High risk pregnancies and future cardiovascular disease

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Management of Cardiovascular Diseases in both sexes is equal, and we as Cardiologists usually meet with our patients of both sexes with CHD late in life, where at this level of interference the damage has already took place and we can only try to manipulate this damage. Women on the other side are at risk of developing CVD across most of their lives due to hormonal level variations, starting in early puberty, passing by adulthood curving around pregnancies and reaching menopause and post menopause era. Here I would like to put emphasis on pregnant women without CVD and developing high risk pregnancies in the form of preeclampsia, GDM, PPCM, and giving birth to low weight babies. At this level these women will start a subclinical progressive atherosclerosis that will be clinical within 1-10 years post-partum, exposing these women to CV events endangering their lives. These changes can be avoided in millions of women if we act early preventing future damage. If we add to this group those who are already having an existing heart diseases-acquired or congenital- and those who had Cancer therapy and developed Cardiotoxicity, the damage is more and may be fatal. The message here is early preventing these women from crossing to the damaging zone.

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Patients with non-obstructive coronary artery disease admitted with acute coronary syndrome carry a better outcome compared to those with obstructive coronary artery disease

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Purpose: The present study aimed to investigate the clinical profile, in-hospital and 3-months outcome of ACS patients with insignificant coronary stenosis on a coronary angiography.

Methods: This prospective observational study included 200 consecutive patients admitted with ACS. Group I (100 patients) included patients with insignificant CAD (all lesions <50% stenosis). Group II (100 patients) included patients with one or more lesions >70% stenosis. Patients with previous CABG were excluded.

Results: Patients with insignificant CAD were significantly younger (61 vs. 67 years, p<0.001), more likely to be females (41% vs. 23%, p=0.006), less likely to smoke (p=0.006), less likely to have diabetes mellitus (p<0.001), and less likely to have history of CAD (p=0.042) or prior PCI (p=0.037). At presentation these patients were also less likely to have typical anginal pain (61% vs 91%, p<0.001), less likely to have heart failure (9% vs 30%, p<0.001), less likely to have ischemic ST-segment changes (10% vs 46%, p=0.001), had lower elevations in peak troponin I (p=0.001) and CK-MB levels (p<0.001), with lower LDL-C (p=0.006), and higher HDL-C levels (p=0.020). They were less likely to be treated with thienopyridines (p<0.001), statins (p<0.001), b-blockers (p=0.002), ACEI/ARBs (p=0.007), and higher rates of calcium channel blocker therapy (p<0.001), this trend continued at discharge. They had lower prevalence of major adverse clinical events at follow up (readmission for ACS (p=0.009), revascularization (p=0.035), recurrent chest pain (p=0.029), cardiogenic shock (p=0.029).

Conclusion: Patients with ACS and insignificant CAD have different clinical profile and outcome compared to those with significant disease.

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