

reported that MLL/AF9 fusion positive acute lymphoblastic leukemia at diagnosis, may relapse with phenotypic switch and present as secondary acute myeloid leukemia. Similar to what is reported in literature, this case also presented with organomegaly. All the reported cases prior to our case are described in Table 1. As summarized in the table; these cases have been rarely reported in adults and prior to the current case only one such case report exists. The frequent association between the t(9;11) and surface light chain restriction suggests that ALL with this profile is a distinct subset of MLL-positive B-ALL. However, unlike in the previously reported case we were not able to demonstrate a surface immunoglobulin light chain restriction. The previously reported cases showed variable response to treatment. Some patients showed poor prognosis with multiple relapses (like most patients with MLL+). Clinical follow up is very important in these cases along with additional studies to understand leukemogenesis involving common pathways. Clinically, MLL positive precursor B-ALL is typically associated with a poor prognosis due to recurrent relapses and the patients are symptomatic to begin with. Interestingly t(9;11) is rarely been reported in adults. There is only one case report of B-ALL with t(9;11) in a 67 year old female and the patient had poor clinical outcome. Hypothetically, there may not be many reports on t(9;11) positive ALL in adults because either the patients would have succumbed due to aggressive disease before cytogenetics could be done or such patients are not reported at all. In conclusion, to the best of our knowledge, this is the only case report of t(9;11) positive B-ALL in an elderly female from the India. It is very important to report and follow such cases to understand additional mechanism involved and affecting the clinical behavior of such cases.

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