Research and Reviews Journal of Pharmaceutical Analysis

e-ISSN: 2320-0812
p-ISSN: 2347-2340

A Strange reason for Hypokalemic loss of Motion: Ceaseless Licorice Ingestion

Chandana Eddula* and Darion Macarius

[1]Sri Krishnadevaraya University, Anantapur, Andhra Pradesh, India
[2]American University, USA

Review Article

Received: 04/11/2016
Revised: 10/11/2016
Accepted: 18/11/2016

***For Correspondence**

Chandana E, Sri Krishnadevaraya University, Anantapur, Andhra Pradesh, India.

E-mail:
chandana.eddula@gmail.com

Keywords: Licorice, Hyperkalemic, Mineralocorticoid, Glycyrrhizin, Pharmaceuticals

ABSTRACT

Hypokalemic intermittent loss of motion is an uncommon, autosomal prevailing channelopathy described by muscle shortcoming or loss of motion with a coordinating fall in potassium levels in the blood. In people with this transformation, assaults regularly start in pre-adulthood and most usually happen on arousing or after rest or rest taking after strenuous work out, high starch dinners, suppers with high sodium content, sudden changes in temperature, and even energy, clamor, blazing lights and instigated by cool temperatures. Shortcoming might be mellow and constrained to certain muscle gatherings, or more extreme full-body loss of motion. Licorice concentrates and its key segment, glycyrrhizin, have broad use in nourishments, tobacco items, and snuff, and in customary and home grown prescription. Licorice remove (square, powder, or fluid) might be connected to cigarette tobacco at levels of around 1-4% to upgrade and fit the flavor attributes of smoke, enhance dampness holding qualities of tobacco, and go about as a surface-dynamic operator for fixing application.

INTRODUCTION

Licorice is a mainstream sweetener found in numerous sodas, nourishment items, snacks and home grown pharmaceuticals. The propensity for utilization of such characteristic refreshment is more prevalent in hot situations [1-8]. The conventional conviction that licorice is a solid regular substance without reactions drives its liberal utilization which can every so often be perilous. A few qualities permit the broad use of licorice. Its sweet taste makes it alluring to numerous assembling organizations as a sweetener for some items to veil its sharp taste [9-15].

Its esteem as a refreshment advances its abundance utilization in specific atmospheres. It is likewise utilized as a part of a few restorative signs [16-21]. Its primary constituent, glycyrrhizic corrosive, mirrors mineralocorticoids in its activity (sodium reabsorbtion and potassium discharge). The degree of metabolic and acid–base disturbance can every so often be sufficiently extreme to bring about genuine entanglements [22-28].

Genetics

In patients with transformations in SCN4A or CACNA1S, hence, the channel has a diminished volatility and signs from the focal sensory system can't depolarise the muscle. Subsequently, the muscle can't contract proficiently (loss of motion) [29-35]. The condition is hypokalemic on the grounds that a low extracellular potassium particle focus will bring about the muscle to repolarise to the resting potential all the more rapidly, so regardless of the possibility that calcium conductance occurs it can't be managed. It turns out to be harder to achieve the calcium limit at which the muscle can contract, and regardless of the possibility that this is achieved then the muscle will probably unwind [36-40]. Due to this, the seriousness would be diminished if potassium particle focuses are kept high.

Sources of Licorice

Licorice removes have been utilized for an expanded timeframe in China and Japan as home grown solutions [41-48]. In the United States, glycyrrhizin is for the most part perceived as a protected enhancing specialist. De-glycyrrhizinated licorice (DGL) has been produced to maintain a strategic distance from the symptoms of licorice by expelling the dynamic compound glycyrrhizin and is accessible in cases, capsules, and wafers and fluid [49-57]. Open

e-ISSN: 2320-0812
p-ISSN: 2347-2340

attention to licorice-containing mixes and their potential difficulties is compulsory to maintain a strategic distance from the coincidental utilization of such items [58-65].

DIAGNOSIS

Determination can be accomplished through a specific type of electromyographic (EMG) testing called the long practice test [66-70]. This test measures the abundancy of a nerve reaction (called the Compound Muscle Action Potential or CMAP) for 40 to 50 minutes taking after a couple of minutes of work out. In influenced patients, there is a dynamic fall in the plentifulness of the potential. Other than the patient history or a report of serum potassium low typical or low amid an assault, the long practice test is the present standard for medicinal testing.

