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Hong Kong Cardiology During the Severe Acute Respiratory Syndrome (SARS) Outbreak

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The first case of Severe Acute Respiratory Syndrome (SARS) broke out at the end of February 2003 in Hong Kong, and had since made significant impact on all walks of life in Hong Kong, including the medical profession. The Cardiology community and practicing cardiologists were affected in different ways during this period.

Right at the time and before anybody knew about SARS, the World Congress on Cardiac Pacing and Electrophysiology were being held between February 19-22, 2003. In retrospect, the organizer, the Hong Kong College of Cardiology, was very fortunate to have successfully hosted the meeting, with over 4,000 participants from 20 countries attending.¹ If SARS had occurred earlier, the attendance would have been dismal, and most probably the entire congress would have to be cancelled. With divine blessing perhaps, all the faculty and attendees had enjoyed their visit and returned home safely.

During the outbreak, there was severe manpower and bed shortage for SARS patients. As a result, routine cardiac activities almost came to a standstill. Many patients cancelled their appointments for fear of contacting SARS, and the numbers of outpatient attendance and cardiac procedures were dramatically reduced. In some hospitals, catheterization laboratory had to be closed e.g. Princess Margaret Hospital. In other hospitals, e.g. Queen Mary Hospital, one of the two cardiac catheterization laboratories was closed during this period. A similar situation also occurred in the private sector. For the usually overworked cardiologist, this seemed to be a good time for respite. However, in reality, we felt very frustrated from this inactivity, and we were not alone.

Many cardiologists in the Hospital Authority took active role in the management of SARS patients. Because of their experience in critical care, some of them worked in high risk areas delivering medical care directly to those in need. They had made important contributions to the medical community. In the early outbreak of SARS, a number of cardiologists had contracted SARS and, some became very sick. We are now thankful that they have all survived, and most have already returned to full clinical duties.

SARS has provided opportunity for those academically inclined. A group of cardiologists in Hong Kong has just published the first landmark study on the effects of SARS virus on cardiac function. Li et al² reported subclinical diastolic dysfunction in 46 patients with SARS, 14 of them were ventilator dependent patients. While there was no systolic dysfunction observed in these cases as a group, ventilated patients tended to have lower ejection fraction which was related to the serum level of lactase dehydrogenese. Left ventricular function recovered among the survivors.

Many meetings were cancelled or postponed. The 11th Annual Scientific Session of the Hong Kong College of Cardiology, scheduled in June 2003, was initially postponed to August, and finally to October

Opinions expressed are views of the authors and not necessarily the view of the editorial board or the Hong Kong College of Cardiology. Received October 20, 2003; revision accepted October 21, 2003.
31-November 2, 2003. The organizing committee has made tremendous effort in liaising with the faculty and the industry to put together a meeting of very high standard. It will be one of the largest medical meetings after the SARS outbreak.

In summary, cardiologists in Hong Kong have suffered, made significant contributions and maintained their duties to their patients during the SARS outbreak. The hosting of the 11th Annual Scientific Meeting is a testimony of their commitment. This editorial in written as a tribute to all cardiologists who have served the people of Hong Kong selflessly during the difficult SARS period.

References

Letter to the Editor

An Open Letter to The Hong Kong College of Cardiology Regarding Electron Beam Computed Tomography (EBT)

Dear Editor,


My experience with Electron Beam Computed Tomography (EBT) now dates me at over 20 years of research and clinical practice, initially in cardiac physiology, but for the past decade in coronary atherosclerosis, atherosclerosis imaging, and Preventive Cardiology. Thus, I feel I can speak from some level of authority as well as considerable experience. The point in question relates to the use of EBT and coronary calcium scoring for defining coronary disease in a non-invasive manner. I will admit, like many trained formally in traditional Cardiology and brought up with the idea that definition of coronary stenoses severity was our standard in defining coronary disease, that my initial efforts in using coronary calcium were directed towards that end.

Our initial studies from autopsy specimens during my tenure at the Mayo Clinic defined that coronary calcium area, by direct histologic comparisons as well as by EBT, was a valid surrogate to defining in situ atherosclerotic plaque.1-3 The next step was to then determine application of the non-invasive EBT calcium score to defining stenosis severity in patients referred for clinically indicated invasive coronary angiography. This is where our initial efforts demonstrated that we could in fact NOT define coronary stenoses severity was our standard in defining coronary disease, that my initial efforts in using coronary calcium were directed towards that end.

