important for AADC stability. It was postulated that the direct enteral administration of this compound could be dephosphorylated by intestinal phosphatases, absorbed into the bloodstream, and cross the blood-brain barrier. Conceivably, this could provide a more efficient supply of PLP to the brain than pyridoxine (that requires multiple metabolic steps).[9,10] However, the necessary efficacious dose has not been established[9] and clinical outcomes still remain unclear. At the moment, even with early diagnosis, the overall prognosis of patients with AADC deficiency remains guarded, particularly in terms of neurological outcomes and autonomic disturbance.[2]

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Corrigendum

"Transient myeloproliferative disorder and non-immune hydrops fetalis in a neonate with trisomy 21" (February 2014;20:78.e3-4). On page 78.e3 (3rd paragraph, lines 6-8), the sentence should have read "Rhesus isoimmunisation is the commonest immune aetiology, and alpha-thalassaemia is a non-immune cause." rather than "Rhesus isoimmunisation is the commonest immune aetiology, and beta-thalassaemia is the commonest non-immune cause." as printed. We regret the error. The article is correct at www.hkmj.org.