Report of chronic myeloid leukemia from All India Institute of Medical Sciences, 1990-2010

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ABSTRACT
All India Institute of Medical Sciences, New Delhi is an Apex Institute and caters to more than 1.5 million out-patients and 80,000 in-patients every year. In this study, we have presented data of patients over a period of 20 years. This encompasses our initial experience with hydroxyurea, then interferon alone or with cytarabine and finally imatinib. We have presented data of 525 patients treated on imatinib alone. Imatinib dose was increased in 56 (10.6%) patients and regain of complete hematological response was seen in 26 patients. A total of 14 patients were transplanted for different indications of chronic myeloid leukemia and out of which 12 patients are doing well, while two died due to Grade IV gut graft-versus-host disease.

Key words: Chronic myeloid leukemia, All India Institute of Medical Sciences, Imatinib

INTRODUCTION
Chronic myeloid leukemia (CML) has seen one of the few success stories of cancer therapy today. One of the authors (PM) saw the arrival of imatinib as a research officer and we have seen it over the time grow into one of the wonder drugs of cancer therapy. We have now attended endless debates and programs on the drug and its newer rivals. We have seen extravagant optimism at its launch, heard the cynics grow louder sometime later (resistance! just you wait and see) and finally a more informed acceptance of the drug and its limitations. Today, there is no denying its place in medical history. We share our own experiences regards the diagnosis and management of CML at our institute over a span of 20 years.

OUR EXPERIENCE WITH CML
We have a separate out-patients department (OPD) register for patients with CML. We had on record 1105 patients as on June 2010. This included all new as well as older patients on follow-up. The Max foundation patient assistance program alone supported 987 patients of whom 639 (64%) patients were in active follow-up with the foundation. Most of the other patients are on generic brands supported by their employers or their own funds. The Max foundation offers 11 months of free treatment for some patients. As per our records only one patient has availed of this scheme. All patients still find it economically viable to take generic brands rather than buy 1 month supply of Glivec. We have not studied all 1105 patients together in detail. Our data is still piecemeal. Thus, we have initial data on patients during the time when almost all patients were on hydroxyurea. Then we have some data on our experience with interferon. The interferon study stopped accrual with the advent of generic imatinib, which was considerably cheaper than Glivec. We supplanted imatinib in the interferon trial initially and have some data on imatinib with cytarabine. We took the invitation by TMH (Tata Memorial Hospital, Mumbai) to present our data as an incentive to put some of our data in order. Over the last few months we collected basic data, which we could analyze quickly in time for the meeting. We could gather data on 525 patients (restricting ourselves to only those who had at least 6 months of follow-up and excluded repeat follow-ups during this time). In addition, we have also presented our experience with adult CML in children, CML in pregnancy and transplant.

Some of the readers might remember the interferon trial funded by Fulford, which studied interferon alpha for CML. At the time imatinib was in phase II trials. The job of one of the authors (PM) was to recruit patients for the project, randomize them to two arms – one receiving interferon alone and the other in combination with cytarabine. A PhD student in anatomy performed cytogenetic studies at the time. Prof. VP Choudhry (then head of the department) had analyzed data on patients presenting with CML prior

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to starting the interferon project. He had compared the presenting features of patients presenting with CML as compared with a North American population [Table 1]. All these patients had been primarily managed with hydroxyurea alone.

We had complete data on 73 patients enrolled in the interferon study. Baseline data and results are tabulated in Tables 2 and 3.

There was no significant difference in toxicities between the two arms (Interferon alone vs. Interferon –cytarabine). The common side-effects were fever (24%), body ache (40%) and loss of appetite (34%), neutropenia (30%). However, all patients could not tolerate doses greater than 3 MIU thrice a week. Two patients developed psychosis and two patients had renal failure requiring dialysis. There was no added toxicity to the addition of cytarabine. However, the cytogenetic responses in this group were very disappointing.

At around this time, Glivec was introduced in the market. The original brand was and still is beyond the reach of most patients in India. With the advent of generic forms, interferon quickly fell out of fashion. The interferon project was fast losing patients who crossed over to the generic forms. We then continued our study, this time substituting imatinib for interferon.