Standard EMG testing can't analyze a patient unless they are in an all-out assault at the season of testing. Inciting an assault with practice and eating regimen then attempting oral potassium can be demonstrative, additionally unsafe as this type of PP has a substitute shape known as hyperkalemic occasional loss of motion [71-77]. The indications are nearly the same, however the treatment is distinctive. The old glucose insulin test is perilous and dangerous to the point of being life-debilitating and ought to never be done when different choices are so promptly accessible.

Individuals with hypokalemic intermittent loss of motion are frequently misdiagnosed as having a transformation issue or insane loss of motion since the shortcoming is muscle-based and doesn't relate to nerve or spinal root dispersions [78-84]. The propensity of individuals with hypokalemic intermittent loss of motion to get incapacitated when epinephrine is discharged in "battle or flight" circumstances promote adds to the allurement to misdiagnose the confusion as psychiatric.

TREATMENT

Treatment of hypokalemic occasional loss of motion spotlights on forestalling further assaults and diminishing intense side effects. Staying away from sugar rich suppers, strenuous practice and other recognized triggers, and taking acetazolamide or another carbonic anhydrase inhibitor, may avoid assaults of shortcoming [85-90]. A few patients additionally take potassium-saving diuretics, for example, spironolactone to keep up potassium levels.

Loss of motion assaults can be overseen by drinking one of different potassium salts broke up in water (face off regarding exists over which, if any one specifically, is best utilized, however potassium chloride and bicarbonate are basic). Quickly ingested boluses of fluid potassium are by and large expected to prematurely end an assault, yet a few patients likewise discover positive support comes about with time-discharged potassium tablets. IV potassium is from time to time defended unless the patient can't swallow [91-94]. Maybe every day potassium measurement ought to be much higher than for potassium substitution from straightforward hypokalemia: 100-150 mEqs of potassium is frequently expected to oversee day by day vacillations in muscle quality and capacity.

PROGNOSIS

The visualization for intermittent loss of motion fluctuates. Overactivity, an eating routine that is not low in sodium and starches, or essentially a tragic quality transformation can prompt to a kind of perpetual, low level shortcoming called a "failed assault," or to changeless muscle harm [95-97]. Failed assaults regularly react to additional potassium, cutting sugars, getting a lot of rest, expanding dosages of solution and tender every day practice, for example, short strolls. Perpetual muscle shortcoming is exactly what it sounds like: Permanent, unsalvageable harm to the muscles and related shortcoming. Vacuoles and tubular totals shape in and annihilate solid muscle tissue.

Life traverse is relied upon to be typical, yet assaults can drop potassium to levels sufficiently low to bring about existence debilitating breathing issues or heart arrhythmia. Patients regularly report muscle torment and psychological issues amid assaults [98-100]. Headaches happen in up to half of all hypokalemic occasional loss of motion patients and may incorporate less basic side effects like ghost odors, affectability to light and sound or loss of words. Therapeutic literary works expresses that muscle quality is typical between assaults, however patients regularly report that their pattern quality is in actuality lower than that of sound people.

DISCUSSION

Licorice is a constituent in numerous sustenance items and is accessible in different structures. The general population regularly devours licorice as a result of a conventional confidence in its medical advantage with ignorance of the potential dangers of overconsumption. Licorice-incited mineralocorticoid impact can be decreased

e-ISSN: 2320-0812
p-ISSN: 2347-2340

after suspension of admission, sufficient potassium substitution and spironolactone treatment. A past study exhibited that aldosterone receptor enmity with either spironolactone or eplerenone standardizes pulse. This is credited to the long half existence of glycyrrhetic corrosive and the long length required for the renin–angiotensin–aldosterone hub to standardize, which can take up to 6 months. As a result of its antagonistic impact profile, DGL has been made trying to maintain a strategic distance from inconveniences from glycyrrhizic corrosive.

