HM-27. A randomized control trial comparing peginterferon-α-2a versus observation after stopping tyrosine kinase inhibitor in chronic myeloid leukaemia patients with deep molecular response for at least two years: Interim analysis

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Introduction: Treatment free remission (TFR) is a fairly new treatment concept in chronic myeloid leukaemia (CML) that develops after two frontier studies from French and Australia published in 2010. About 40% of CML patients, who have achieved deep molecular response (DMR) with tyrosine kinase inhibitor (TKI), are able to remain in TFR after stopping their TKI. Studies are going to search means to increase TFR rate. Consolidative therapy using interferon (IFN), the standard treatment of CML before era of TKI, is a logical possibility because of data suggesting IFN-induced immunity towards the leukemic clone. We conducted the first randomized controlled trial comparing the use of pegIFN versus observation in CML patients attempting TFR. *Materials & Methods:* Adult CML patients from multi-centre in Malaysia with stable DMR for 2 years or more and at least two readings of MR4.5, were stopped TKI and randomized into two arms: (1) subcutaneous pegIFN-α-2a starting at 180μg weekly for a year, followed by observation, or (2) observation. Outcome is relapse, defined as either (i) one reading of loss of major molecular response (>0.1%^{IS}), or (ii) positivity of *BCR-ABL1* transcripts, as confirmed by a second analysis point, indicating the increase (≥1 log) in relation to the first analysis point at two successive assessments. *Results & Discussion:* A total of 30 patients started intervention from July 2015 to October 2018 (pegIFN n=15, observation n=15). Analysis was taken on 13th Mar 2019. A total of 9 patients relapsed (pegIFN n=4, observation n=5). The median time of relapse was 13.1 months (range 9.2 to 25.5) and 1.8 (1.2 to 12.0) after stopping TKI in pegIFN and observation arm, respectively. Dose of tolerable pegIFN was age dependent. Commonest adverse event of pegIFN was transaminitis. Quality of life assessment using EORTC QLO-C30 showed similar result between the two arms. *Conclusion:* PegIFN is a potential consolidative therapy to increase TFR.

HM-28. Prevalence of normal population harbouring *BCR-ABL1* fusion gene in Southern Sarawak, Borneo Island

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Introduction: Level of *BCR-ABL1* fusion gene, the driver mutation in chronic myeloid leukaemia (CML), is monitored using quantitative polymerase chain reaction (PCR) (qPCR) reported in International Scale (IS) to guide disease treatment. *BCR-ABL1* was also found in asymptomatic normal subject without blood/marrow feature of CML. Previous studies used convenient sampling and qualitative PCR or qPCR but not IS to study normal subjects harbouring *BCR-ABL1*. Hence, the result could neither infer to normal population nor impact treatment of CML. *Materials & Methods:* We conducted the first normal population study to determine population prevalence of normal subject harbouring *BCR-ABL1* using qPCR^{IS}. It was a cross sectional community-based study studying southern Sarawak population aged ≥18 and using two-stage sampling (stratified followed by cluster) based on Malaysia Department of Statistics population survey procedure. The sampling frame was divided into enumeration block (EB) and subdivided into living quarter (LQ). qPCR^{IS} *BCR-ABL1* was done using validated commercial kit. *Results & Discussion:* A total of eight EBs, total of 88 LQs and total subject of 190 were studied and analysed. 23 (12.1%) out of 190 samples had poor quality with sum of control gene, *ABL1*, less than 10,000 copy number, while 102 (53.7%) had good quality with sum of *ABL1* more than 100,00. Quality of each run of qPCR^{IS} *BCR-ABL1* was satisfactory fulfilling the evaluation criteria. One subject was found positive, *i.e.* 0.0023%^{IS}. Repeat qPCR^{IS} was 0.0032%^{IS}. Sequencing confirmed e13a2 transcript. *Conclusions:* Prevalence of normal population harbouring *BCR-ABL1* in southern Sarawak was 0.5% to 1%. Sum of control gene *ABL1* copies number in two replicates should be adequate (>100,000) to enable efficient screening.

HM-29. Genomic landscape of *BCR-ABL* kinase domain mutation in chronic myeloid leukemia patients with imatinib resistance

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Introduction: Chronic myeloid leukemia (CML) is a clonal myeloproliferative disorder involving the pluripotent haemopoietic stem cell compartment. It is caused by a reciprocal translocation between chromosomes 9 and 22, t(9;22) (q34;q11) which encodes for the *BCR-ABL* fusion protein. Discovery of imatinib mesylate (IM) as targeted *BCR-ABL* protein kinase inhibitor