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Life has changed for patients with chronic myeloid leukaemia (CML) since the introduction of breakpoint cluster region–Abelson (Bcr-Abl) tyrosine kinase inhibitors (TKIs) in the late 1990s. Before the era of the TKIs began, the 5-year overall survival rates for patients with CML were approximately 28–66%, depending on risk group, chemotherapy regimen and interferon use [1]. In 1996, the first oral TKI was investigated in pre-clinical studies and showed an astonishing reduction of tumour cell formation [2]. This TKI, later named imatinib, was the first to gain US and EU approval for CML in 2001 [3–5] and dramatically increased long-term survival rates to 76–94% at 6 years, depending on risk group [6]. Since then, various generations of Bcr-Abl TKIs have been developed, which induce even higher and faster rates of complete cytogenetic response than first-generation TKIs. To date, Bcr-Abl TKIs provide the basis for successful treatment of the underlying myeloproliferative disease [7].