REFERENCES

1. Kung AW. Clinical review: Thyrotoxic periodic paralysis: a diagnostic challenge. J Clin Endocrinol Metab. 2006; 91:2490-2495.
2. Jurkat-Rott K and Lehmann-Horn F. Genotype-phenotype correlation and therapeutic rationale in hyperkalemic periodic paralysis. Neurotherap. 2007;4:216-224.
3. Rüdel R, et al. Hypokalemic periodic paralysis: in vitro investigation of muscle fiber membrane parameters. Musc Nerv. 1984;7:110-120.
4. Jurkat-Rott K and Lehmann-Horn F. Muscle channelopathies and critical points in functional and genetic studies. J Clin Invest. 2005;115 :2000-2009.
5. Levitt JO. Practical aspects in the management of hypokalemic periodic paralysis. J Transl Med. 2008;6:18.
6. Kim SJ, et al. Reduced expression and abnormal localization of the KATP channel subunit SUR2A in patients with familial hypokalemic periodic paralysis. Biochem Biophys Res Commun. 2010;391:974-978.
7. Kim JB and Kim MH. The Genotype and Clinical Phenotype of Korean Patients with Familial Hypokalemic Periodic Paralysis. J Korean Med Sci. 2007;22:946-951.
8. Arwal A, et al. An evaluation of the efficacy of licorice gargle for attenuating postoperative sore throat: a prospective, randomized, single blind study. Anesth Analg. 2009;109:77-81.
9. Armanini D, et al. Pseudo hyper aldosteronism: pathogenetic mechanisms. Crit Rev Clin Lab Sci. 2003;40:295-335.
10. Armanini D, et al. Treatment of polycystic ovary syndrome with spironolactone plus licorice. Eur J Obstet Gynecol Reprod Biol. 2007;131:61-67.
11. Armanini D, et al. Effect of licorice on the reduction of body fat mass in healthy subjects. J Endocrinol Invest. 2003;26:646-650.
12. Armanini D, et al. Further studies on the mechanism of the mineralocorticoid action of licorice in humans. J Endocrinol Invest. 1996;19:624-629.
13. Armanini D, et al. Licorice reduces serum testosterone in healthy women. Steroids. 2004;69:763-766
14. Arola O. Black arrhythmias. Duodecim. 2003;119:2145-2147.
15. Bannister B, et al. Cardiac arrest due to liquorice induced hypokalaemia. Br Med J. 1977;2:738-739.
16. Baron J. Side-effects of carbonoxolone. Acta Gastroenterol Belg. 1983;46:469-484.
17. Basso A, et al. Licorice ameliorates postural hypotension caused by diabetic autonomic neuropathy. Diabet Care. 1994;17:1356.
18. Bernardi M, et al. Effects of prolonged ingestion of graded doses of liquorice by healthy volunteers. Life Sci. 1994;55:863-872.
19. Blachley J and Knochel J. Tobacco chewer's hypokalemia: licorice revisited. N Engl J Med 1980;302:784-785
20. Bocker D and Breithardt G. Induction of arrhythmia by licorice abuse. Z Kardiol. 1991;80:389-391.
21. Bramont C, et al. Cerebral vascular accident caused by alcohol-free licorice. Presse Med. 1985;14:746.
22. Calo L, et al. Effect of aldosterone and glycyrrhetinic acid on the protein expression of PAI-1 and p22(phox) in human mononuclear leukocytes. J Clin Endocrinol Metab. 2004;89:1973-1976.
23. Caradonna P, et al. Acute myopathy associated with chronic licorice ingestion: reversible loss of myoadenylate deaminase activity. Ultrastruct Pathol. 1992;16:529-535.
24. Campana A, et al. An unusual cause of cardiac arrest. Ital Heart J Suppl. 2003;4:510-513.
25. Cartier A, et al. Occupational asthma due to liquorice roots. Allergy. 2002;57:863.
26. Cayley F. Potassium deficiency in p-aminosalicylic acid therapy: cardiac and paralytic effects. Lancet. 1950;1:447-448.
27. Chamberlain J and Abolnik I. Pulmonary edema following a licorice binge. West J Med. 1997;167:184-185.

