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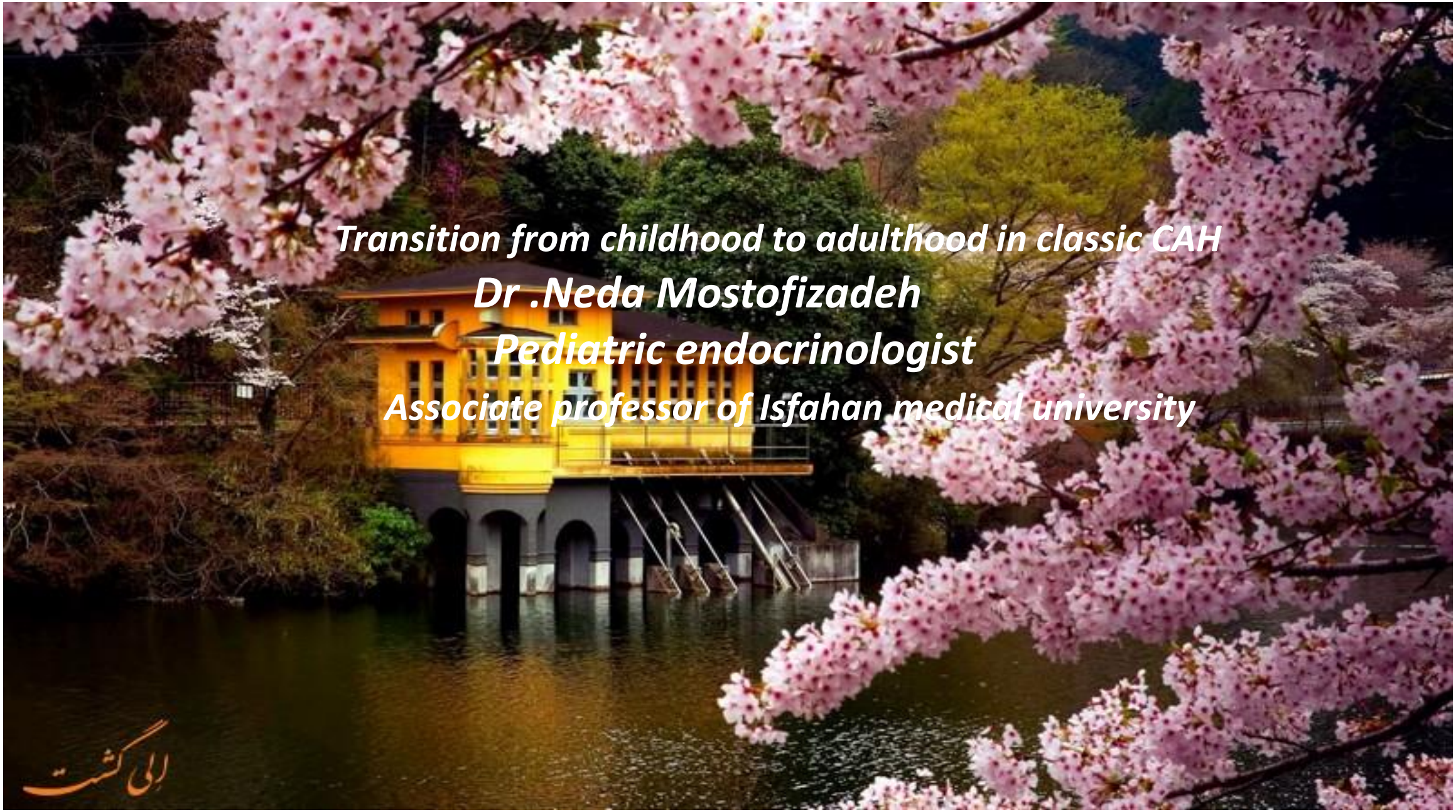
Transition from childhood to adulthood in classic CAH
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Establishing care

- *Management of adrenal insufficiency .*
- *Adrenal hyperandrogenism*
- *Sexual function*
- *Fertility*
- *complications (abdominal obesity, adrenal rest tumors, bone loss, and impaired quality of life).*

