On August 3, 1987, a 45-year-old, 74-kg Hispanic man was admitted to the urology service with sudden onset of sharp right flank pain radiating to his right testicle, gross hematuria for two days, and an inability to void on the morning of admission. Past medical history was significant for AIDS and toxoplasmosis of the central nervous system. He was receiving sulfadiazine, 1.5 g (80 mg/kg) orally every six hours, and pyrimethamine, 25 mg orally every day, for six weeks prior to admission and had been compliant. He had not been advised to increase fluid intake despite the warm weather. Other medications included daily folinic acid 5 mg and clotrimazole 10 mg five times daily. Physical examination only revealed a moderately distended abdomen with involuntary guarding, decreased bowel sounds, and right lower quadrant and flank pain. Urinalysis showed cloudy urine with a pH of 6.0, three to five white blood cells per high-power field, gross hematuria, and many crystals identified under light microscopy by one of us (K.C.) and described as "shocks of wheat" (diagnostic of acetylsulfadiazine) by the Director of the Clinical Pathology Laboratory. Laboratory values included a serum creatinine of 3.9 mg/dl (baseline 1.2 mg/dl), and serum albumin of 4.2 mg/dl. An abdominal radiograph revealed multiple stones in the upper right ureter. Sulfadiazine was discontinued and fluid and oral sodium bicarbonate therapy started. On Day 3 of hospitalization, a ureteral stint was placed to relieve the obstruction. Acetylsulfadiazine crystals were again identified. Serum creatinine level returned to baseline on Day 6, and he was discharged receiving clindamycin and pyrimethamine after eight days of hospitalization.

Animal models and clinical reports have demonstrated that sulfonamides of low solubility such as sulfadiazine may form concretions in the urinary tract under appropriate conditions, such as dehydration [2]. Increasing fluid intake to 1 to 2 liters per day is recommended [3]. Because of this practice and the use of more soluble agents, sulfonamide-induced crystalluria is now uncommon. Sulfonamide-induced crystalluria has also been reported in two patients with hypoalbuminia [4]. Since many patients with AIDS are prone to hypoalbuminia [5], this may be an additional predisposing factor in this population. Physicians and pharmacists should continue to advise patients to increase their fluid intake when taking sulfonamides.

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1. Armstrong D, Gold JWM, Dryjanski J, et al: Treatment of infections in patients with the acquired immunodeficiency syndrome. Ann Intern Med 1985; 103: 738–743.
2. Fox CL, Jensen OJ, Mudge GH: The prevention of renal obstruction during sulfadiazine therapy. JAMA 1943; 121: 1147–1150.
3. Barnes RW, Kawaichi GK: Factors affecting the formation of sulfonamide urinary concretions. J Urol 1943; 49: 324–333.
4. Buchanan N: Sulphamethoxazole, hypoalbuminaemia, crystalluria and renal failure. Br Med J 1978; 2: 172.
5. Kotler DP, Gaetz HP, Lange M, et al: Enteropathy associated with the acquired immunodeficiency syndrome. Ann Intern Med 1984; 101: 421–428.
6. Goadsby PJ, Donaghy AJ, Lloyd AR, Wakefield D: Acquired immunodeficiency syndrome (AIDS) and sulfadiazine-associated acute renal failure. Ann Intern Med 1987; 107: 783–784.

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## TURNER'S SYNDROME WITH ANOREXIA NERVOSA

**To the Editor:**

The rare concurrence of Turner's syndrome and anorexia nervosa has some interesting aspects from the standpoints of psychiatrics and endocrinology, and about 10 such cases have been reported [1,2]. This rare association may provide evidence to clarify changes in gonadotropin responsiveness in the concurrence of hypergonadotropic and hypogonadotropic hypogonadism. Some authors reported estrogen decreased food intake [3]. Onset of anorexia nervosa in a hypogonadal patient receiving estrogen may provide further evidence of a pathogenetic role of the hormone in anorexia nervosa.

