Response to Are Aldosterone Levels Inappropriately Low in Preeclampsia?

Verdonk et al.\(^1\) raise an interesting hypothesis that increased angiotensin II sensitivity and angiotensin II type 1 receptor autoantibodies in preeclampsia may not lead to inappropriately lower aldosterone levels, a hypothesis not supported by both experimental and clinical studies. In humans, aldosterone levels are relatively high throughout gestation in the setting of higher plasma volume.\(^2\) In contrast, in subjects with preeclampsia, aldosterone levels are relatively low (when compared with normotensive pregnancy), despite lower plasma volume. In addition, circulating levels of renin and angiotensin II are lower in preeclampsia.\(^3\) As noted by Verdonk et al.,\(^1\) suppressed renin–angiotensin II–aldosterone levels during preeclampsia are accompanied by enhanced angiotensin II sensitivity in the vasculature. Recently, Siddiqui et al.\(^4\) reported that angiotensin II type 1 receptor autoantibodies, which have been implicated in enhancing angiotensin II sensitivity during preeclampsia, directly inhibit aldosterone production by inducing soluble fms-like tyrosine kinase 1 and by causing vascular impairment in adrenal glands. These data support our experimental findings that aldosterone levels are correlated inversely with circulating soluble fms-like tyrosine kinase 1 levels in rodents.\(^5\) In summary, aldosterone is lower during preeclampsia because of excess circulating soluble fms-like tyrosine kinase 1 levels in preeclampsia.

Disclosures

S.A. Karumanchi is a coinventor of multiple patents on preeclampsia markers. He serves as a consultant to Roche, Siemens, and Beckman Coulter and has financial interest in Aggamin LLC. The other authors report no conflicts.

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