Initial evaluation

- ***Glucocorticoid and mineralocorticoid treatment regimens** :*
- ***All** adults with classic 21OHD require continued glucocorticoid therapy, and **most** require continued mineralocorticoid therapy.*
- *In adults, a **distinction** between "salt wasting" versus "simple virilizing" subtypes of classic 21OHD should be avoided.*
- *This distinction is important for newborns with classic 21OHD, but it is irrelevant for adults and often encourages withholding of vital therapies.*
- *Adrenal hormone dosing requirements often change with age and cessation of growth.*

Glucocorticoid and mineralocorticoid treatment

- ***Glucocorticoid therapy** :*
- *Hydrocortisone therapy often achieves the goals and imparts lower risk of long-term complications than longer-acting glucocorticoids.*
- ***Mineralocorticoid therapy** :*
- *In virtually all adults with classic 21OHD, continued fludrocortisone therapy is needed to treat mineralocorticoid deficiency.*
- *Dosing requirements are usually lower than in children or adolescents*.

cont

- ***Genetic counseling*** *:Genetic counseling should be provided during adolescence and again during the transition to adult care to help inform decisions about fertility .*

- ***Testicular ultrasound (males)*** *: For males, a testicular ultrasound should be performed to evaluate for testicular adrenal rest tumors (TARTs) .*

- *Thereafter, the frequency of monitoring depends in part on laboratory and physical examination findings.*

Menstrual history and gynecologic examination (females) :

- *For females, a genitourinary examination should be performed (if not performed previously during adolescence), and menstrual history should be assessed.*

- *In postpubertal females with classic 21OHD, central goals of management are to optimize menstrual and sexual function.*

Selection of regimen

- *For adults we suggest the short-acting glucocorticoid hydrocortisone.*
- *hydrocortisone is the least associated with cushingoid complications.*
- *It is less convenient than other synthetic glucocorticoids as it is typically administered in multiple daily doses.*
- *Adolescents may be switched from hydrocortisone to longer-acting glucocorticoid regimens to promote treatment adherence.*
- *When such individuals establish adult care, we encourage transition back to hydrocortisone therapy.*

Selection of regimen

- ***If an individual anticipates substantial difficulty adhering to three times daily hydrocortisone, or if adherence proves difficult after a trial of hydrocortisone therapy, we substitute a longer-acting glucocorticoid typically prednisolone or methylprednisolone.***

Selection of regimen

- *In adults with severe 21OHD who do not achieve adequate adrenal androgen suppression on hydrocortisone alone, we add a small dose of a longer-acting glucocorticoid at bedtime to suppress overnight ACTH secretion.*

Hydrocortisone (preferred glucocorticoid)

- ***Transition to adult dosing*** *: For adults with classic 21OHD, a typical regimen is hydrocortisone 15 to 30 mg daily administered in three divided doses; alternatively, a calculated daily dose of 10 to 15 mg/m2 body surface area can be used.*

- *The largest dose is taken in the morning upon waking, and progressively smaller doses are taken late morning/midday and late afternoon/early evening (eg, 15, 5, and 2.5 mg).*

Hydrocortisone (preferred glucocorticoid)

- *In contrast to other types of adrenal insufficiency, adequate treatment of classic 21OHD usually requires three rather than two daily doses of hydrocortisone.*
- *The third dose helps to limit the overnight rise in ACTH secretion that promotes adrenal androgen production.*
- *Hydrocortisone administration three times daily usually provides sufficient suppression of adrenal steroids without imparting clinical manifestations of glucocorticoid excess.*

Monitoring and dose adjustment

- *During the transition to adult care or titration of hydrocortisone therapy, adults with classic 21OHD should be evaluated every three to four months through both clinical and laboratory assessments.*

- *Adults who are on stable hydrocortisone regimens should be seen at least once annually.*

- *The goal of dose titration is to achieve the lowest hydrocortisone dose that provides both adequate cortisol replacement and sufficient suppression of adrenal steroid production.*

Clinical assessment

- *Signs and symptoms that suggest undertreatment include weight loss, nausea, weakness, hypotension, and fatigue.*
- *Signs and symptoms of overtreatment include body weight gain, impaired sleep, increased blood pressure, and edema.*
- *Signs of chronic overtreatment include dermal atrophy, bruising, proximal muscle weakness, purple striae, and other manifestations of Cushing syndrome.*
- *females should be assessed for signs of androgen excess .*

