate (68.25 50mg tablets), and 968.71 mg of tamoxifen (48.44 20mg tablets) were used to treat all patients to eugonadal. Total treatment time ranged from 43-120 days. Mean elapsed time from initiation of treatment to eugonadal was 68.6 days. Statistical analysis was performed using repeated measures ANOVA. Pre and post treatment testosterone values were significantly (p<.001) different as were the LH values (p<.0008). Table 3 demonstrates the hormone changes during the treatment period and the duration to eugonadal.

ADVERSE EVENTS

None of the study subjects had any serious or treatment-terminating effects as a result of the multi-drug protocol. No problems were noted with regards to parameters of normal urologic function or treatment causing gynecomastia. Any side effects documented at presentation were
reversed by the conclusion of treatment.

DISCUSSION

This observational study demonstrates the possible efficacy of HCG, clomiphene citrate, and tamoxifen citrate in returning the HPGA to normal physiological function in adult males suffering from androgen induced hypogonadotropic hypogonadism. In the case of decreased testicular function manifested by low testosterone levels, it is of primary importance to first return the normal function of the testicular cells. The initial lack of response to HCG should not immediately be a cause for the initiation of testosterone replacement therapy, as with the current accepted therapy modality by many physicians. Blood analysis confirmed that no exogenous testosterone was administered during the treatment period, as exogenous androgens would have had a suppressive effect on endogenous gonadotropin production. Therefore, because of the corresponding normal gonadotropin and testosterone values, it is accepted that gonadotropin and testicular function were normal by the conclusion of treatment.

The standard treatment of HIV-related muscle wasting, AAS therapy, may involve decades of treatment and the attendant problems with any therapy of a prolonged nature. Polycythemia vera, elevated hepatic enzymes, and prolonged negative alterations in lipid profile are a few of the dangers experienced by HIV patients administered AAS for extended periods. Of greatest concern is the increasing numbers of individuals who are currently being treated with AAS to increase muscle mass either for medicinal or recreational means without attention being given to periodically returning the HPGA to normal. With roughly 4 million men in the U.S. being considered hypogonadal (Lacayo R., 2000; Sheffield-Moore et al, 1999; Shelton DL, 2000), an estimated 200,000 men are currently receiving testosterone treatment for the condition (Shelton DL, 2000). As stated earlier, AAS are being prescribed to HIV & AIDS sufferers to combat progressive muscle loss. The Centers for Disease Control and Prevention (CDC) reported an estimated 635,000+ men diagnosed with AIDS through December 2000 while an estimated 97,700 have been reported with HIV (Centers for Disease Control, vol.12, No. 2, table 5; Centers for Disease Control, vol. 12, No. 2, table 6). In 2000 alone over 31,000 men were diagnosed with the AIDS virus (Centers for Disease Control, vol. 12, No. 2, figure 3). Between hypogonadal, AIDS, & HIV males, potentially over 900,000 men are being administered AAS therapy.

Studies recently published on patients suffering from various tissue-depleting conditions and HIV affliction (Bhasin et al, 2000; Grinspoon et al, 1998; 1999; 2000; Rabkin et al, 1999; 2000; Sattler et al, 1999; Strawford et al, 1999;1999; Van Loan et al, 1999) have not identified what should be done to restore normal endocrine status post-treatment. Considering the dosages and compounds administered in many studies, there is no question that subjects were left hypogonadal after therapy. In the cases where the periodic use of testosterone or AAS are necessary, intervention to return the HPGA to normal should be initiated as soon as possible after the cessation of the AAS. As described herein, a possible treatment modality may be the combined regimen of HCG, clomiphene citrate, and tamoxifen.

Medical history has demonstrated examples of physician-induced complications resulting from treatment. Iatrogenic hyperthyroidism (Bartsch & Scheiber, 1981) and iatrogenic Cushing’s syndrome (Cihak & Beary, 1977; Kimmerle & Rolla, 1985; Smidt & Johnston, 1975; Tuel et al,
1990) are cases were administered medications or treatments provoked abnormalities in patients’ normal physiology. The administration of testosterone as a treatment for hypogonadotrophic hypogonadism falls into this same category of causing endocrine related abnormalities (Bhasin et al, 1996; Marynick et al, 1979; Strawford et al, 1999; Tenover, 1992). Testosterone replacement therapy has proven to be very effective in reversing the symptoms of suppressed testosterone production, but does not treat the underlying cause of the deficiency. Positive effects of testosterone treatment; i.e. improved sex drive, improved sense of well-being, lean body mass; are all transient in light of plummeting gonadotropin levels. Upon cessation of testosterone treatment patients can expect a complete reversal of positive benefits as exogenously influenced testosterone levels metabolize and decline rapidly. Further controlled studies need to be performed showing the combined effects of HCG, clomiphene citrate, and tamoxifen in returning HPGA functioning to normal. Long-term follow-up on these patients returning to normal will be necessary to ensure permanent reversal of hypogonadotropic hypogonadal conditions. In addition, studies documenting dose-response curves for pituitary inhibition and reversal due to AAS administration are critical in determining the correct dose, duration, and form of treatment that is optimal without causing permanent damage. When the need for long-term androgen use presents, using moderately supraphysiologic doses of androgens as suggested by Strawford and colleagues (1999) coupled with post-treatment HPGA restoration as demonstrated here, may be a more effective means over high-dose protocols used to offset negative alterations in lean body mass. Unfortunately current studies have yet to adequately address a standard of patient care post-androgen therapy. Because of the negative impact of the hypogonadal state on physical and mental well-being, pharmacotherapy that restores HPGA function more rapidly than current modalities would greatly benefit men with hypogonadotropic hypogonadism.