e-ISSN: 2320-0812
p-ISSN: 2347-2340

28. Chamberlain T. Licorice poisoning, pseudoaldosteronism and heart failure. JAMA. 1970;213:1343.
29. Crampton J. Glycyrrhizinophilia as a cause of edema. Bull Mason Clin. 1961;15:89-92.
30. Crean A, et al. A sweet tooth as the root cause of cardiac arrest. Can J Cardiol. 2009;25:357-358.
31. Dobbins K and Saul R. Transient visual loss after licorice ingestion. J Neuroopthalmol. 2000;20:38-41.
32. Dong S, et al. Activation of rapid signaling pathways and the subsequent transcriptional regulation for the proliferation of breast cancer MCF-7 cells by the treatment with an extract of Glycyrrhiza glabra root. Food Chem Tox. 2007;45:2470-2478.
33. De klerk G, et al. Hypokalaemia and hypertension associated with use of liquorice flavoured chewing gum. Br Med J. 1997;314:731-732.
34. Eriksson J, et al. Life-threatening ventricular tachycardia due to liquorice-induced hypokalaemia. J Intern Med. 1999;245:307-310.
35. Epstein M, et al. Liquorice toxicity and the renin-angiotensinaldosterone axis in man. Br Med J. 1977;1:209-210.
36. Farese S, et al. Glycyrrhetinic acid food supplementation lowers serum potassium concentration in chronic hemodialysis patients. Kidney Int. 2009;76:877-884.
37. Fenwick G, et al. Liquorice, Glycyrrhiza glabra L.-composition, uses and analysis. Food Chem. 1990;38:119-143.
38. Francini-Pesenti F, et al. Liquorice-induced hypokalaemia and water retention in the absence of hypertension. Phytother Res. 2008;22:563-565.
39. Fraunfelder F. Ocular side effects from herbal medicines and nutritional supplements. Am J Ophthalmol. 2004;138:639-647.
40. Funder J, et al. Mineralocorticoid action: target tissue specificity is enzyme, not receptor mediated. Science. 1988;243:583-585.
41. Gross E, et al. Hypokalemic myopathy with myoglobinuria associated with licorice ingestion. N Engl J Med. 1966;274:602-606.
42. Hall R and Clemett R. Central retinal vein occlusion associated with liquorice ingestion. Clin Exp Ophthalmol. 2004;32:341.
43. Harada T, et al. Congestive heart failure caused by digitalis toxicity in an elderly man taking a licorice-containing Chinese herbal laxative. Cardiol. 2002;98:218.
44. Hasegawa J, et al. Echocardiographic findings of the heart resembling dilated cardiomyopathy during hypokalemic myopathy due to licorice-induced pseudoaldosteronism. Cardiovasc Drugs Ther. 1998;12:599-600.
45. Heard K, et al. Hypokalemia complicating sodium para-aminosalicylate therapy for pulmonary tuberculosis. Med J Australia. 1950;2:606-612.
46. Heck A, et al. Potential interactions between alternative therapies and warfarin. Am J Health Syst Pharm. 2000;57:1221-1227.
47. Hoffmann D and Hoffmann I. The changing cigarette, 1950–1995. J Toxicol Environ Health. 1997;50:307-364
48. Holmes A, et al. Pseudohyperaldosteronism induced by habitual ingestion of liquorice. Postgrad Med J. 1970;46:625-629.
49. Hughes J, et al. Re: 'preterm birth and licorice consumption during pregnancy'. Am J Epidemiol. 2003;158:190-191.
50. Ishiguchi T, et al. Myoclonus and metabolic alkalosis from licorice in antacid. Intern Med. 2004;43:59-62.
51. Kaufman MR, et al. Patient Susceptibility & Technical Factors Associated with Persistent Diaphragmatic Paralysis after Interscalene Nerve Block. J Anesth Clin Res. 2016;7:667.
52. Alam N, et al. Thyrotoxic Periodic Paralysis. J Clin Case Rep. 2016;6:839.
53. Guevara JC. Is Unilateral Vocal Fold Paralysis a Rare Complication of Spinal Anesthesia? A Case Report. J Anesth Clin Res. 2016;7:640.
54. Rotratsirikun Y and Talmadge RJ. Rapid Changes in NFAT-directed Transcriptional Activity after Muscle Paralysis Induced by a Spinal Cord Injury. Int J Phys Med Rehabil. 2014;2:241.
55. Homagk L, et al. Therapy of Spine Metastasis Causing Paralysis Symptoms-Operation and Rehabilitation. J Surg 2014;10:145-148.

