Comment on "Cardiac troponin I and creatine kinase MB isoenzyme in patients with chronic renal failure"

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To the Editor:

We read the article "Cardiac troponin I and creatine kinase MB isoenzyme in patients with chronic renal failure" written by Larry M. Flores-Solís et al. with interest.[1] The authors concluded that the cut-off value proposed in this study for both cardiac troponin I (cTI) in patients with chronic renal failure (CRF) (stage 3 to 5) to diagnose acute coronary syndrome (ACS) is significantly different from that of the general population.[1] Thank to the authors for their contribution of a study designed and documented successfully. We believe that these findings will guide further studies about cTI and creatine kinase MB(CK-MB) levels and CRF.

cTI and CK-MB levels in patients presenting with a suspected acute coronary syndrome help in the diagnosis of patients. It was proposed in a previous study that estimation of the glomerular filtration rate based on a normal creatinine level on admission provided important information on short-term prognosis[2] and also it was recommended that glomerular filtration rate should be included in the risk assessment of patients with normal serum creatinine levels. But there is another issue which is the basement of the present study that patients with high creatinine levels due to CRF may be expected to have elevated cTI levels due to decreased glomerular filtration. However, cTnI is again the preferred biomarker for myocardial damage in patients with CRF[3] as it is one of the least changed markers. When the patients with an elevated serum creatinine levels, especially those on dialysis treatment,[4] are candidates for increased cardiovascular accidents, it is an additive factor that this situation may lead to some difficulties and can be more problematic in diagnosis of patients with a suspected acute coronary syndrome.

In addition to renal failure, cTnI levels may frequently be measured above normal values in several disease states in which myocardial necrosis is not a prominent aspect, especially in pulmonary embolism, heart failure, liver cirrhosis, septic shock, and arterial hypertension.[5] Our challenge is on the issue that the study would be stronger if all the additional factors that might elevate cTI levels were mentioned in the study.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Response to the comment on "Cardiac troponin I and creatine kinase MB isoenzyme in patients with chronic renal failure"

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To the Editor:

We are grateful for the comments and interest of Dr Cakar M. and his collaborators regarding our study entitled "Cardiac troponin I and creatine kinase MB isoenzyme in patients with chronic renal failure" in which we included 484 consecutive chronic renal failure (CRF) patients with suspected acute coronary syndrome (ACS). Due to CRF patients being a high cardiovascular risk population, we decided to apply a strategy to improve diagnostic sensitivity and we obtained a cardiac troponin I (cTnI) cut-off value that was significantly different from that used for the general population to diagnose ACS, although it had been reported that by decreasing the 99th percentile of cTnI (cut-off value),

more patients with ACS[1] at risk of recurrent myocardial infarction (MI) or death would be identified, but the diagnosis of MI would increase by 47%.[2]

Moreover, it is important to know that any cardiac damage may cause the release of troponins from myocytes as complexes or as free troponin in circulation and there are many causes of increased troponins that are not related to ACS, among which are: 1) cardiac causes (congestive heart failure, tachycardia, left ventricular hypertrophy, myocardial contusion, myocarditis, pericarditis, cardiac amyloidosis, heart surgery, cardioversion, percutaneous coronary intervention, coronary vasospasm, radiofrequency ablation, other), 2) non-cardiac causes (chronic renal failure, sepsis, stroke, pulmonary embolism, primary pulmonary hypertension, critically ill patients, chemotherapy, sympathomimetic agents, intense resistance exercise, other) and 3) methodological causes (fibrin, haemolysis, heterophile antibodies, rheumatoid factor, other).[3,4]

In patients with CRF, the mechanisms that may explain the nonspecific increases in cTnI are mainly further heart damage and decreased kidney function.[5] In our study, the main causes of increased cTnI in the Other Cardiac Pathologies (OCP) group were congestive heart failure, atrial fibrillation and tachycardia, while in the Other Non-cardiac Pathologies (ONCP) group they were high blood pressure, stroke and pneumonia, but we believe that all the causes described should be taken into consideration as potential confounding factors whenever high cTnI concentrations are found in patients with CRF and an absence of ACS.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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B) BRIEF PAPERS ON RESEARCH AND CLINICAL EXPERIMENTS

Immunoglobulin A nephropathy could be a clue for the recurrence of gastric adenocarcinoma

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To the Editor:

The association of nephrotic syndrome that was presented with IgA nephropathy (IgA-N) and malignancy has been previously reported.

IgA –N was first described by Berger and Hinglais in 1968. It is now generally known to be the most common cause of primary glomerulonephritis worldwide and is characterized by mesangial proliferation and deposition of IgA.[1] Malignancies that have been reported to be associated with IgA-N include Hodgkin's disease,[2] and non-Hodgkin lymphoma[3] renal cell carcinoma,[4] cancers of the lung,[5] larynx and esophagus.[6]