Clinical assessment

- *In males, testicular atrophy on examination suggests chronic gonadotropin suppression from adrenal-derived androgens and thus indicates glucocorticoid undertreatment.*

Laboratory assessment

Routine laboratory measurements (ideally in the morning) should include serum concentrations of the following :

- *Androstenedione (generally considered the most relevant biomarker)*
- *Testosterone*
- *SHBG(used to calculate free and bioavailable testosterone)*
- *FSH and LH routinely in males and in females with oligo- or amenorrhea.*

Laboratory assessment

- *Serum 17 OHP also may be useful for detecting glucocorticoid overtreatment, but routine measurement is not essential for treatment monitoring in adults.*

- *In undertreated males and all females with classic 21OHD, the most abundant circulating androgen is not testosterone but rather 11-ketotestosterone, which is only measured in certain reference and research laboratories .*

Laboratory assessment

- **Males** : *In males, glucocorticoid therapy should be titrated to maintain testicular function, which generally requires the serum androstenedione level at or near the upper limit of the normal range.*
- *Normalization of 17-oh p indicates glucocorticoid overtreatment.*
- *FSH and LH levels should be maintained in the normal range.*
- *suppressed gonadotropins and/or an androstenedione/testosterone ratio >1 indicate inadequate suppression of adrenal androgen production.*
- *Such findings should prompt both consideration of glucocorticoid therapy intensification and additional evaluation for TARTs with testicular examination and/or ultrasound.*

Laboratory assessment

- ***Females*** *:The goals(normal menstrual function and unwanted hair) are usually achieved with androgen (androstenedione, testosterone) levels at or near the upper limit of the normal range.*

- *Testosterone and androstenedione should* ***not*** *be suppressed below the normal range, as this indicates overt glucocorticoid overtreatment.*

- *A normalized 17-OHp level similarly indicates glucocorticoid overtreatment.*

Dose adjustment

- *We adjust the hydrocortisone dose in 2.5 to 5 mg increments as needed based on evidence of over- or undertreatment.*
- *If signs or symptoms of cortisol deficiency are evident throughout the day, we preferentially increase the morning dose.*
- *If additional suppression of adrenal androgens is needed, we empirically increase the doses throughout the day; increasing more than one dose is often necessary for sufficient adrenal steroid suppression.*

Dose adjustment

- *Impaired sleep may limit the acceptability of increased evening doses of hydrocortisone. If impaired sleep or other evidence of glucocorticoid excess develops, or if adequate adrenal androgen suppression cannot be achieved on a total daily hydrocortisone dose ≤30 to 35 mg, combination therapy may be helpful.*

- *In females with persistent signs of hyperandrogenism despite normalized serum androgen levels, we pursue alternative treatment strategies.*

Alternative regimens

- ***Combined regimens for persistent hyperandrogenism*** *: In adults in whom adequate adrenal steroid suppression cannot be achieved with standard hydrocortisone regimens, we suggest combination therapy .*

- *Combination therapy entails typical cortisol replacement doses of hydrocortisone (10 to 25 mg daily) during the day and a very small dose of a longer-acting glucocorticoid at bedtime (1 to 2 mg prednisolone or methylprednisolone) .*

- *The lowest dose of the longer-acting glucocorticoid that achieves adequate adrenal steroid suppression should be used.*

- *Combination therapy is very effective for suppressing ACTH and minimizes total glucocorticoid exposure.*

Alternative regimens

Combined regimens may be particularly helpful in the following settings:

- *In females pursuing pregnancy who need additional suppression of serum progesterone.*
- *In males in whom reducing TART size is a goal of care.*

Longer-acting glucocorticoids for adherence challenges

- ***Prednisolone and methylprednisolone(preferred longer-acting agents)*** *: For individuals with classic 21OHD who have difficulty adhering to three daily doses of hydrocortisone prednisolone or methylprednisolone prefered.*
- *These longer-acting glucocorticoids are typically administered twice a day with a larger dose in the morning to replace the cortisol deficiency (eg, 3 to 5 mg) and a small dose at bedtime to attenuate the pre-dawn ACTH rise (eg, 1 to 2.5 mg) .*
- *We avoid "inverse diurnal rhythm" dosing, in which the larger dose is given at bedtime (eg, methylprednisolone 2 mg upon waking and 5 mg at bedtime); such regimens do not replace the cortisol deficiency well and overtreat during the night.*
- *Prednisolone is available as a 1 mg/mL liquid, which facilitates titration of doses <2 mg.*

