

# The Endocrine and Metabolic Effects of Alcoholism

Shahid Athar

## Abstract

This review outlines the endocrine and metabolic effects of acute and chronic alcohol abuse. Effects of alcohol on endocrine organs can be a direct toxic effect or an indirect effect through metabolism of hormones in associated liver and nutritional disorders, which are seen in chronic alcoholics. <sup>(1-2)</sup> it is hoped that more awareness of the effects of alcohol on endocrine organs will strengthen the belief in the Quranic prohibition.

**Keywords:** Alcohol, endocrine effects, hypogonadism, pseudo-Cushing's, alcoholic hypoglycemia.

## Alcohol

addiction is a major health and social problem in the United States. Close to 18 million Americans are habitual drinkers. Medical conditions, accidents, and loss of work account for \$26 billion in health care costs. The effect of alcohol on major organs is well known. Practically, alcoholism damages all organs, the important ones being the liver, gastrointestinal tract, central nervous system, pancreas, and heart. However, the effect of alcohol on endocrine and metabolic systems <sup>(1-2)</sup> is neither well described nor appreciated by the general public.

The endocrine effects of alcohol are acute or chronic, and can be contributed indirectly to be existing malnutrition and liver disease. At the pituitary level, one of the actions of alcohol is to decrease the release of vasopressin. Vasopressin is secreted from the hypothalamus, is stored in the posterior pituitary, and it is the hormone that controls urinary output. In chronic alcoholism, as a result of a lack of vasopressin, there is a diuretic state leading to further dehydration. <sup>(3-4)</sup>

Alcoholics <sup>(1)</sup> serum calcium and magnesium levels are low because of malnutrition, malabsorption, diarrhea, and pancreatitis with increased urinary loss.

Serum albumin is also low; therefore, the total calcium is falsely reduced further.

Magnesium and calcium contribute to alcoholic tetany and cardiac arrhythmia. <sup>(5-6)</sup> Alcohol can increase serum triglycerides and VLDL associated with chronic fatty liver and acute pancreatitis, can cause exacerbation of the primary liver and acute pancreatitis, and can cause exacerbation of primary hypertriglyceridemia and

diabetic hypertriglyceridemia. There is also a question of whether it will increase HDL. (7)

There is sodium retention in alcoholics because of increased aldosterone and estrogen and abnormality of the prostaglandin and kallikrein/kinin system. There is also a decrease in the natriuretic factor. The cortisol output from the adrenal cortex increases due to dual action of alcohol on the hypothalamus and pituitary. At the level of the hypothalamus, it causes an increase in CRF; at the level of the pituitary, it causes an increase in CRF; and at the level of the pituitary, it causes an increase in ACTH production.

As a result, pseudo-Cushing's syndrome is not uncommon in alcoholics.

The physical features are very similar to classical Cushing's syndrome, and it is confirmed with excess corticosteroids both in serum and urine. However, it suppresses normally on Dexamethasone, and there is a spontaneous resolution with abstinence. (8)

Alcohol has many effects on carbohydrate metabolism, including glucose intolerance, alcoholic hypoglycemia, alcoholic ketoacidosis, and lactic acidosis. The mechanism of alcoholic hypoglycemia includes a decrease in glycogen storage and decreased production of glucose of decrease gluconeogenesis. One of the features of alcoholic hypoglycemia is that it is prolonged and does not respond to glucagon injections. Therefore, 50% or 10% Dextrose must be used to correct it until the effect of the alcohol has passed.

Alcohol depresses LH and FSH secretion at the pituitary level, and there is also a possibility that it might affect LH-RH releasing hormone at the hypothalamus. The combination of this affects both testicular and ovarian functions. (9) As a result, the serum testosterone is decreased, not only because of a lack of gonadotropin acting on the testes, but also there is an increased clearance of testosterone and metabolites in the liver. Alcohol may also block LH receptors at the testes.

Another factor in both decreased testosterone secretion and decreased spermatogenesis is malnutrition, which lowers FSH and LH hormones.

As a result of decreased testosterone formation, there is a decreased libido, gynecomastia, atrophied testes, and impotence.

Therefore, the myth of alcohol being an aphrodisiac is broken by actually causing more permanent damage on the gonadal system. Not only is there a decrease in testosterone level, but there is also an increased estradiol-testosterone ratio, increased LH and FSH due to decreased feedback and a decreased sperm count.

In women, alcohol can cause oligomenorrhea, amenorrhea, and decreased fertility by acting directly on the ovaries or decreasing LH and FSH to the pituitary. Also, malnutrition in alcoholics affects LH-RH secretion in the hypothalamus, and LH-FSH secretion from the pituitary. (10)

Some of the features of hypermetabolism are seen in alcoholics with proptosis of both eyes, plethora, tachycardia, warm and sweaty palms, and tremors. Thus, the question is, is there any thyroid dysfunction in alcoholism? Indeed, serum T4 is decreased in cirrhosis with a decrease in thyroid-binding globulin. However, there is an increase in the reverse T3 level in liver disease and acute illness as a compensatory phenomenon.

The free T3 level actually may also increase, explaining some of these hypermetabolic features of alcoholism. (11)

In summary, alcohol, in both acute form and in chronic cirrhosis, affects the endocrine and metabolic systems by acting directly or through malnutrition, affecting hormone releasing secretions. We hope that the above information will be understood and passed on to patients by the physicians so that they will practice abstinence or enter into a treatment program and not wait for end-stage liver disease, pancreatitis, or GI bleeding.

## References

1. Van Thiel D, Gavaler J: The endocrine consequences of alcohol abuse. *Alcohol and Alcoholism* 1990;25:341-4.
2. Adler RA: Clinical review 33: Clinically important effects of alcohol on endocrine function. *J Clin Endocrinol Metab* 1992;74:957-60.
3. Loosen PT, Range AJ: Alcohol and anterior pituitary hormone secretion. *Lancet* 1975;11:985-6.
4. Linkola J, Fyhrquist F, and Forsander O: Effects of ethanol on urinary AVP secretion. *Acta Physiologica Scandinavica* 1974; 10 1: 126-8.
5. Laitinen K, Lanberg-Allardt C, Tunninen R, et al.: Transient hypoparathyroidism during acute alcohol intoxication. *N Engl J Med* 1991;324:721-7.
6. Lindholm J, Steiniche T, Rasmussen E, et al.: Bone disorders in men with chronic alcoholism: A reversible disease? *J Clin Endocrinol Metab* 1991;73:118-24.
7. Ben G, Gnudi L, Maran A, et al.: Effects of chronic alcohol intake on

carbohydrate and lipid metabolism in subjects with Type II (non-insulin dependent) diabetes. Am J med 1991;90:70-6.

8. Kirkman S, Nelson DH: "Alcohol-induced Pseudo-Cushing's. Metabolism 1988;37:390-4.

9. VanThiel DH, Cobb CF, Herman GB, Perez L, Gavalier JS: Ethanol-induced testicular injury. Endocrinology 1981;109:2009-15.

10. Van Thiel DH: Alcohol-induced ovarian failure. J Clin Invest 1975;61:624-32.

11. Israel Y, Walfish PG, Orrego H: Thyroid hormone in alcoholic liver disease. Gastroenterology 1979;76:116- 22. Page 90