Our initial studies from autopsy specimens during my tenure at the Mayo Clinic defined that coronary calcium area, by direct histologic comparisons as well as by EBT, was a valid surrogate to defining in situ atherosclerotic plaque.1-3 The next step was to then determine application of the non-invasive EBT calcium score to defining stenosis severity in patients referred for clinically indicated invasive coronary angiography. This is where our initial efforts demonstrated that we could in fact NOT define coronary stenosis severity adequate for clinical diagnostic purposes using the EBT calcium score.4,5 I was initially taken-aback by these findings and also joined with my cardiology colleagues in defining the potential for "false positive" results using EBT. In fact, however, as I have come to learn, the results were NOT false, but clearly showed that coronary calcium in any given site is 100% specific for coronary atherosclerosis, but NOT specific for the degree of narrowing. My Mayo Clinic colleagues and I in fact further investigated this and reported on a resolution of the problem in a separate necropsy study.6 What we found was that coronary calcium did continue to define coronary atheromatous plaque, but failed to define site-by-site stenosis severity due to the phenomenon of coronary artery remodeling, mentioned by Dr. Ko in his Editorial. This concept was initially reported by Dr. Glagov, at the University of Chicago, where he and his colleagues demonstrated that coronary plaque may progress by expanding the mural surfaces and yet not necessarily, until its later stages, result in luminal narrowing.7

The traditional view from most Cardiologists had been that a definition of "coronary artery disease" was a stenosis of significant magnitude, defined as generally representing a singular or multiple narrowing defined on a "luminogram" of >50-70%. Over the years, however, we have subsequently learned that atherosclerosis is a diffuse disease and that focusing, as a means of defining "disease", on a stenosis of >50% vastly underestimates the severity and extent of the underlying atherosclerosis in a given individual. Dr. Steven Nissen, Vice-Chair of Cardiology at the Cleveland Clinic, has perhaps been the best at pointing out the inadequacies of traditional coronary angiography by performing simultaneous investigations using intravascular ultrasound (IVUS). He has demonstrated that individuals may well have a "normal" coronary angiogram (i.e. luminogram) and yet have "significant" atheromatous disease8 due to "positive coronary remodeling". Subsequent to these investigations my colleagues and I compared "apples with apples" demonstrating that the coronary artery calcium score...
by EBT correlated well with IVUS definition of atheromatous plaque.9,10

Dr. Ko states in his Editorial that coronary angiography is "…the diagnostic gold standard…". Many recent authors would dispute that statement as it relates to atheromatous plaque, as discussed above, although it does remain the clinical standard for defining the site of "significant luminal narrowing" that are targets for PCI and/or road maps for bypass surgery. His statement later however that "EBCT (is)…not specific or accurate in so far as the diagnosis or the staging of clinically significant coronary disease…" is frankly and explicitly inaccurate. The goal of EBT is to define the extent of atherosclerotic plaque and thus its "accuracy" in defining clinically significant disease should be better understood in its ability to predict risk of coronary events or clinically significant outcomes. The premise (or promise, more appropriately) of EBT is not to "define significant coronary artery disease", as implied by Dr. Ko, but to define the extent of coronary atheromatous plaque. Thus he has mis-stated his claim when he indicated that "The premise of EBCT is thus defeated". In fact the "promise" of EBT has been fortified by collective literature that has added incrementally to our understanding over the past decade.

Dr. Ko is indeed correct in that the formation of mural coronary artery calcification, in response to the inflammatory nature of coronary disease, may well form to provide a lattice of support in an attempt to render the plaque stable. However, the pathological literature shows11 that 80% of the "culprit" lesions in necropsy studies contain histologic calcium hydroxyapatitie. However, many stable lesions show the same predilection underscoring that coronary calcium is neither a unique marker for stable nor unstable plaque. Although so called "vulnerable" plaques have been shown to be more predominantly lipid laden than calcium laden, this does not limit the value of EBT and calcium scoring in estimating the overall extent of atherosclerosis plaque present. In any given individual about 2/3 of plaque is in fact "scar" and about 1/3 is more predominantly "lipid laden". Thus EBT cannot be used to define which plaque is unstable, but can and has been shown to define disease extent (even compared with the "extent" of angiographic disease or the extent of thallium perfusion defects12).

