

last 3 years, was discontinued in December 2021. In the 2nd and 4th months, the requested bcr-abl was negative. Second case; a 42-year-old male patient was diagnosed with Ph-positive chronic phase CML in 2006. The patient, who had been using imatinib for about 16 years and was bcr-abl negative for the last 3 years, was discontinued in April 2022. bcr-abl came back negative. Third case; a 42-year-old female patient was diagnosed with Ph positive (IS: 31,824) chronic phase CML in 2019. He received Nilotinib and Dasatinib treatment after he had an allergic skin reaction using imatinib for about 2 years. The patient had drug intolerance and was bcr-abl negative for about 2 years, and the treatment was stopped. The 1-month follow-up was negative for bcr-abl. Fourth case; Imatinib was started in a 50-year-old female patient with the diagnosis of Ph-positive chronic phase CML in 2012. Due to imatinib intolerance, the treatment of the patient who was using nilotinib and was bcr-abl negative for the last 3 years was discontinued in February 2021. The bcr-abl negativity continues in the follow-ups. Fifth case: A 58-year-old female patient was diagnosed with Ph-positive chronic phase CML in 2013. The imatinib treatment of the patient, who has been using imatinib for about 8 years and has been negative for bcr-abl for the last 3 years, was discontinued in November 2021. The bcr-abl negativity continues in the follow-ups. **Conclusion:** STIM 1 (Stop Imatinib) study is the first multicenter, non-randomised, prospective study on TKI termination. Then, TWISTER, A-STIM, ENEST, STOP2GTKI, EURO SKI studies were carried out. In these studies, TKI treatment was discontinued in patients who used TKI for at least 3 years and had a major molecular response for the last 2 years. Remission was achieved in approximately 50% of the patients who were followed up without medication. In our study; The treatment of patients who had been using TKI for 5 years and had a major molecular response for about 2 years was stopped. The follow-up of the patients (minimum: 1 month, maximum: 15 months) continues, and all of them are in remission.

<https://doi.org/10.1016/j.htct.2022.09.1237>

PP 03

TREATMENT-FREE REMISSION IN PATIENTS WITH CHRONIC MYELOID LEUKEMIA: MANAGEMENT APPROACHES

Vasile Musteata

State University of Medicine and Pharmacy
“N. Testemitanu”, Institute of Oncology

Objective: The study objective was to analyze the short- and long-term results of treatment discontinuation in patients with chronic myeloid leukemia (CML) and complete molecular response (CMR). **Methodology:** This prospective study enrolled 22 patients (pts) with chronic phase of CML, managed at the Oncologic Institute from Moldova between 2017–2022. The age range was 29–73 years. The male/female ratio was 1:1.2. The real-time quantitative PCR revealed the wide range of BCR-ABL p210 transcript: 21.84–100% IS. In 7 (31.8%) pts the rate of BCR-ABL p210-positive cells was less than 50%.

CMR was achieved in 15 (68.2%) pts after imatinib therapy and in 7 (31.8%) pts after the 2nd generation of TKIs. **Results:** The therapy with TKIs was stopped due to the different reasons in all patients after the CMR was obtained. Two (9.1%) pts stopped the TKIs treatment due to the pregnancy. The molecular relapse occurred in 6 (27.3%) pts, including one pregnant female. All relapsed pts had the initial BCR-ABL p210 transcript expression > 50%. The CMR span ranged between 2.5–26 months in relapsed pts. The range of BCR-ABL p210 transcript in the relapsed pts was 0.002–0.56%. These pts achieved the 2nd CMR after restarting TKIs treatment. All pts are alive, with the ECOG score of 0–1. **Conclusion:** TKIs discontinuation may be considered an option in CML patients with CMR, especially in those with the initially low BCR-ABL p210 transcript expression. The 2nd CMR may be obtained after restarting the TKIs treatment in pts with minor molecular relapse.

<https://doi.org/10.1016/j.htct.2022.09.1238>

COAGULATION DISEASES

PP 04

PHENOTYPE/ GENOTYPE SCREENING PATTERN OF HEMOPHILIA A AND B IN SAUDI ARAB

Tarek Owaidah¹, Salwa Bakr²,
Hala AbaAlkhail¹, Hazza Alzahrani¹,
Mahasen Saleh¹, Abdulrahman Almusa¹,
Nouf Al-Numair³, Haitham Khooger¹,
Faisal Al-Allaf⁴

¹ Department of Pathology, King Faisal Specialist hospital and Research Center, Riyadh, Saudi Arabia

² Department of Clinical Pathology/Hematology, Faculty of Medicine, Fayoum University, Egypt

³ Saudi Human Genome Program, King Abdulaziz City for Science and Technology, Riyadh, Saudi Arabia

⁴ Department of Medical Genetics, Umm Al-Qura University Faculty of Medicine, Makkah, Saudi Arabia

Objective: Hemophilia A and B are X-linked recessive bleeding disorder caused by variants in the factor VIII (FVIII) and factor IX (FIX) genes. There is correlation between the type of mutation and clinical severity of these patients. Establishing national screening program for haemophilia patients is highly encouraged by the World Health Organization (WHO) and World Federation of Haemophilia (WFH). Hence we aimed to establish a genotypic data base for the nature of mutations present in Saudi population. **Case report:** This retrospective descriptive study on a cohort of 136 Saudi hemophilia A and B patients. **Methodology:** Molecular studies were performed to identify known and novel causative variants in hemophilia A and B families and correlated with some clinical features. **Results:** There were 129 male and 7 females with age ranged from 2 - 62 years old, 97 (71.3%) hemophilia A (HA) and 39