While we believe that the treatment protocol was effective in returning normal hormonal function to these men, the lack of randomization or a control group leaves room for speculation. Although cases of spontaneous return to eugonadism with no medicinal intervention have been published, these reports documented durations anywhere from 6-18 months before normal hormone status was achieved (Gazvani et al, 1997; Wu et al, 1996). If the alternative treatment modality described herein can reverse suppressed gonadotropin production and AAS associated side effects much sooner than non-treatment, further evaluation of this therapy should continue.

**HPGA Normalization Protocol After Androgen Treatment**

_N Vergel, AL Hodge, MC Scally_

Program for Wellness Restoration, _PoWeR_

**Objective Results Discussion**

To develop an approach to cycle androgens that would result in significant changes in body composition and accelerate the normalization of the hypothalamic pituitary gonadal axis (HPGA) after cessation of androgens.

**Methods**

An uncontrolled study of 19 HIV-negative eugonadal men, ages 23 – 57 years, administered testosterone cypionate and nandrolone decanoate for 12 weeks, and then were treated
after the cessation of the AAS. As described herein, a possible treatment modality may be the combined regimen of HCG, clomiphene citrate, and tamoxifen. Medical history has demonstrated examples of physician-induced complications resulting from treatment. Iatrogenic hyperthyroidism (Bartsch & Scheiber, 1981) and iatrogenic Cushing’s syndrome (Chichak & Beary, 1977; Kimerle & Rolla, 1985; Smidt & Johnston, 1975; Tuel et al, 1990) are cases were administered medications or treatments provoked abnormalities in patients’ normal physiology. The administration of testosterone as a treatment for hypogonadotropic hypogonadism falls into this same category of causing endocrine related abnormalities (Bhasin et al, 1996; Marynick et al, 1979; Strawford et al, 1999; Tenover, 1992). Testosterone replacement therapy has proven to be very effective in reversing the symptoms of suppressed testosterone production, but does not treat the underlying cause of the deficiency. Positive effects of testosterone treatment; i.e. improved sex drive, improved sense of well-being, lean body mass; are all transient in light of plummeting gonadotropin levels. Upon cessation of testosterone treatment patients can expect a complete reversal of positive benefits as exogenously influenced testosterone levels metabolize and decline rapidly. Further controlled studies need to be performed showing the combined effects of HCG, clomiphene citrate, and tamoxifen in returning HPGA functioning to normal. Long-term follow-up on these patients returning to normal will be necessary to ensure permanent reversal of hypogonadotropic hypogonadal conditions. In addition, studies documenting dose-response curves for pituitary inhibition and reversal due to AAS administration are critical in determining the correct dose, duration, and form of treatment that is optimal without causing permanent damage. When the need for long-term androgen use presents, using moderately supraphysiologic doses of androgens as suggested by Strawford and colleagues (1999) coupled with post-treatment HPGA restoration as demonstrated here, may be a more effective means over high-dose protocols used to offset negative alterations in lean body mass. Unfortunately current studies have yet to adequately address a standard of patient care post-androgen therapy. Because of the negative impact of the hypogonadal state on physical and mental well-being, pharmacotherapy that restores HPGA function more rapidly than current modalities would greatly benefit men with hypogonadotropic hypogonadism. While we believe that the treatment protocol was effective in returning normal hormonal function to these men, the lack of randomization or a control group leaves room for speculation. Although cases of spontaneous return to eugonadism with no medicinal intervention have been published, these reports documented durations anywhere from 6-18 months before normal hormone status was achieved (Gazvani et al, 1997; Wu et al, 1996). If the alternative treatment modality described herein can reverse suppressed gonadotropin production and AAS associated side effects much sooner than non-treatment, further evaluation of this therapy should continue.

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> ABBREVIATIONS

AAS Anabolic-Androgenic Steroids
AIDS Acquired Immunodeficiency Virus
ALT Alanine aminotransferase
AST Aspartate aminotransferase
BMI Body Mass Index
dl deciliter
FSH Follicle Stimulating Hormone
GGT Gamma-glutamyl transferase
GnRH Gonadotropin Releasing Hormone
HCG Human Chorionic Gonadotropin
HIV Human Immunodeficiency Virus
HPGA Hypothalamic Pituitary Gonadal Axis
kg kilogram
LH Luteinizing Hormone
mg milligram
mIU milli International Units
mL milliliter
ng nanogram
PSA Prostate Specific Antigen
T3 Triiodothyronine
T4 Thyroxine
TSH Thyroid Stimulating Hormone