Our job of enrolling patients on imatinib was aided in no small measure by the Max foundation, which freely provided imatinib (Glivec) to almost all patients excepting those covered by the employer’s (or government service) medical cover. Professor Saxena had already established the molecular laboratory with Dr Sudha Sazawal. The PhD student had completed her theses by this time and there was no one else doing cytogenetics in the hospital. Therefore, we decided to make do with what we had and decided to monitor these patients with quantitative polymerase chain reaction (PCR). The test was not standardized at the time and we kept samples at 3 monthly intervals. We enrolled 85 patients in this study.

Only those patients in chronic phase with no coexisting morbidities or pregnancy were included in the study. All patients were started on imatinib within 6 months of diagnosis. Hydroxyurea was the only treatment allowed prior to starting imatinib for inclusion in the study. Imatinib was started in 40 patients at the standard recommended dose of 400 mg/day. Cytarabine was added in 45 patients at a dose of 10 mg/m² for 10 days every month, after 3 months of imatinib. We had problems with quantifying the molecular response at the time and out of the 85 patients enrolled in the study, we were able to perform the real

Table 1: Patient characteristics of patients presenting with CML in comparison with West data

<table>
<thead>
<tr>
<th>Variables</th>
<th>AIIMS (N=300)</th>
<th>MD Anderson (N=494)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age</td>
<td>35</td>
<td>50</td>
</tr>
<tr>
<td>Male:female</td>
<td>1.6:1</td>
<td>1.3:1.41</td>
</tr>
<tr>
<td>Presenting symptoms (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>&lt;5%</td>
<td>20-50%</td>
</tr>
<tr>
<td>Fatigue</td>
<td>66.7</td>
<td>83</td>
</tr>
<tr>
<td>Weight loss</td>
<td>32.7</td>
<td>61</td>
</tr>
<tr>
<td>Abdominal fullness</td>
<td>43.5</td>
<td>38</td>
</tr>
<tr>
<td>Bruising</td>
<td>12</td>
<td>35</td>
</tr>
<tr>
<td>Fever</td>
<td>60</td>
<td>11</td>
</tr>
<tr>
<td>Examination findings (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>94.3</td>
<td>54</td>
</tr>
<tr>
<td>Massive splenomegaly</td>
<td>33</td>
<td>2</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>93</td>
<td>18</td>
</tr>
<tr>
<td>Lymphadenopathy</td>
<td>12</td>
<td>38</td>
</tr>
<tr>
<td>Purpura</td>
<td>7</td>
<td>27</td>
</tr>
<tr>
<td>Sternal tenderness</td>
<td>2</td>
<td>78</td>
</tr>
<tr>
<td>Good Sokal score</td>
<td>1%</td>
<td>52%</td>
</tr>
<tr>
<td>High risk Sokal score</td>
<td>40%</td>
<td>22.8%</td>
</tr>
</tbody>
</table>

CML – Chronic myeloid leukemia; AIIMS – All India Institute of Medical Sciences

Table 2: Patient characteristics in the interferon trial

<table>
<thead>
<tr>
<th>Variables</th>
<th>Interferon</th>
<th>Interferon plus cytarabine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>38</td>
<td>35</td>
</tr>
<tr>
<td>Males</td>
<td>29</td>
<td>20</td>
</tr>
<tr>
<td>Median age (range)</td>
<td>42 (16-60 years)</td>
<td>40 (30-56 years)</td>
</tr>
<tr>
<td>Median duration before interferon treatment (months)</td>
<td>8</td>
<td>12 months (range 4-14 months)</td>
</tr>
<tr>
<td>Mean Hb g/dl</td>
<td>11.2</td>
<td>11.4</td>
</tr>
<tr>
<td>Mean TLC in mm³</td>
<td>65000</td>
<td>75000</td>
</tr>
<tr>
<td>Mean blasts at diagnosis %</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Hb – Hemoglobin; TLC – Total leukocyte count

Table 3: Treatment results

<table>
<thead>
<tr>
<th>Variables</th>
<th>Interferon</th>
<th>Interferon plus cytarabine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median duration of treatment</td>
<td>24 months</td>
<td>18 months</td>
</tr>
<tr>
<td>Median follow-up (range)</td>
<td>24 (1-56 months)</td>
<td>25 (4-54 months)</td>
</tr>
<tr>
<td>Complete cytogenetic responders</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Partial cytogenetic responders</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Minor cytogenetic response</td>
<td>20</td>
<td>21</td>
</tr>
<tr>
<td>No cytogenetic response</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>Blast crisis</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td>Median duration to blast crisis</td>
<td>36 months</td>
<td>34 months</td>
</tr>
</tbody>
</table>