- ***Prednisone*** *: Prednisone is a pro-drug that requires hepatic conversion to prednisolone to be biologically active, and interindividual variability in prednisolone exposure.*

- *Prednisone is therefore not generally preferred but is effective in some patients.*

Dexamethasone

- *In adults with classic 21OHD, dexamethasone is not preferred. It has a narrow therapeutic index, and oral dose forms are designed for anti-inflammatory uses rather than cortisol replacement, which makes dose titration challenging.*

- *Dexamethasone is a very potent and long-acting glucocorticoid and effectively suppresses ACTH secretion. However, its long duration of action and variable interindividual metabolism may increase risk of cushingoid features with chronic use .*

- *Further, bedtime administration of 0.25 to 1 mg does not replace the cortisol deficiency well, although this is the most effective regimen for ACTH suppression.*

- *Dexamethasone therapy should be of limited duration and reserved for specific treatment goals (eg, TART shrinkage).*

Modified-release hydrocortisone

- ***Modified-release hydrocortisone (limited availability)** :*
- *Is authorized for use in adolescents and adults with CAH in regions including the United Kingdom and Europe.*
- *In individuals with inadequate adrenal androgen suppression on immediate-release hydrocortisone, modified-release hydrocortisone is a reasonable alternative .*
- *The total daily dose is 15 to 25 mg, administered in two divided doses. The first dose is taken upon waking, and the second dose is taken at night immediately prior to sleep.*
- *The bedtime dose should be approximately 65 to 75 percent of the total daily dose, with the remainder taken in the morning.*

Glucocorticoid dose adjustment during acute stress :

- *Increased glucocorticoid doses are usually **not** needed during routine psychological stress or exercise .*

Crinecerfont

- ***Comorbidities due to glucocorticoid therapy*** *:*

- *For adults with classic 21OHD and significant comorbidities or risk of comorbidities due to the chronic, supraphysiologic glucocorticoid treatment required for disease management, we suggest adding crinecerfont as adjunctive therapy (if available).*

Crinecerfont

- ***Corticotropin-releasing factor (CRF) type 1 receptor antagonist (crinecerfont)*** *:*
- *Crinecerfont, an oral CRF type 1 receptor antagonist, has regulatory approval in the United States as adjunctive therapy for patients with classic 21OHD .*
- *For most adults, the initial dose is 100 mg orally twice daily with meals.*
- *Dose adjustments might be needed for patients taking medications that induce cytochrome P450 3A4 (table 3).*

Efficacy

- *Crinecerfont reduces ACTH production and thereby attenuates adrenal steroid production, potentially enabling disease management without supraphysiologic glucocorticoid dosing.*

- *In a 24-week trial in 182 adults with classic 21OHD taking supraphysiologic glucocorticoid therapy (mean dose equivalent to hydrocortisone 17.6 mg/m2body surface area daily), participants who were randomly assigned to treatment with crinecerfont 100 mg twice daily (n = 122) achieved a greater glucocorticoid dose reduction, while maintaining androstenedione at or below baseline values, compared with those assigned to placebo (n = 60; 27.3 versus 10.3 percent dose reduction, respectively).*

Efficacy

- *During down-titration of the glucocorticoid dose, the fludrocortisone dose and stress dosing instructions for glucocorticoid treatment might require adjustment.*

Adverse effects

- *In the trial described immediately above, fatigue and headache were the most common side effects.*

- *One participant in the crinecerfont group experienced adrenal crisis.*

Other treatments

- *Although a few cases have been reported of unilateral or bilateral adrenalectomy for severe 21OHD , we avoid this intervention.*
- *The major benefit of adrenalectomy is the immediate elimination of adrenal androgen and progesterone secretion, which allows treatment with lower glucocorticoid doses.*
- *The reduction in adrenal androgen production may be only temporary with unilateral adrenalectomy.*
- *Bilateral adrenalectomy heightens the dependency on glucocorticoid and mineralocorticoid replacement therapy and therefore increases risk of adrenal crisis.*
- *Adrenalectomy does not always prevent subsequent development of adrenal rest tumors, even in females .*

Androgen excess (females)

