Reunião anual SPH
SOCIEDADE PORTUGUESA DE HEMATOLOGIA
15·16·17 novembro 2018
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POSTERS

16 de Novembro de 2018 17h00 | Foyer dos Posters

PO54

PONATINIB INDUCES A SUSTAINED DEEP MOLECULAR RESPONSE IN A CML PATIENT WITH AN EARLY RELAPSE WITH A T315I MUTATION FOLLOWING ALLOGENEIC HSCT

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Background: Less than 5% of patients of chronic myeloid leukemia (CML) patients express atypical transcript types, of which e19a2, with breakpoints in the micro breakpoint cluster region (μ-BCR) and coding for the p230 BCR-ABL1 protein, is the most frequently encountered. p230 CML is associated with various clinical presentations and courses with variable responses to first-line Imatinib.

Case presentation: Here we report a case of Imatinib resistance due to an E255V mutation, followed by early post-transplant relapse with a T315I mutation that achieved a persistent negative deep molecular response ($MR^{5.0}$) after treatment with single-agent Ponatinib. Using CastPCR, we could trace back the presence of the T315I mutation to all the RNA samples up to the detection of T315 mutation by Sanger sequencing shortly after allogeneic hematopoietic stem cell transplantation (HSCT).

Conclusion: This case illustrates the major interest of Ponatinib as a valid treatment option for e19a2 CML patients who present a T315I mutation following relapse after HSCT.