The proof of the above statements must be founded in the prediction of clinical outcomes using coronary calcium by EBT as a measure of disease. Although Dr. Ko quotes one paper suggesting that EBT provided no incremental value over the sum of ALL conventional risk factors13 [of note and lost to most "crities" is that risk factors also failed to predict events adequately in the same cohort], subsequent research (even from that same laboratory) has demonstrated that the coronary calcium score by EBT provided prognostic information independent and incremental to conventional risk factors.14-17 Angiographic studies over the past 25 years have demonstrated that the extent of coronary disease is directly related to prognosis.18,19 The presence of moderate amounts of coronary calcium (i.e., EBT scores exceeding 100) has been shown in several studies to predict cardiac events in symptomatic20 and asymptomatic21 individuals. The relative risk of a cardiac event in an individual with a moderate to high calcium score by EBT compared to an individual who has no or minimal coronary calcium has ranged form a mean of 8.66:122 in a meta-analysis review of EBT to as high as 15:123 over a 2-4 year follow up time period in a more recent publication. The magnitude of the risk associated with coronary calcium is underscored when one considers the relative risks of developing symptomatic coronary artery disease in younger patients based upon conventional individual "risk factors" (15 year follow up Framingham study of initially asymptomatic men) is only 1.9:1 for an elevated Lp(a), 1.8:1 for total cholesterol >240 mg/dl., 1.8:1 for an HDL <35 mg/dl, 3.6:1 for cigarette smoking, and 1.2:1 for systolic hypertension.24

The data show that, although EBT cannot define coronary stenosis severity, it CAN suggest that with higher scores, or in the presence of symptoms24 that further examination such as stress testing, EBA (electron beam non-invasive coronary angiography), or even formal angiography may be warranted. However, this determination must be made on a case-by-case basis and is a clinical decision made by an experienced practitioner. Outcomes of such subsequent testing are then used to assist in the patient's care.

The current overall guidelines for EBT test interpretation can be expressed in several bullet points
as provided below. These statements, supported by published literature, still must function only as overall "guides" for the clinician. However, in my opinion, these interpretation are in fact being well put forward by experienced practitioners currently performing EBT examinations in Hong Kong.

Interpretation and recommendation for EBT heart scanning and CAC scoring:

- A negative test (i.e. score=0, no detectable coronary calcium) makes the presence of atherosclerotic plaque, including unstable or vulnerable plaque, highly unlikely.
- A negative test (score=0) makes the presence of significant luminal obstructive disease highly unlikely (negative predictive power by EBT on the order of 95-99%).
- A negative test is consistent with a low risk (reported to be <0.5% per year26) of a significant cardiovascular event [infarction or sudden death] in the next 2-5 years.
- A positive test (i.e. a score >0) confirms the presence of a coronary atherosclerotic plaque (100% specificity for fibro-atheromatous plaque).
- The greater the EBT calcium score, the greater the atherosclerotic burden in men and women, irrespective of age, and the greater "likelihood" of more advanced luminal disease.5
- The total (summed) coronary calcium score correlated best with the total amount of coronary atherosclerotic plaque, although the true "atherosclerotic burden" is underestimated and, without use of contrast (as in an EBA study), "soft" plaque may not be well appreciated (but data are advancing to address this issue using non-contrast EBT27).
- A high calcium score (an Agatston score >100 or any score above the 75th percentile for age and sex) denotes advanced coronary atherosclerosis and provides a rationale for intensified atherosclerosis lowering therapy and assignment of coronary risk to a "coronary disease risk-equivalency" status (i.e. secondary prevention goals such as a target LDL-c range <100 mg/dl).

References


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Letter to the Editor

CT Scanning the Coronaries for Calcium – Why the Controversy?

Dear Editor,

Few recent topics in cardiovascular medicine have engendered as much debate as the now vexed issue of high resolution CT scanning of the coronary arteries for calcium, as a predictor of coronary disease risk. As presymptomatic detection of atherosclerosis is a vital and still unsolved problem in clinical practice, open discussion about this topic is very important. It is equally important, however, that clinical practitioners have a clear idea of the 'state of the art', in order to help guide management of individual patients.