- *In females with 21OHD, additional intervention may be necessary to manage hyperandrogenic symptoms (eg, hirsutism, acne) or to regulate menstrual function.*
- *In such cases, combined OCP can be used in conjunction with glucocorticoid therapy.*
- *Combined oral contraceptives both regulate menstrual function and mediate antiandrogenic effects by raising SHBG.*
- *In females with hirsutism, mechanical or topical hair removal methods also can be used.*
- *we **avoid** spironolactone for antiandrogen treatment, as it antagonizes the effect of fludrocortisone and can cause volume depletion.*

Mineralocorticoid replacement

- *Although mineralocorticoid replacement needs usually decrease in adulthood, most adults with classic 21OHD require continued mineralocorticoid therapy with fludrocortisone .*

- *The reduced dose requirement reflects the increases in both dietary sodium intake and mineralocorticoid signaling that occur with progression from infancy to adulthood .*

Transition to adult fludrocortisone dose

- *In individuals transitioning from pediatric to adult care, the initial adult fludrocortisone dose is determined by the patient's current dose and assessment of seated and standing blood pressure, serum potassium concentration, and PRA or DRC.*

- *In adults with classic 21OHD, a typical fludrocortisone dose is 0.05 to 0.2 mg daily . This may be taken once daily or in two divided doses.*

Transition to adult fludrocortisone dose

- *Hydrocortisone has greater mineralocorticoid activity than other options for glucocorticoid therapy, so adults on hydrocortisone typically require lower fludrocortisone doses.*

- *For example, hydrocortisone 20 mg daily has mineralocorticoid activity approximately equivalent to 0.05 mg fludrocortisone.*

Monitoring and dose adjustment

- During the transition to adult care or titration of fludrocortisone therapy, adults with classic 21OHD should be evaluated every three to six months through both clinical and laboratory assessments, and then at least once annually .

- Optimal mineralocorticoid replacement may enable reduction of the glucocorticoid dose .

Fludrocortisone therapy

- **Clinical assessment:**

  Elevated blood pressure and dependent edema can be evidence of overtreatment.

Laboratory assessment

- *The goals of fludrocortisone treatment are to normalize the serum potassium level and achieve a PRA or DRC in the normal reference range.*

- *If PRA (or DRC) remains mildly elevated in adults who are asymptomatic with a normal k level, the fludrocortisone dose should **not** be further increased. in this setting, an increased dose can lead to hypokalemia.*

Fludrocortisone therapy

- *Laboratory assessment is essential and should be performed even in asymptomatic individuals.*
- *Undertreatment can lead to chronic volume depletion that may be clinically silent and evident only on laboratory testing.*
- *Irrespective of whether it causes symptoms, undertreatment results in persistent overproduction of renin and angiotensin II.*
- *Angiotensin II and volume depletion can stimulate ACTH secretion, leading to higher adrenal androgen synthesis .*

Dose adjustment

- *We adjust the fludrocortisone dose as needed in 0.05 to 0.1 mg increments based on evidence of under- or overtreatment.*
- *In adults, once a therapeutic replacement dose is established, the fludrocortisone dose generally remains stable for years.*
- *Further dose adjustments may be needed in the setting of increased salt losses (eg, exposure to warm climates, vigorous exercise) or the development of primary hypertension.*

Genetic counseling

- *Based on the incidence of classic 21OHD and an estimated carrier frequency of 2 percent of the population, an individual with classic 21OHD has an 1:120 probability of having a child with classic 21OHD .*

Normalize menstrual function

- *Glucocorticoid under- and overtreatment both can cause menstrual irregularity.*
- *If adequate adrenal steroid suppression cannot be achieved without excessive glucocorticoid exposure, combined oral contraceptives might help to regulate menstrual function.*
- *Adrenal-derived progesterone, rather than androgens, is the major cause of menstrual irregularity and infertility in females with classic 21OHD.*

Fertility

- ***Prior to attempted conception** :*

- *Females with classic 21OHD should have gynecologic consultation, ideally with a surgeon who can provide initial genital reconstruction surgery or modification of previous surgery if needed.*

