direct effect on vascular endothelium, nerves and astrocytes [8]. Being rare, little has been reported about the patterns of electronic fetal heart rate monitoring in fetal alkalosis. In our case, loss of variability and late decelerations might have reflected the fetal alkalosis and brain injury, though they are not specific. EEG has been shown as useful tool for assessment of neonatal brain injury [9]. As our case had shown severe EEG abnormalities in form of burst suppression and cranial scan suggestive of porencephalic cysts, shortly after birth, the timing of cerebral insult was considered perinatal in origin.

In conclusion, our case is unique suggesting that maternal excessive vomiting could have contributed to fetal alkalosis and pseudo-Barter syndrome, and severe perinatal brain injury. We recommend that any acid-base imbalance in pregnant women should be monitored carefully and corrected optimally to ensure the fetal well-being.

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REFERENCES