Our Japanese patient was diagnosed as having Turner's syndrome with a karyotype of 45XO at the age of six years. She was 103.2 cm tall (mean ± SD, 114.7 ± 4.9 cm) and had some stigmata of the syndrome, that is, low hairline, a shield-like chest, and cubitus valgus. Administration of stanozolol 1 mg a day was started, and ethinyl estradiol methyl ether (0.0025 mg per day; 80 ng/kg per day) was added when she was 13 years and one month old for the purpose of promoting linear growth [4]. Her height was 129.8 cm and weight was 33 kg at the age of 13 years and six months. Breast development was in the prepubertal stage. After that, food intake decreased remarkably and she lost 8.4 kg (25.5 percent of her weight) during seven months. Her serum estradiol level was 9 pg/ml. She was diagnosed as having anorexia nervosa according to the criteria proposed by Feighner et al [5]. Gonadotropin-releasing hormone (Gn-RH) (70 $\mu g/m^2$ body surface) was injected intramuscularly at the age of six years and at 14 years of age, when she was anorectic. The estrogen therapy was discontinued five months before the second examination (**Table I**). Responses of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) in the anorectc period were suppressed below the prepubertal ranges. Thyrotropin (TSH) and prolactin responses to thyrotropin-releasing hormone (TRH) were within normal limits.

It has not been completely documented yet whether elevated gonadotropin levels in primary gonadal failure remained in the supranormal ranges, were within normal limits, or suppressed below normal limits when the patient had accompanying hypogonadotropic hypogonadism. Turner's syndrome with anorexia nervosa is considered to be a good experiment in nature to study such a condition. Dissociated responses of the two gonadotropins were reported [2]. The peak LH level after Gn-RH stimulation was beneath the prepubertal control value, but the serum FSH level in the patient rose higher than in normal subjects. In the present study, hypergonadotropinemia in gonadal dysgenesis, either LH or FSH, was demonstrated for the first

**TABLE I Responses of Luteinizing Hormone (LH) and Follicle-Stimulating Hormone (FSH) to Gonadotropin-Releasing Hormone**

| | 6 Years | | 14 Years | | Control* | |
|---|---|---|---|---|---|---|
| | Basal | Peak | Basal | Peak | Basal | Peak |
| LH (mIU/ml) | 8.0 | 43.0 | 5.3 | 10.4 | 5.0 ± 2.9 | 19.0 ± 6.0 |
| FSH (mIU/ml) | 29.5 | 165 | 2.4 | 7.8 | 4.7 ± 1.5 | 21.2 ± 7.0 |

* Normal values in prepubertal females.

time to be suppressed to the hypogonadotropic range in the anorectic condition.

Fear of obesity may be one of the causes of anorexia. Treatment with estrogen has also been considered to contribute to the onset of anorexia nervosa in these cases [1]. Ovariectomy is reported to increase appetite and is known to decrease the opioid peptide dynorphin [3], which has some influences on eating behavior. A case of anorexia nervosa with Turner's syndrome treated with low doses of estrogen has never been reported previously.

The present case demonstrates that elevated gonadotropin levels of the patient with gonadal dysgenesis were reduced below the normal limits in prepubertal girls during the anorectic period and also suggests the possibility that very small doses of estrogen may contribute to the onset of anorexia nervosa associated with Turner's syndrome.