PCR for BCR-ABL at baseline and follow-up in 56 patients. 36 patients had received imatinib and 20 patients had received cytarabine in addition to imatinib. The results are tabulated in Tables 4-7.
Mishra, et al.: CML data from AIIMS

### Table 4: Baseline data in the AIIMS imatinib study

<table>
<thead>
<tr>
<th>Variables</th>
<th>Imatinib only</th>
<th>Imatinib-cytarabine combination</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. of patients</td>
<td>Median (range)</td>
</tr>
<tr>
<td>Total number</td>
<td>40</td>
<td>45</td>
</tr>
<tr>
<td>Age</td>
<td>32 (15-45)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>Sokal risk group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Intermediate</td>
<td>8</td>
<td>15</td>
</tr>
<tr>
<td>Low</td>
<td>24</td>
<td>18</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>40</td>
<td>11 (5.6-15.4)</td>
</tr>
<tr>
<td>TLC count (×10⁹)</td>
<td>40</td>
<td>139 (112-245)</td>
</tr>
<tr>
<td>Platelet count (×10⁹)</td>
<td>40</td>
<td>589 (125-1126)</td>
</tr>
</tbody>
</table>

TLC – Total leukocyte count; AIIMS – All India Institute of Medical Sciences

### Table 5: Hematological toxicities

<table>
<thead>
<tr>
<th>Toxicity</th>
<th>Imatinib–only group</th>
<th>Combination therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=40</td>
<td>n=45</td>
</tr>
<tr>
<td>Anaemia</td>
<td>5 (12.5)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Neutropenia</td>
<td>10 (25)</td>
<td>14 (31)</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>15 (37.5)</td>
<td>14 (31)</td>
</tr>
</tbody>
</table>

### Table 6: Non-hematological toxicities

<table>
<thead>
<tr>
<th>Toxicity</th>
<th>Imatinib–only group</th>
<th>Combination therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=40 n (%)</td>
<td>N=45 n (%)</td>
</tr>
<tr>
<td>Nausea</td>
<td>16 (40)</td>
<td>15 (33)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>5 (12.5)</td>
<td>3 (6.6)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>7 (17.5)</td>
<td>7 (15.4)</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>7 (17.5)</td>
<td>8 (17.6)</td>
</tr>
<tr>
<td>Fever</td>
<td>5 (12.5)</td>
<td>9 (19.8)</td>
</tr>
<tr>
<td>Cramps</td>
<td>20 (50)</td>
<td>18 (39.6)</td>
</tr>
<tr>
<td>Anorexia</td>
<td>3 (7.5)</td>
<td>4 (8.8)</td>
</tr>
<tr>
<td>Edema/weight gain</td>
<td>4 (10)</td>
<td>4 (8.8)</td>
</tr>
<tr>
<td>Headache</td>
<td>2 (5)</td>
<td>3 (6.6)</td>
</tr>
<tr>
<td>Hyperpigmentation</td>
<td>12 (30)</td>
<td>8 (17.6)</td>
</tr>
<tr>
<td>Nasal catarrh</td>
<td>2 (5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Skin rash</td>
<td>6 (15)</td>
<td>5 (11)</td>
</tr>
<tr>
<td>Aphthous ulcers</td>
<td>3 (7.5)</td>
<td>4 (8.8)</td>
</tr>
<tr>
<td>Loss of sweating</td>
<td>4 (10)</td>
<td>5 (11)</td>
</tr>
<tr>
<td>Gynecomastia</td>
<td>2 (5)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Intracranial bleed</td>
<td>0 (0)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Progression to blast crises</td>
<td>1 (2.5)</td>
<td>3 (6.6)</td>
</tr>
<tr>
<td>Causes for permanent interruption</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin rash</td>
<td>1 (2.5)</td>
<td>2 (4.4)</td>
</tr>
<tr>
<td>Severe cramps</td>
<td>1 (2.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Fever</td>
<td>0 (0)</td>
<td>1 (2.2)</td>
</tr>
</tbody>
</table>

