Improving the Quality of Reporting a Cohort Study

Dear Editor:

An article titled «The effect of extremely low frequency electromagnetic fields on pregnancy and fetal growth, and development» was published in Arch Ion Med. 2013; 16(4): 221 – 224.1 This study was an epidemiologic analytical cohort study based on Strengthening the Reporting of Observational Studies in Epidemiology (STROBE). The title of the article should specify the study design and indicate the methodology,2 however, the above mentioned title seems to indicate that this study is a randomized trial.3,4 Rather, this is a cohort study.

Epidemiological studies are prone to confounders. The use of appropriate statistical tests is necessary to control confounding. One of the methods used in data analysis is the regression model, which by entering all confounding factors causes a decrease in confounding.5 Chi-square and t-tests alone cannot control confounding. Therefore, the use of a regression model reduces confounding factors and adjusts the results.2,5 In cohort studies, incidence, relative risk and absolute risk (in a meaningful time period) should be reported.2 In conclusion, I would like to mention that for improving the results of the study, relative risk and absolute risk should be reported and confounding should be controlled.

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References


Peroneal Nerve Palsy after Rapid Weight Loss Due To Uncontrolled Diet in a Patient Treated With Oral Isotretinoin

Dear Editor:

We read the study of Entezari-Maleki et al. with great interest.1 The authors assessed evaluation of isotretinoin use and they found that the doses of isotretinoin are incorrect in many cases, nor have patients been properly counseled about isotretinoin teratogenicity and the seriousness of its side effects.

Isotretinoin’s main indication is for the treatment of severe nodular cystic acne. Although it is a potent therapeutic agent, the drug has several adverse effects. If patients are not accurately informed about adverse effects and how to manage them, negative results may occur.

On this occasion we would like to share our experience. We present a rare case of peroneal palsy due to massive weight loss that resulted from an uncontrolled diet in order to avoid triglyceride alteration induced by isotretinoin.

Case: An 18-year-old male patient presented with right foot drop accompanied by decreased sensation over the dorsum of the right foot. The patient’s medical findings and family history were unremarkable. The risk factors related to peroneal palsy in a healthy adolescent were searched. He denied prolonged leg crossing, history of any leg fracture and casting. He had been on isotretinoin treatment for his acne for a three-month period and was advised to pay attention his diet because this drug is associated with increased triglyceride levels. The patient undertook an uncontrolled strict diet and lost 15 kg (20% of his body weight) over a two-month period. Systemic examination was normal. On the neurological assessment, there was complete weakness (1/5 strength) of his right foot dorsiflexion and eversion in addition to numbness over the dorsum of the right foot. Deep tendon reflexes were normal. Routine laboratory examinations, radiographic examination of the patient’s right knee and the lumbar spine magnetic resonance imaging were normal. On electrophysiological evaluation, there was a complete conduction block at the level of the fibular head. He was diagnosed with peroneal neuropathy (PN) due to weight loss for which he received nutritional support, physical therapy and a foot orthosis. Within a few months, improvement was observed.

The peroneal nerve usually develops entrapment neuropathy caused by external compression located mainly at the fibular head where the nerve runs superficially and causes PN. PN has been linked to endocrine or metabolic disorders such as diabetes mellitus, alcoholism, thyrotoxicosis or vitamin B depletion as well as mechanical causes. Also the association between weight loss and the development of pressure paralysis of the peroneal nerve has been described. Because the nerve is only covered with skin, subcutaneous tissue and fat pad, any excessive weight loss that reduces the fatty cushion protecting the nerve is susceptible to pressure damage.2-4 There is no established association between PN and isotretinoin. Chroni et al. have determined in their study that short-term administration of oral isotretinoin in young patients does not cause clinical or subclinical neuropathy.5

In conclusion, the loss of more than 15% of body weight can lead to the development of PN. Therefore it is important to keep this mind. In patients who take isotretinoin, a balanced diet should be recommended.

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shown that adipokines regulate different stages of atherosclerosis and secreted by adipocytes and adipose tissue.4 It has also been shown that visfatin is a novel adipokine with different functions, for which exist a plethora of research on its characteristics and roles. Visfatin levels may change in patients with inflammation such as diabetes mellitus, hypertension, obesity, metabolic syndrome, cardiovascular disease, hyperlipidemia, hormonal abnormality, known malignancy and smoking habit.

In addition, any abnormality in thyroid function tests, known malignancy, anemia, smoking and any medication such as statins can potentially interfere with inflammation and might change visfatin levels. Thus the authors should have excluded all of these factors in this study. Secondly, the selection of control group was not correctly defined in this study. For example, they have not explained whether or not the control group underwent coronary angiographies. Therefore some control group patients might have coronary artery disease. It would be better if the authors had defined these factors in the study group.

Finally, visfatin alone in the absence of other inflammatory markers may not give information to clinicians regarding the patient’s endothelial inflammatory condition. In our opinion we believe it should be evaluated together with other serum inflammatory markers.

There is no conflict of interest.

Serum Visfatin Levels Should Be Evaluated with Further Markers of Endothelial Inflammation

Dear Editor:

We read the article “Elevated Serum Visfatin Levels in Patients with Acute Myocardial Infarction” written by Mazaherioun et al. with interest.1 The authors aimed to assess any potential relationship between blood visfatin levels, anthropometric variables, and known risk factors of atherosclerosis and acute myocardial infarction (AMI). They determined that serum visfatin levels were significantly higher in AMI patients compared to controls. This finding was consistent with other studies that have shown a contribution of visfatin to atherosclerosis and plaque destabilization which, in turn, leads to myocardial infarction. We believe that these findings will act as a guide for further studies about risk factors of atherosclerosis and AMI.

Coronary artery disease is a major public health problem in Iran2 and one of the leading causes of death worldwide.3 Obesity is one of the most important risk factors for atherosclerosis of the coronary arteries, and consequently, increases the risk of myocardial infarction. Adipose tissue is an energy storing organ with endocrine properties which has a role in systemic vascular inflammation. Various pro- and anti-inflammatory mediators and cytokines are secreted from adipose tissue. Adiponectin, a peptide hormone and member of the family of adipokines, is absolutely expressed in and secreted by adipocytes and adipose tissue.4 It has also been shown that adipokines regulate different stages of atherosclerosis, from endothelial dysfunction to plaque destabilization and rupture. Visfatin is a novel adipokine with different functions, for which exist a plethora of research on its characteristics and roles. Visfatin levels may change in patients with inflammation such as diabetes mellitus, hypertension, obesity, metabolic syndrome, cardiovascular disease, hyperlipidemia, hormonal abnormality, known malignancy and smoking habit.

In addition, any abnormality in thyroid function tests, known malignancy, anemia, smoking and any medication such as statins can potentially interfere with inflammation and might change visfatin levels. Thus the authors should have excluded all of these factors in this study. Secondly, the selection of control group was not correctly defined in this study. For example, they have not explained whether or not the control group underwent coronary angiographies. Therefore some control group patients might have coronary artery disease. It would be better if the authors had defined these factors in the study group.

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