The essential facts concerning coronary calcium scanning are not in dispute. Non-invasive tests to assist in risk stratification are clearly needed, for low and intermediate-risk patients. CT scanning can detect calcium in the coronary arteries and this is clearly a marker of the burden of atherosclerosis in the coronary circulation. If the coronary calcium score is '0', subsequent risk of events in the medium term is very low (although not zero) and if the calcium score is high, this clearly indicates not only the presence of extensive atherosclerotic plaque, but also improved risk prediction, compared with conventional risk factor assessment only. Nevertheless, despite excellent sensitivity for disease, CT scanning for coronary calcium has much less good specificity, resulting in a high number of 'false positive' tests (in terms of predicting actual events). This may cause undue concern in asymptomatic subjects and can lead to a series of subsequent cardiac diagnostic tests, which may have low yield. It is also not in dispute that this is an area worthy of further investigation, and accordingly data are accumulating rapidly about the role of this test in clinical practice.

Against this very promising background information, the experience in many countries, including Australia as well as Hong Kong, has been that the test has been introduced to the public with occasionally with aggressive marketing and advertising strategies, directed at the general community. This has sometimes occurred before the medical profession has accepted the benefits of coronary CT scanning. Publicity such as 'the most important advertisement you'll ever read' (or phrases similar) has appeared in high circulation newspapers and journals. Not surprisingly, the uptake rate by a public well educated and concerned about the dangers of heart disease has been high. Some subjects have been very relieved by early detection of a high calcium score and subsequent workup, others have been relieved by a reassuringly negative calcium score, but many have been worried by the news that they have 'coronary calcium', without a clear diagnostic or therapeutic pathway for them or their doctors to follow. Marketing direct to the public along these lines is contrary to the more traditional medical model with which doctors are comfortable, wherein evidence is carefully evaluated, learned societies and/or key opinion leaders come to a consensus, and then information is disseminated to the public through specialists, GPs and medically-based education.

Herein lies the genesis of some of the controversy surrounding coronary CT scanning. Those specialists involved in centres which have 'marketed' coronary CT scanning have been cast by some as 'irresponsible entrepreneurs' preying on the latent fears of a gullible public, whereas members of the profession who have adopted a more conservative stance can be cast as latter-day Luddites, delaying the uptake of promising new technologies which may have real health benefits, to the detriment of the general community. Such tensions often accompany the introduction of novel and exciting technologies, but can be particularly acute when significant sums of money can be made or lost, on the acceptance or otherwise of expensive new techniques.

A series of excellent review articles and letters have appeared in the Journal this year, commenting with different degrees of enthusiasm on the burgeoning use of electron beam CT for the non-invasive assessment...
of coronary artery calcium and its role in predicting coronary risk. These are all sensible and well-balanced viewpoints from highly regarded experts in the practice of cardiovascular medicine.

How then, to find perspective on this issue? This is an exciting area of research, about which we will all read more in the coming years. At the time of writing, the American College of Cardiology and the American Heart Association have produced consensus documents on electron beam CT for the diagnosis and prognostic significance of coronary artery disease, suggesting that this technique not be employed for non-invasive screening of asymptomatic subjects at risk of cardiovascular events. Newer data have since been published, however, which may in time result in the revision of these recommendations, especially if similar results can be obtained in other supportive studies. As a community, we need to keep an open mind about such new data, whilst vigorously protecting our patients from unproven diagnostic and/or therapeutic strategies.

Some other questions need to be answered about coronary CT scanning. Firstly, cost/benefit analyses will be important in a cost-conscious era where other putative markers of coronary risk (such as high sensitivity CRP assays and ultrasound measurement of arterial wall thickness) may be considerably cheaper than CT scanning, and not carry any radiation risk. Secondly, are non-electron beam CT scanners as reliable, in the detection of calcium? Most importantly of all, cardiologists will want to have practical guidance on which calcium scores in which patients should prompt which further diagnostic tests and/or treatment options.

This is an important topic, and the Journal is to be congratulated for publishing high quality articles, commentaries and letters on this subject. In the next 5 years, we will almost certainly find out whether coronary CT scanning for calcium is 'hype' or 'hope'. In the interim, a search for the right test for presymptomatic determination of coronary risk remains a 'Holy Grail' of internal medicine, as the worldwide prevalence of atherosclerotic disease continues its inexorable climb.

References