Fertility

- *Once functional anatomy is achieved, glucocorticoid therapy is intensified to achieve greater suppression of adrenal steroid production.*
- *The goal of therapy is to achieve a follicular-phase progesterone <0.6 ng/mL (2 nmol/L).*
- *This intensified treatment is generally necessary for at least several months to achieve conception and often requires combination therapy with both immediate-release hydrocortisone and a longer-acting glucocorticoid administered at bedtime .*

Outcomes

- *In females with classic 21OHD, fertility rate is reduced .*
- *Initial studies found that only 25 percent of females with classic 21OHD and 10 percent of those with severe disease ever attempted to conceive .*
- *In contrast, a 2021 study from Sweden found that 40 to 42 percent of females with either nonclassic or "simple virilizing" classic 21OHD had borne children, similar to the general population (45 percent).*
- *<10 percent of females with severe "salt wasting" classic disease had biological children .*
- *In individuals who receive proper treatment and have regular intercourse, pregnancy rates exceed 90 percent.*

Outcomes

- *females with classic 21OHD have normal fertility potential , and the low desire to pursue parenthood appears restricted to those with the most severe disease.*
- *Factors that contribute to impaired fertility include:*
- *Increased adrenal-derived progesterone, which unfavorably changes cervical mucus and endometrial function, similar to progestin-only contraceptives*
- *Anovulation*
- *Vaginal stenosis, from intrauterine virilization and/or sequelae from prior genital reconstructive surgery*
- *Psychologic factors*
- *Ovarian hyperandrogenism secondary to chronic anovulation*
- *Ovarian adrenal rest tumors*

No role for screening for ovarian adrenal rest tumors

- *In females with classic 21OHD, we do **not** screen for adrenal rest tumors.*

- *Ovarian adrenal rest tumors appear uncommon in treated females with classic 21OH.D*

ovarian adrenal rest tumors

- *As in males with testicular adrenal rest tumors, the etiology of rest tumors in females appears related to sustained elevations in ACTH due to glucocorticoid undertreatment or nonadherence.*
- *In females, whether glucocorticoid therapy reduces adrenal rest development and/or size is unknown.*
- *In females, adrenal rests may develop in the retroperitoneum, including the ovaries and surrounding structures.*
- *They occur primarily in the ovarian tissue and, less often, in the paraovarian/adnexal area.*

Screening for testicular adrenal rest tumors

- **Males**
- *Adult males with classic 21OHD are at risk for testicular adrenal rest tumors (TARTs).*
- *The pathogenesis of TARTs is not known, but they are believed to derive either from ectopic adrenal cortex remnants in the testis or from reprogrammed Leydig stem cells, which differentiate and grow under the influence of chronically elevated ACTH.*
- *TART cells express genes that encode enzymes and markers characteristic of Leydig and adrenal cells .*
- *The mass effect of TARTs increases the intratesticular pressure, impairs blood flow to the normal testis, and hinders outflow of semen.*
- *TARTs can cause infertility through multiple mechanisms.*

Screening

- *In males with classic 21OHD, testicular ultrasound screening should begin in adolescence to detect TARTs, including once at the time of transition to adult care.*

Screening

- *Thereafter, we perform a physical examination and/or ultrasound monitoring for TARTs at least annually, with additional evaluation performed whenever a period of glucocorticoid undertreatment is detected through laboratory monitoring.*
- *On physical examination, TARTs are firm, irregular masses originating near the rete testes.*
- *They are typically bilateral and can be painful when large.*
- *Masses may be small and not palpable on physical examination.*
- *Ultrasonography is the most sensitive method of detection*
- *Multiple studies have shown that 30 to 50 percent of adolescent and adult males with classic 21OHD develop TARTs*

Management

- *The best approach to prevent TARTs is to provide adequate glucocorticoid therapy and to avoid long lapses in treatment .*
- *Intensified glucocorticoid treatment is sometimes, but not always, effective for decreasing the size of TARTs, relieving pain, and restoring fertility.*
- *Case reports have noted either a decrease in size or even disappearance of testicular masses with a course of supraphysiologic doses of dexamethasone or with daytime hydrocortisone plus bedtime dexamethasone.*

Management

- *TART shrinkage typically requires several months of treatment intensification, but side effects from these dexamethasone-containing regimens limits the duration of use.*

- *Surgical removal provides good long-term control of TART growth and pain, but as demonstrated in a series of eight patients with TART , surgery is unlikely to restore testicular testosterone and sperm production.*