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1. Dougherty GG Jr, Rockwell WJK, Sutton G, Ellinwood EH Jr: Anorexia nervosa in treated gonadal dysgenesis: case report and review. J Clin Psychiatry 1983; 44: 219–221.
2. Kauli R, Gurewitz R, Galazer A, et al: Effect of anorexia nervosa on gonadotropin secretion in a patient with gonadal dysgenesis. Acta Endocrinol 1982; 100: 363–368.
3. Morley JE, Levine AS, Grace M, Kneip J, Gosnell BA: The effect of ovariectomy, estradiol and progesterone on opioid modulation of feeding. Physiol Behav 1984; 33: 237–241.
4. Cuttler L, Van Vliet G, Conte FA, Kaplan SL, Grumbach MM: Somatomedin-C levels in children and adolescents with gonadal dysgenesis: differences from age-matched normal females and effect of chronic estrogen replacement therapy. J Clin Endocrinol Metab 1985; 60: 1087–1092.
5. Feighner JP, Robins E, Guze SB, et al: Diagnostic criteria for use in psychiatric research. Arch Gen Psychiatry 1972; 26: 57–63.

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## PNEUMOCOCCAL LUNG ABSCESS

**To the Editor:**

Although abscess formation has been reported in association with pneumococcal pneumonia, controversy remains over the role played by superinfection with other pathogens in causing the lung necrosis [1,2]. We report herein an unusual case of a community-acquired bacteremic pneumococcal lung abscess.

A 61-year-old diabetic woman, with a 110 pack-year smoking history, was admitted with cough productive of green odorless sputum, right subscapular pain, and chills. Her temperature was 98.2°F. Chest examination disclosed dullness to percussion and rales over the right posterior mid-lung field. The chest radiograph (**Figure 1**) revealed a large abscess with an air-fluid level in the posterior segment of the right upper lobe as well as bulging of the major fissure. The leukocyte count was 25,700/mm$^3$ with 77 percent neutrophils, 10 percent band forms, 2 percent metamyelocytes, 7 percent lymphocytes, and 4 percent monocytes. Gram-positive diplococci and neutrophils were seen on Gram stain of the sputum. Results of blood cultures were positive for Streptococcus pneumoniae. There was a normal reaction to skin testing with Candida antigen and none to tuberculin.

Initial therapy, consisting of intravenous erythromycin 2 g per day, resulted in clinical improvement and a reduction in leukocyte count. However, the chest radiograph continued to show a large abscess with a better-defined air-fluid level (**Figure 2**). Clindamycin was added for three days and then discontinued. Because a cavitary lung cancer was suspected, bronchoscopy and transbronchial biopsies were performed on the 10th day.

Inflammatory endobronchial changes were seen in the posterior segment of the right upper lobe. Biopsy specimens of the abscess wall revealed an organizing pneumonia and medial hypertrophy of pulmonary arteries. No infectious organisms were identified by tissue Gram stain of the lung specimen or by acid-fast stains of bronchial washings. Erythromycin was discontinued on the 11th day and the patient was discharged. A chest radiograph two months later revealed almost complete clearing. Treatment with isoniazid and rifampin was subsequently begun, when it

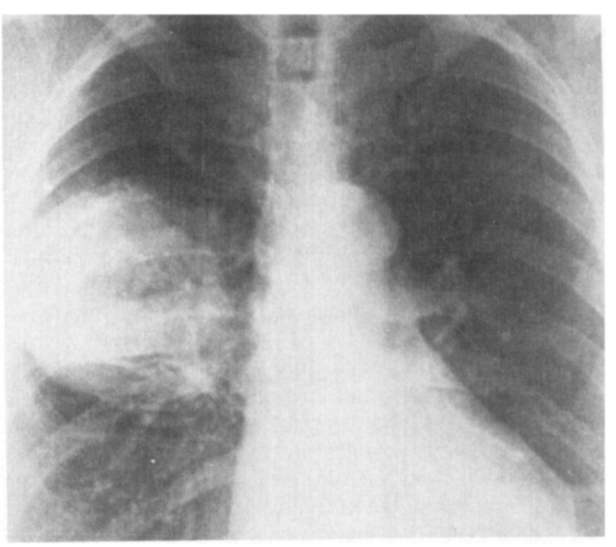

***Figure 1.*** *Chest radiograph shows large abscess in posterior segment of right upper lobe.*