e-ISSN: 2320-0812
p-ISSN: 2347-2340

56. Bell AH and Smith DM. When the Leader Hits the Wall: A Case of Organizational Paralysis. J Entrepren Organiz Manag. 2012;1:e101.
57. Habaragamuwa BWP and Halpegamage NW. Flaccid Motor Paralysis Induced by Hyperkalemia. J Neurol Neurophysiol. 2012;3:134.
58. Vijaya Krishna V. Congenital Facial Paralysis. Anaplastol. 2012;1:e101.
59. Tung NH, et al. (2016) *In vitro* Fertilization with Mouse Sperm Activated by Components of Licorice Root Extract. Nat Prod Chem Res. 4:217.
60. Uto T, et al. Interaction Analysis of Glycyrrhizin on Liquorice Extract-Induced Apoptosis of Human Leukemia Cells by Knockout Extract. Nat Prod Chem Res. 2013;1:105.
61. Konik E, et al. Coronary Artery Spasm, Hypertension, Hypokalemia and Licorice. J Clin Case Rep. 2012;2:143.
62. Takeuchi M, et al. Hypokalemia and Related Symptoms by Yokukansan in Patients with Behavioral and Psychological Symptoms of Dementia (BPSD): A Retrospective Study of Elderly Inpatients. Adv Pharmacoepidemiol Drug Saf. 2016;5:210.
63. Mohta M, et al. An Unusual Presentation of Hypokalemia. J Anesth Clin Res. 2014;5:389.
64. Hayriye G, et al. A Rare Cause of Hypokalemia in the Emergency Department: Gitelman Syndrome. J Clin Case Rep. 2012;2:231.
65. Comachio J. Clinical Commentary: About Acupuncture and Electro acupuncture in Advances in Muscle Weakness. J Mult Scler. 2016;3:168.
66. Hardjo Lugito NP, et al. Worsening Muscle Weakness in Myasthenia Gravis Patient Suffering Dengue Infection. J Trop Dis. 2014;2:140.
67. Mahmoodpoor A, et al. Optimizing Energy Supply by Parenteral Nutrition in the Critically-Ill: Muscle Weakness and its Monitoring. Emergency Med. 2014;4:174.
68. McCormack T. Pre-Operative Blood Pressure Measurement and Management in the United Kingdom and Ireland – A Joint Guideline by the Association of Anaesthetists and the British Hypertension Society. J Perioper Crit Intensive Care Nurs. 2016;2:130.
69. Porpino SKP, et al. Developing New Organic Nitrates for Treating Hypertension: A Review. J Hypertens. 2016;5:232.
70. Chaowu Y, et al. Diastolic Pulmonary Arterial Pressure as a Prognostic Indicator for Closure of Atrial Septal Defect with Severe Pulmonary Arterial Hypertension. J Hypertens. 2016;5:231.
71. Zha P, et al. An RN/CHW Exemplar: Managing Hypertension in an Urban Community. J Comm Pub Health Nurs. 2016;2:135.
72. Kabore T and Lazar J. Prevalence and Risk Factors for Pre-Hypertension Among Adults in Burkina Faso, West Africa. J Comm Pub Health Nurs. 2016;2:130.
73. Chalupova L, et al. CTRP1: A Molecular Link between Obesity and Hypertension. J Mol Biomark Diagn. 2016;7:289.
74. Ali SA. Use of Smokeless Tobacco in Medical Students and Hypertension. Occup Med Health Aff. 2016;4:240.
75. Stoicescu M. The Risk of Sudden Decrease of Severe Arterial Hypertension. J Clin Exp Cardiolog. 2016;7:460.
76. Chauhan R, et al. Hypertension and the Aged. J Gerontol Geriatr Res. 2016;S5:002.
77. Pagano D, et al. Portal Hypertension Model in Pigs. J Clin Exp Transplant. 2016;1:e101.
78. Aberha M, et al. Prevalence and Factors Associated with Anxiety among Patients with Hypertension on Follow Up at Menelik- II Referral Hospital, Addis Ababa Ethiopia. J Psychiatry. 2016;19:378.
79. Trailokya A. Will Azilsartan - An Eight ARB Bring Paradigm Shift in Hypertension Management Practices in India? Cardiovasc Pharm Open Access. 2016;5:189.
80. Li M, et al. To Live Long, Eat Less Salt: Salt Intake Reduction Promotion and Hypertension Control in China. Health Care: Curr Rev. 2016;4:169.
81. Soltani HM, et al. The Effect of Fasting During Ramadan on Blood Pressure in Patients with Controlled and Mild Hypertension. J Hypertens. 2016;5:227.
82. Skride A, et al. Pulmonary Arterial Hypertension Associated with Adult Congenital Heart Disease, when Inoperable becomes Operable: A Case Report. J Pulm Respir Med. 2016;6:350.