- *In the presence of TART(s), an elevated follicle-stimulating hormone (FSH) level indicates testicular injury and is a poor prognostic factor for fertility .*

Fertility

- *In males with classic 21OHD, a normal semen analysis is the best evidence of good disease control without overtreatment, and sperm banking is an option for young males who want to preserve their fertility.*
- *In males, both elevated and suppressed FSH levels can indicate impaired fertility.*
- *In males with TARTs, an elevated FSH level suggests testicular injury.*
- *A suppressed FSH (and LH) level reflects inadequate glucocorticoid treatment and leads to impaired spermatogenesis.*
- *In some males with gonadotropin suppression, glucocorticoid treatment intensification can restore sperm production.*
- *Gonadotropin replacement also has been used treat infertility due to classic 21OHD and hypogonadotropic azoospermia.*

Fertility

- *Undertreatment leads to impaired sperm production for two reasons.*
- *First, adrenal-derived androgens suppress gonadotropins, leading to reduced testosterone production from Leydig cells.*
- *High adrenal-derived androgens compensate for hypogonadism and maintain male secondary sexual characteristics, creating the false impression that testicular function is normal.*

Pregnancy

- ***Glucocorticoid and mineralocorticoid therapy*** *:Females with classic 21OHD require continued glucocorticoid and mineralocorticoid therapy throughout pregnancy.*
- *Hydrocortisoneand/or prednisolone should be used in combination with fludrocortisone.*
- *Pregnant females should* ***not*** *receive glucocorticoids that cross the placenta (eg, dexamethasone).*
- *During the first and second trimesters, the preconception glucocorticoid regimen is typically continued if it is well tolerated.*
- *If concerning glucocorticoid-related side effects occur, the dose can be moderated, primarily by reducing or eliminating the bedtime dose.*
- *During the third trimester, most patients require an increase in the glucocorticoid dose (20 to 40 percent) .*

- *The fludrocortisone dose does not usually require adjustment during pregnancy, but clinical and laboratory monitoring are needed.*

Delivery

- *Cesarean section is almost always required at delivery due to vaginal inadequacy.*

Outcomes

- *Full-term pregnancies can be achieved with delivery of healthy infants who have normal growth and development .*
- *Female infants without classic 21OHD are born with typical external genitalia; even when maternal androgen production is not normalized during pregnancy.*
- *Placental aromatase activity protects the fetal genitalia and brain from excess androgen exposure .*

MONITORING FOR LONG-TERM COMPLICATIONS

- *These individuals have increased risk of low bone density, obesity, insulin resistance, and hypertension.*
- *They also frequently report reduced quality of life.*
- *In addition to routine clinical and laboratory assessments for adrenal hormone therapy, we monitor the following:*

MONITORING FOR LONG-TERM COMPLICATIONS

- **Cardiometabolic risk factors** :
- *Body weight and BP should be measured at least annually.*
- *Additional cardiometabolic screening (FBS, HbA1c,lipid panel) should be performed as for the general adult population .*

MONITORING FOR LONG-TERM COMPLICATIONS

- *One study found that females with classic 21OHD have an increased risk of gestational diabetes mellitus.*

Bone mineral density measurement by dual x-ray absorptiometry

- *We perform baseline BMD in all patients at age 25 years, the approximate expected age of peak bone accrual .*

- *Since lifelong glucocorticoid therapy can impact bone accrual, this baseline measurement is important for interpreting subsequent BMD values.*

BMD

- *After baseline assessment, we remeasure BMD in adults who have had a prolonged (≥12 months) exposure to high-dose glucocorticoid treatment (>10 mg/day prednisone or equivalent), who develop other risk factors for bone loss, or who experience an atraumatic fracture.*

- *In the absence of these triggers for interim measurement, we remeasure BMD according to age- and sex-based guidelines for the general adult population.*

- *All adults with classic 21OHD should follow lifestyle measures for optimizing bone health, including adequate intake of calcium and vitamin D and routine physical activity.*

- *Vitamin D deficiency is common in children and adults with classic 21OHD.*

osteoporosis

- *In adults with classic 21OHD who are diagnosed with osteoporosis based on BMD measurement or atraumatic fracture, management is the same as for other populations with glucocorticoid-induced osteoporosis.*


Have a wonderful spring