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Background: Many relapsing remitting multiple sclerosis (RRMS) patients develop irreversible progressive neurological disability. Reported clinical outcome varied. We aimed to study clinical outcome of Chinese RRMS patients.

Methods: Only RRMS patients with MRI brain and/or spinal cord abnormalities fulfilling Barkhof's criteria for dissemination in time and space followed up in our hospital were recruited for this retrospective study. Patients with neuromyelitis optica or neuromyelitis optica spectrum disorders were excluded.

Results: Eighty RRMS patients were studied. Their mean onset age was 27.5 (range, 12-50) years, mean disease duration was 16.8 (range, 1.5-30) years; 61 (73%) were female. Seventy-two (90%) patients had CSF oligoclonal bands; 74 (93%) patients had spinal cord MRI abnormalities compatible with inflammatory demyelination. Their mean number of relapses in the first 2 years was 1.8 (range, 0-6). At latest follow-up, 24% patients had EDSS score 2 or less, 33% had EDSS 2.5-4, 54% had EDSS 4 or more, 36% had EDSS 6 or more, and 41% patients developed secondary progressive multiple sclerosis. The median time from symptom onset to reach EDSS 6 was 22 years, and the median age at reaching EDSS 6 was 59 years old. Multivariate cox-regression analysis revealed that demographic characteristics, presenting neurological features, number of relapses in the first 2 years and immunomodulatory therapies (azathioprine and beta-interferon) did not affect time to reach EDSS 6.

Conclusion: The median time for our relapsing remitting multiple sclerosis patients from symptom onset to reach EDSS score 6 (walking require unilateral assistance) was 22 years. Beta-interferon did not confer long-term neurological benefit in RRMS.

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Introduction: Sox proteins are a family of transcription factors with high-mobility-group DNA-binding domain (HMG box) homologous to SRY, which are implicated in embryogenesis and neoplastic transformation. However, their roles in leukaemogenesis are unclear. Human Sox7, constituting a subfamily with Sox17 and Sox18 were cloned and characterised. In this study, we examined the expression of Sox7 in leukaemia, especially in lymphoid leukaemia cells and umbilical cord blood (UCB) cells.

Methods: Bone marrow (BM) or peripheral blood samples of patients with myeloid (acute myeloid leukaemia [AML], myelodysplastic syndrome [MDS], chronic myelogenous leukaemia [CML]) and acute lymphoid leukaemia (ALL) and samples of UCB were prospectively collected. Mononuclear cells were isolated by Ficoll. CD34⁺, CD34⁻ subfractions and CD34⁺CD38⁺ and CD34⁺CD38⁻ subfractions were isolated by microbeads or by sorting. Expression of Sox7 was evaluated by reverse transcriptase-polymerase chain reaction (RT-PCR) and quantitative real-time PCR (Q-PCR). Methylation of CpG island in Sox7 promoter was evaluated by methylation-specific PCR.

Results: Sox7 was relatively highly expressed in ALL and normal samples while exhibited almost undetectable expression in myeloid leukaemia cells. In cord blood, all of the six samples showed Sox7 expression. Sox7 expression in CD34⁺ subfraction is higher than that in CD34⁻ cells of ALL samples and UCB. Sox7 expression in CD34⁺CD38⁺ is lower than that in CD34⁺CD38⁻ subfraction of ALL samples and UCB. In the 12 patients selected, Sox7 promoter of the patient without Sox7 expression is methylated. The other samples showed Sox7 expression and no methylation was observed in the respective Sox7 promoter.

Conclusion: Sox7 gene is expressed in most cases of ALL and normal BM and UCB. However, it was silenced in myeloid malignancies including AML, MDS and CML. In ALL and UCB, it was preferentially expressed in the primitive CD34⁺CD38⁻ population, suggesting its role in the regulation of haematopoietic and leukaemia stem cells.

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