This initial study showed that all patients achieved complete hematological response within a period of 1-3 months after starting imatinib. None of these patients lost their hematological responses at the end of the 3 years study period. We had several problems during the molecular analyses. We had just started out with the molecular laboratory and it was some time before we could get reliable results. The test was not standardized and our inexperience with the test at the time was one of the major limitations of our study and could be the reason for the seemingly poor molecular response. All patients analyzed were newly diagnosed patients who received Imatinib soon after diagnosis. Subsequently the laboratory got around to giving results in the more commonly used BCR-ABL/ABL ratio format. Even so we felt that the best way about would be to standardize our results with one of the IRIS laboratories and we are currently working on collaborating with the laboratory in Adelaide. We have also understood the importance of maintaining a minimum cutoff level for the endogenous copy number i.e. ABL for purposes of reporting on BCR-ABL/ABL ratio, particularly for samples which are negative on imatinib therapy (<10000 ABL copy numbers to reject a report in our lab).

One of the major blocks in getting patients to do cytogenetic/molecular response analyses is that most of our patients at All India Institute of Medical Sciences (AIIMS) do not have any option beyond imatinib owing to financial reasons. Those who can afford and are motivated do agree for regular analyses at 6 monthly intervals and this has helped us to increase the dose of imatinib for those whose BCR-ABL levels showed a rising trend.

### Our data on imatinib in CML (Till 2010)

Some of the 525 patients who are currently been analyzed have been included in the previous data already presented above. Therefore, the baseline data is not very different from those already presented in Tables 1 and 4 [patients enrolled in the interferon study had a higher median age in the and lower baseline total leukocyte count levels -Table 2]. We did not again analyze the hematological and non-hematological toxicities of imatinib, which were already analyzed in the imatinib/ imatinib-cytarabine combination study [Tables 5 and 6]. Thus, this data is more of a follow-up data of those patients who are currently attending the OPD [Table 8].
We do not currently have data on those patients who died as this data is OPD based. We did not actively persuade any patient to undergo analysis to look for molecular or cytogenetic response considering the fact that most of these patients do not have recourse to any other treatment option and molecular analyses even at AIIMS costs Rs. 3,000. We did explain the importance of such this test to each patient. Of these 525 patients 123 patients underwent molecular or cytogenetic testing at least once during follow-up [Table 9]. We generally advised such tests after at least 6-12 months of follow-up.

Imatinib dose was increased in 56 of 525 patients. The reason for dose increase was a loss in hematological response in 25, inadequate molecular response in 18 and accelerated phase in 13 patients. Of these 56 patients an increase in dose helped regain complete hematological response in 26 patients.

**CML in children and pregnancy**

We analyzed 41 cases of adult CML in childhood, seen over a period of 10 years from 1994 to 2004 in our department. The disease was usually seen in children older than 12 years (87.8%) (age range: 2-16 years). Males predominated in this group (27 vs. 14). Most of the children presented in the chronic phase (85%). Using the Sokal score based on age, spleen, platelets and blast count 80%, 14.6% and 29.2% were in the low, intermediate and high-risk groups respectively. Our patients usually presented with fever, abdominal mass/pain and anorexia. Though, hepatosplenomegaly was common, lymphadenopathy was rare (one patient). The median survival was 13 months (range: 2-144 months). The b2a2 transcripts were seen more commonly in our pediatric population.[3] We had one interesting case of Philadelphia positive thrombocytosis in a girl, 9 years old at the time who required both hydroxyurea and imatinib initially to correct her counts. Imatinib was able to bring her BCR-ABL levels down, but somehow did not affect her platelet counts.[4] She is 6 years into follow-up and is on imatinib alone. Her BCR-ABL levels are undetectable. She does not have a matched sibling.

One of the authors (PM) had presented our data on CML in pregnancy at the last meeting hosted by TMH. We now have 10 patients who became pregnant during the course of their disease. All became pregnant while on imatinib. We had followed the manufacturer advice and asked them to stop the medication. Only three complied with the instruction. The rest had uneventful outcomes, except for one whose child had a meningocele.[3,4]

**Tolerability and primary resistance**

Imatinib has largely been a safe and well tolerated drug except for occasional cases.[3] We had one patient who attempted suicide by consuming a large number of Imatinib tablets, but failed miserably.[8] We have three patients on record who did not tolerate even 200 mg of imatinib and one of whom is now on interferon. Three other patients are on 300 mg of imatinib. Of these six patients on a suboptimal dose only one is in complete hematological

