Abstract:
The repeated demonstration of biomarkers of endothelial cell and leukocyte activation has suggested that the maternal syndrome of pre-eclampsia arises from a generalised maternal inflammatory systemic response incorporating a substantive component of endothelial cell dysfunction. Reports of reduced endothelium dependent dilatation in isolated resistance arteries and from non-invasive methods in vivo indicate a major contribution to the systemic vasoconstriction, characteristic of the syndrome. The recent discovery of raised concentrations of soluble fms-like tyrosine kinase1 (sFlt1) and the soluble transforming growth factor β (TGF-β) coreceptor (sEng) which indirectly may compromise endothelial function, adds to the growing list of potential origins of endothelial disturbance. Most are proposed to originate from placental underperfusion and associated placental oxidative stress, although it is clear that not all women with pre-eclampsia have reduced utero-placental blood flow, and other precipitating factors, including dyslipidaemia and hyperglycaemia are likely to contribute. Endothelial dysfunction, unlike pre-eclampsia, does not resolve post-partum, and persistence of the defect may underpin the increased risk of cardiovascular disease in later life.

Key words:
pre-eclampsia, endothelium, resistance artery, oxidative stress, sFlt1