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| CLINICAL Utility of Molecular Testing in  T-lymphoblastic leukaemia/lymphoma  Diagnostic Utility  T-lymphoblastic leukaemia/lymphoma (T-ALL) is characterised by recurrent chromosomal rearrangements leading to aberrant expression of transcription factors including *TAL1*, *TAL2*, *TLX1*, *TLX3*, *HOXA*, *LMO1,* *LMO2*, *LYL1*, *NKX2-1* and *SPI1*. Transcriptomic analysis has also been used to identify T-ALL subgroups based on the unique gene expression profile associated with these aberrant transcription factors1,2.  Mutations in *NOTCH1* (~70%) and its negative regulator *FBXW7* (15%-25%) are frequently observed in T-ALL and are found in all genetic subgroups1,3,4.  Loss of *CDKN2A*/*CDKN2B* by 9p21 deletion is a highly recurrent finding in T-ALL (~70%)5.  Several pathways are dysregulated by secondary somatic mutations or copy number changes including the RAS-MAPK signalling pathway (*NRAS*, *KRAS*, *NF1*), JAK-STAT pathway (*IL7R, JAK1*, *JAK3*, *STAT5B*) and PI3K-AKT pathway (*PTEN* deletion)1,6,7.  Additionally, alterations in epigenetic modification (*PHF6*, *EZH2*, *EED*, *SUZ12*, *DNMT3A*), transcription (*ETV6*, *RUNX1*, *GATA3*, *WT1*)and ribosomal genes (*RPL5*, *RPL10*, *CNOT3*) are recurrently observed1,8-10.  Early T-cell precursor acute lymphoblastic leukaemia (ETP-ALL) is enriched for mutations in genes involved in JAK-STAT signalling, RAS signalling and epigenetic regulation1,2. *FLT3* mutations are more frequently observed in ETP-ALL than T-ALL1.  Some cases of ETP-ALL are characterised by *BCL11B* rearrangement, which shares gene expression signature with mixed phenotype acute leukaemias (T/myeloid MPAL) with *BCL11B* rearrangement11.  *TP53* mutations at diagnosis are less common in paediatric T-ALL (<5%) compared to adult T-ALL (~10%)1,12,13 and are more frequently observed at relapse7.  Prognostic Utility  The prognostic effect of gene mutations in T-ALL is not consistent across studies and needs to be interpreted in the context of co-mutation patterns and measurable residual disease.  A *NOTCH1*/FBXW7/*RAS*/*PTEN–*based classifier has been proposed in T-ALL where low risk is defined by the presence of *NOTCH1*/*FBXW7* mutations in the absence of *RAS*/*PTEN* mutations14,15.  *TP53* mutations have been associated with inferior outcomes in T-ALL in some studies12,13.  References  **1.** Liu Y, et al. 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