### Table 7: Real time PCR results

<table>
<thead>
<tr>
<th>Variables</th>
<th>Imatinib alone</th>
<th>Combination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median number of BCR-ABL transcripts at diagnosis</td>
<td>345808 (605-10125616)</td>
<td>332498 (2712-12112832)</td>
</tr>
<tr>
<td>Median number of BCR-ABL transcripts at follow-up</td>
<td>18286 (0-5412661)</td>
<td>15415 (4-4328466)</td>
</tr>
<tr>
<td>Median follow-up</td>
<td>1.5 (0.6-1.8)</td>
<td>1.8 (0.6-2)</td>
</tr>
<tr>
<td>Median log reduction</td>
<td>1.255 (0.975-4.02)</td>
<td>1.305 (0.12-8.2)</td>
</tr>
<tr>
<td>No. of patients with ≥3-log reduction in BCR-ABL transcripts</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>No. of patients with no transcript at end of study</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

PCR – Polymerase chain reaction

### Table 8: Follow-up data on 525 patients currently on follow-up in hematology OPD as on June 2010

<table>
<thead>
<tr>
<th>Duration of follow-up</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median 24 months</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(range 6-120 months)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dose</td>
<td></td>
<td></td>
</tr>
<tr>
<td>600 mg</td>
<td>41</td>
<td>7.8</td>
</tr>
<tr>
<td>800 mg</td>
<td>15</td>
<td>2.8</td>
</tr>
<tr>
<td>300 mg</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>200 mg</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Patients on sprycel</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Interferon</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Trial drug (NATCO)</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Current disease status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Accelerated phase</td>
<td>30</td>
<td>5.7</td>
</tr>
<tr>
<td>Blast crisis</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Loss of complete hematological response</td>
<td>48</td>
<td>9.1</td>
</tr>
</tbody>
</table>

### Table 9: Molecular/cytogenetic analyses for 123 patients (out of 525 patients)

<table>
<thead>
<tr>
<th>Log reduction</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2 log reduction</td>
<td>20</td>
<td>16.2</td>
</tr>
<tr>
<td>2-3 log reduction</td>
<td>25</td>
<td>20.3</td>
</tr>
<tr>
<td>&gt;3 log reduction</td>
<td>35</td>
<td>28.4</td>
</tr>
<tr>
<td>Complete molecular response/FISH negative</td>
<td>20</td>
<td>16.2</td>
</tr>
<tr>
<td>&lt;1 log reduction</td>
<td>23</td>
<td>18.6</td>
</tr>
</tbody>
</table>
response. We have had primary resistance in two patients till date. We found a matched sibling for one of them and he was transplanted. He had a T315I mutation to start with. After transplant his BCR-ABL levels showed a rising trend after 3 months. He was planned for donor lymphocyte infusion (DLI) but has extensive skin graft-versus-host disease (GVHD) and also liver involvement. He was negative for the T315I mutation after transplant and he is currently on Dasatinib.

Transplant in CML
We have transplanted 14 patients with CML since 2004 till 2010. Eight patients were in accelerated or blast phase at the time of transplant. One patient was primary refractory to imatinib. Six patients were in chronic phase showing loss of molecular response and progressively rising transcript levels even after increasing the dose of imatinib in three patients. Three of these six patients elected to have a transplant instead of increasing their dose. We lost two of these patients, both to grade IV gut GVHD. Two of these 14 patients were children one of whom; a 3-year-old was initially managed for a lymphoid blast crisis. Four of these 14 patients lost their molecular response and two opted for imatinib alone and subsequently went into complete molecular response. One of the other two patients is the primary refractory patient described above and we are planning a DLI for the latest patient who has lost molecular response. A small study from our department seemed to suggest that patients transplanted earlier did better than delaying till they progressed on imatinib. However these were patients who had not received second line tyrosine kinase inhibitors and had been transplanted in accelerated phase.

We hope to standardize our laboratory results for molecular monitoring. We are also currently part of a drug trial sponsored by NATCO who have developed an indigenous tyrosine kinase inhibitor. We are steadily building up some data on patients on Dasatinib. All patients on Dasatinib are currently government employees whose health expenditures are completely covered.

**ACKNOWLEDGMENT**

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**REFERENCES**


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