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Correlation Management of Blast Phase Chronic Myeloid Leukaemia: A Rare Phenomenon

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Description

Chronic Myeloid Leukaemia (CML) is an uncommon kind of myeloproliferative neoplasm, with a frequency of 0.7-1.0/100,000 people. CML is distinguished by the Philadelphia (Ph) chromosome, a reciprocal translocation between the long arms of chromosomes 9 and 22, with the resulting shorter chromosome 22 containing the oncogenic fusion protein *BCRABL1*, a constitutively active tyrosine kinase.

CML is a three-part condition. The majority of patients arrive in Chronic Phase (CP), which is characterized by an elevated white cell count, splenomegaly, and, on rare occasions, hyper viscosity symptoms. According to the EUTOS population based registry, 4%-5% of patients appear in the Accelerated Phase (AP), a transitory stage of disease associated with additional genetic alterations, an increase in blasts and primitive cells, cytopenias, and, in many cases, treatment resistance. An additional 1% to 2% have Blast Phase (BP; sometimes known as 'blast crisis') CML, a leukaemia with a dismal prognosis that can be myeloid, lymphoid, or mixed. Patients may progress on therapy in addition to presenting with *de novo* BPCML. The introduction of *BCRABL1* specific Tyrosine Kinase Inhibitors (TKIs) has reduced the rate of progression to AP or BP from 5% to 20% to 1% to 5% annually, with the greatest risk of advancement occurring within the first year after diagnosis. While TKIs provide great results for patients with CPCML, with the majority of CP patients expecting to live normal lives, TKI responses in BP are few and of short duration, with most patients dying from refractory disease within a year after developing BPCML. As a result, BPCML is a key area of therapeutic need in which innovative ways to improve patient outcomes are urgently needed.

The World Health Organization and European Leukemia Net have different diagnostic criteria for BP CML, with the WHO citing 20% or more blasts in peripheral blood or bone marrow and the ELN 30% or more blasts. Cortes examined outcomes for BPCML patients using both WHO and ELN criteria.

Overall Survival (OS) was considerably higher in patients with 20%-29% blasts against those with 30% or more blasts, with three year OS of 42% *versus* 10%, respectively. The ELN criteria of 30% or more blasts were utilized as an admission criterion in the recent MATCHPOINT clinical study for UK patients with BPCML. Extra medullary blast growth, regardless of disease phase in the bone marrow, is also indicative of BPCML.

If BPCML is suspected, a bone marrow aspirate should be taken to evaluate morphology, immunophenotyping to establish if the phenotype is myeloid, lymphoid, or mixed, and a thorough karyotype to identify Any further Chromosomal Abnormalities (ACAs).

ACAs that are diagnostic of APCML, such as extra Ph chromosome, iso chromosome 17q, trisomy 8 or 19, may also be present at the time of diagnosis of *de novo* BPCML or after progression from CP or APCML. 12 If the aspirate is a dry tap, a trephine should be performed, and a part of this should be sent for karyotyping if possible. A BCRABL1 kinase domain mutation test should be undertaken at the time of *de novo* BP diagnosis or when the patient progresses to AP or BPCML.

A panel assessment using Next Generation Sequencing (NGS) to detect typically mutant lymphoid or myeloid genes may also be investigated; however this is still an experimental method in CML.