e-ISSN: 2320-0812
p-ISSN: 2347-2340

83. Sarfaraz S, et al. Non Pharmacological Use of *Daucus carota* Juice (Carrot Juice) as Dietary Intervention in Reducing Hypertension. Enz Eng. 2016;5:147.
84. Berezin AE. Is Elevated Circulating Galectin-3 Level A Predictor of Pulmonary Artery Hypertension Development and Progression? Clin Med Biochemistry Open Access. 2016;2:114.
85. Manolis A and Doumas M. Erectile Function in Cardiovascular Disease and Hypertension: the Role of Nebivolol. J Hypertens. 2016;5:226.
86. Lv Y, et al. Non-Hypersplenism Causes of Peripheral Cytopenias in Patients with Cirrhotic Portal Hypertension: A Review. J Hypertens. 2016;5:223.
87. Li X, et al. Angiotensinogen M235T, β2 Adrenergic Receptor Arg16Gly and Aldosterone Synthase C-344T Gene Polymorphisms and Essential Hypertension among Han Population Living at High Altitude in China. J Hypertens. 2016;5:222.
88. Abdel-hamid ER, et al. Association of Angiotensin Converting Enzyme Gene Polymorphism and Possible High Risk Factors with Essential Arterial Hypertension in Egyptian Patients. Mol Biol. 2016;5:165.
89. EL-Adawy NM, et al. Fibroblast Growth Factor-23: A Possible Cause of Pulmonary Hypertension and Left Ventricle Hypertrophy in Hemodialysis Patients. J Clin Exp Cardiolog. 2016;7:449.
90. Kiuchi MG, et al. Case Report: Renal Sympathetic Denervation + Pulmonary Vein Re-isolation in Patients with Long-stand Persistent Atrial Fibrillation and Resistant Hypertension. Does it Work? Surgery Curr Res. 2016;6:268.
91. Rosemberg MAS, et al. Significant Associative Factors for Hypertension Among New US Immigrants: An exploration of the 2003 New Immigrant Survey (NIS) Data. J Comm Pub Health Nurs. 2016;2:118.
92. Mandapaka RT and Rachabathuni S. Prevalence of Hypertension and its Relationship between Dietary Salt Intake in Urban Population. J Community Med Health. 2016;6:426.
93. Shahsuvaryan M. Systemic Hypertension and the Eye: Highlighting a Comorbidity. J Clin Exp Cardiolog. 2016;7:442.
94. Chatterjee A, et al. Congenital Cytomegaloviral Infection Causing Severe Pulmonary Hypertension in a Newborn with a HIV Seropositive Mother - A Case Report from Eastern India. J AIDS Clin Res. 2016;7:567.
95. Cifuentes D, et al. Targeting Hypertension to Manage Alzheimer's Disease: Rational and Promise. J Alzheimers Dis Parkinsonism. 2016;6:228.
96. Feyh A, et al. Role of Dietary Components in Modulating Hypertension. J Clin Exp Cardiolog. 2016;7:433.
97. Guney F, et al. Intracranial Hypertension in Behcet Disease: A Case Report. J Clin Case Rep. 2016;6:748.
98. Bogari DF, et al. The Prevalence of Hypertension in Endodontic Clinics: A Pilot Study. Dentistry. 2016;6:370.
99. Vongskan N, et al. The Effect of an Integrated Savings and Community Based Health Education Program among Older Adults with Hypertension: A Quasi-Experimental Controlled Study, Bangkok Province, Thailand. J Health Edu Res Dev. 2016;4:167.
100. Silva RP, et al. Who is the Patient with Suspected White Coat Hypertension? J Clin Exp Cardiolog. 2016;7:428.