

性型の自験例のうち、増悪・急性転化した症例について、その時点における臨床所見に関して後方視的な検討を行なった。

B. 研究方法

当科で経験した慢性型、くすぶり型90症例 (Takasaki Y. et al. Blood 2010) について、病型分類の診断基準に着目し、増悪様式について検討した。90例中12例では診断時にLDH上昇など (11例) または骨髓浸潤による血球減少 (1例) のため治療を開始されていた。残りの症例のうち、経過中に増悪した44例を解析対象とした。病型分類の基準に準じて、以下のように増悪様式を分類した。①ATLの悪化による中枢神経、骨、腹水、胸水、消化管病変の出現、②ATLの悪化による11.5mg/dlを超える補正血清Ca値の上昇、③ATLの悪化による正常上限の1.5倍 (慢性型では2倍) を超える血清LDH値の上昇、④リンパ節病変の増悪 (増加)、⑤上記以外で問診、理学所見、血液検査などに基づいて臨床的に判断されたATLの増悪・急性転化。

C. 研究結果

検討した44例全例で、①から④のいずれかの項目に該当しており、⑤に該当する症例は認めなかった。44例中32例で高LDH血症を、29例でリンパ節病変の増悪を、20例で臓器浸潤を、8例で高カルシウム血症を来していた。多くの症例で複数項目の増悪を同時に来していたが、臓器浸潤単独例を1例、高LDH血症単独例を4例、リンパ節病変増悪単独例を3例認めた。増

悪時のPSについて検討したところ、44例中16例でPSの悪化 (≥ 3) を伴っていた。

(倫理面への配慮)

ヘルシンキ宣言および臨床試験に関するわが国の倫理指針に従って研究を実施した。

D. 考察

臨床的に増悪と判断された症例のうち、リンパ節病変単独の増悪を認めた症例の中には慢性型の症例が1例含まれていた。この症例では、現行の病型分類の基準では増悪の基準は満たしていなかった。増悪・急性転化と判断された症例のうち約35%では、PSの悪化 (≥ 3) を伴っており、病型分類の診断基準は急性転化の早期診断には不十分な可能性が示唆された。ATLに対して、近年、同種造血幹細胞移植や抗体医薬などの有用性が報告されており、適切な診断に基づく治療介入時期の判断が必要である。

E. 結論

ATLのゲノム異常を対象とした本研究班の解析結果により、ATLの病勢・病態に特異的な遺伝子異常が同定され、急性転化の診断に有用な分子マーカーが開発されることが期待される。

F. 健康危険情報

なし

G.研究発表

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H. 知的財産権の出願・登録状況

なし

III. 研究成果の刊行に関する一覧表

研究成果の刊行に関する一覧表

雑誌

発表者氏名	論文タイトル名	発表誌名	巻号	ページ	出版年
Karube K, Nakagawa M, Tsuzuki S, Takeuchi I, Honma K, Nakashima Y, Shimizu N, Ko YH, Morishima Y, Ohshima K, Nakamura S, Seto M.	Identification of FOXO3 and PRDM1 as tumor suppressor gene candidates in NK cell neoplasms by genomic and functional analyses.	Blood	118	3195-3204	2011
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