Cerebral Infarction in Childhood Acute Lymphoblastic Leukaemia Treated with Low Dose *E. coli* Asparaginase

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**Abstract**

The occurrence of thrombosis complicating L-asparaginase therapy in childhood acute lymphoblastic leukaemia has been supported by both laboratory and clinical evidences, but the exact pathogenic mechanism and predisposing factors remain elusive. Two children with acute lymphoblastic leukaemia (ALL) treated with chemotherapy developed cerebral infarction during the third week of induction therapy which consists of relatively low dose *E. coli* asparaginase (6,000 iu/m²/dose three times weekly). Altered hemostatic profile was observed in both patients during the attack but they were likely to be induced by the asparaginase treatment as shown by the return of normal profile in the post-chemotherapy period for the survived patient. There were inconsistent observations on the role of pre-existing prothrombotic conditions in previous studies and our findings further suggest that the cause is likely to be multifactorial. More investigations are needed to clarify if co-existing prothrombotic defects, either inherited or acquired, play a significant role in the causation of thrombosis in local childhood ALL patients receiving asparaginase therapy.

**Key words**

Childhood acute lymphoblastic leukaemia; Cerebral thrombosis; L-asparaginase

**Introduction**

Alterations in haemostasis have been well documented in patients with acute lymphoblastic leukaemia (ALL).1–3 These thrombotic events are typically seen during the period of induction therapy consisting of L-asparaginase, which is an essential drug in the treatment of childhood ALL. Both laboratory and clinical evidences supported the occurrence of thrombosis as a complication of L-asparaginase therapy, 2–10 but the role of other predisposing factors such as co-existing congenital thrombophilic condition remain elusive. We report two cases of cerebral thrombosis in children with ALL managed in our unit using the Hong Kong Children Cancer Study Group (HKCCSG-93) protocols which was modified from the UKALL-XI and UKALL-R1 regimens.11 The thrombosis in these two cases has been attributed as sporadic complication of L-asparaginase treatment.

**Case 1**

An 8-year-old Chinese girl with standard risk B-lineage ALL (low initial white count of 2.8 x 10⁹/L; CD 10(+), cytoplasmic mu chain negative; hyperdiploidy by cytogenetics study) developed cerebral infarct during the third week of induction therapy using the HKCCSG chemotherapy regimen A protocol. The induction therapy