LETTERS TO THE EDITOR

Transient Left Ventricular Apical Ballooning Without Coronary Artery Stenosis: A Form of Stunning-Like Phenomenon?

We read with great interest the study published by Tsuchihashi et al. (1) in the July issue of the Journal. The study presented detailed findings with regard to the unusual phenomenon that mimic acute coronary syndrome.

Left ventricular (LV) contraction abnormalities without coronary stenosis have been previously reported in physically or emotionally stressed patients (2–4). Epicardial coronary spasm has not been confirmed in these patients even while ST-segment is elevated in the electrocardiogram (ECG). Kawai et al. (3) stated this could be a form of cardiomyopathy, termed “ampulla cardiomyopathy”. However, Tsuchihashi et al. (1) suggested that the histological change was similar to that of catecholamine-induced myocardial damage and that microvascular spasm was involved.

We have been interested in this form of reversible LV dysfunction, and recently reported the involvement of impaired coronary microcirculation in transient LV contraction abnormalities (5). Coronary arteriography revealed no significant stenosis in the epicardial arteries. Relative coronary flow reserve measured by intracoronary Doppler guide wire was significantly reduced, which suggested severely decreased coronary microcirculation in these patients. Contrast myocardial echocardiography revealed that the impaired ventricular perfusion was reversible. According to our findings about coronary microcirculation, the histological changes seen in these patients might be a result of a stunning-like phenomenon due to microvascular abnormalities.

Finally, the investigators carefully used the term “transient left ventricular apical ballooning” in the title of their study (1). The exact mechanism of this reversible contraction abnormality still remains unclear. A term defining the pathogenesis of this syndrome will be necessary in the near future.

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REFERENCES


REPLY

We appreciate the interest shown and the comment given by Ako et al. in regard to our recent clinical study (1) on a heart syndrome with transient left ventricular (LV) apical ballooning without coronary artery stenosis mimicking acute myocardial infarction. As mentioned in my study and others (2,3), coronary vasospasm under various physical and mental stresses, including administration of adrenergic drugs, might be considered as an initial etiological basis of this novel syndrome. Impaired coronary microcirculation in this syndrome was shown by Ako et al. (4) using an intracoronary Doppler flow-wire technique. The possibility of transient ischemia including microvessel vasospasm as an initiating factor of this syndrome could not be ruled out; however, we speculate that vasospasm is not the main cause, for the following several reasons. First, autopsy findings in some cases were different from those of myocardial ischemia (5). Second, impaired microcirculation during the follow-up period will not be direct evidence for considering the etiology of this syndrome, because the possibility of delayed recovery of impaired microcirculation due to transient wall motion abnormality is not excluded in this syndrome. Recent scintigraphic evaluation by Dr. Owa (co-author) showed a transient (but persistent for several months) perfusion–metabolism mismatch in the apex (6). Our study also showed a representative case with delayed recovery of coronary microcirculation (1).

Important etiological causes suspected from our study include stress cardiomyopathy caused by vigorous stress (catecholamine exposure) (6–8), dynamic midventricular obstruction due to basal hypercontraction (9) and/or secondary myocardial ischemia caused by apical ballooning (increased wall tension). However, as already mentioned in the discussion (1), our study was a retrospective investigation and there are several limitations. Further cases, therefore, should be investigated to determine the pathogenesis of this heart syndrome.

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REFERENCES

Anasarca and Low Electrocardiogram Voltage

In a recent article in the Journal, Madias et al. (1) reported an inverse relationship between electrocardiogram (ECG) voltage and changes in body weight due to fluid loss or retention in patients with anasarca. The investigators could find no reference in the literature to a link between low ECG voltage and anasarca. A direct correlation has been reported, however, between ECG voltage and the concentration of serum albumin (2). Anasarca is frequently associated with a lowered serum albumin concentration (3). To ascertain how much the Madias et al. (1) study expands existing knowledge, it would be necessary to correlate the observed changes in ECG voltage with concomitant changes in serum albumin. Are the investigators able to do this?

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REFERENCES


REPLY

I am grateful to Dr. Brennan for directing me to literature describing a direct correlation of albumin and amplitude of QRS complexes (1). To his request I have modeled an analysis of our data after the study of Dr. Heaf (1). Our patients were critically ill, were followed for a long time (34.5 ± 36.7 days) (2) and inevitably experienced gradual reduction of albumin, despite our concerted efforts to use enteral and/or parenteral feeding. We had not measured albumin daily, but frequently. Thus, I was able to employ values that were the closest to the admission and peak weight albumin values of our patients. The albumin levels available for my analysis were checked on days 0.61 ± 1.71 for admission, and on days 0.43 ± 1.79 of peak weight time point.

Correlation of the sums of all 12 QRS leads (ΣQRSs) (2) from admission with the corresponding albumin values revealed an r = 0.057 and a p = 0.77. A similar analysis, carried out for the ΣQRSs obtained on the day of the peak weight and the corresponding albumin values, showed an r = 0.38 and a p = 0.04. Correlation of the percent change in the ΣQRSs between admission and points of the peak body weights, with the percent change in the corresponding albumin values, revealed an r = 0.37 and a p = 0.053.

The difference in our results from the admission time point and the time of peak weight is hard to explain. If there was a real relationship between QRS amplitude and albumin, such a discrepancy should not have been encountered. Moreover, I find it odd to correlate absolute values of albumin with absolute values of amplitude of QRS complexes, taking into consideration that the latter can vary widely among both healthy subjects and patients, while the normal range of the former is 3.3 to 5.3 g/dl in our laboratory.

I find the correlation of change in each of the two correlates more meaningful scientifically, and in our data we found an almost significant association between the drop of albumin and loss of QRS amplitudes. The correlation coefficient was much smaller than the one we reported (r = 0.61) (2) for the association of weight gain and ΣQRSs drop. However, some association may exist between albumin and QRS amplitudes, and may be confirmed in patients in whom both daily electrocardiograms and albumin measurements are implemented.

Does this mean that the hypoalbuminemia was causally related to the drop of ΣQRS in our patients, or was this biochemical abnormality “an innocent bystander”? Innocent it is not, for certainly it contributes to the extent of water retention; however, the causative mechanism for the drop in ΣQRSs must have been the weight gain (or fluid retention). Correlation of % weight gain and % albumin drop in our 28 patients was poor (r = 0.02, p = 0.9); also, correlation of the % weight loss and % albumin rise in 9 patients was poor (r = 0.5, p = 0.1). It would have been very interesting to have data on weight change in the patients reported by Heaf (1).

There is evidence that edema fluid has low resistivity (3); thus, its effect on the transfer of cardiac potentials from the heart to the body surface can be explained by Ohm’s law. I am not aware of any work ascribing any mechanistic role for albumin per se in modulating the ECG voltage. Finally, the fact that patient 26 (2) gained 122.4 lb and lost 68.3% of his ΣQRS, while his albumin increased from 1.8 g/dl to 2.7 g/dl, and subsequently lost 77.4 lb and gained 185.9% of his ΣQRS (Fig. 6 of Madias et al. [2]) while his albumin increased merely from 2.9 to 3.0 g/dl, casts serious doubt on the contention that a causative relation between albumin and QRS amplitudes really exists.