A 64 year old diabetic female develops CML and receives imatinib treatment develops aggressive bilateral neovascular glaucoma within 1 month of initiation of treatment. The left eye is lost and the right eye was hardly salvaged through panretinal photocoagulation and substitution of imatinib therapy to desatinib therapy. Systemic imatinib therapy for CML.

Imatinib may be implicated in the causation of NVG in CML patients, who should thus receive regular thorough ophthalmic evaluation as long as imatinib therapy continues.
Dasatinib (Bristol Myers Squibb) tablets, 100 mg daily. Monthly CBCs were normal thereafter.

Discussion

The ophthalmic manifestations of CML are quite variable and include intraretinal hemorrhages, Roth spots, nerve fiber layer infarcts, subhyaloid and vitreous haemorrhages and papilloedema secondary to raised intracranial pressure [1]. There are various reports on the ocular side effects of imatinib therapy, most reporting periorbital oedema [2], besides others including epiphora, recurrent subconjunctival hemorrhage and optic neuritis. There are variable reports about glaucoma developing in CML patients with imatinib therapy [3,4], even the drug information leaflet points this out as a rare possibility. However, the reports are inconsistent and do not demonstrate the exact type of glaucoma. In our case, the type of glaucoma was neovascular, possibly due to retinal ischemia. We recognise that our patient—being originally diabetic—was already prone to develop glaucoma, especially neovascular glaucoma (as a result of retinal ischemia), yet the imatinib therapy may have aggravated the condition. We hypothesize that the very high WBC and PLT counts induced a hyperviscosity state that initiated or aggravated retinal ischemia. In such a situation, the imatinib therapy may actually have protected the remaining eye from visual loss by improving the haematalogic parameters of the patient. Alternatively, imatinib may have aggravated retinal ischemia, or induced neovasculariation by another mechanism as recently reported by Gulati and Saif [5]. Wherever the true situation is regarding the relationship between CML, Imatinib and vision loss, it is worth noting that CML patients on Imatinib therapy should have regular thorough ophthalmic evaluations, especially if diabetic. Better still, it may be advisable to avoid imatinib therapy in a patient with a known risk factor for glaucoma, and possibly resort to a safer drug, like nilotinib, for which no ocular side effects are reported so far.

References


Table 1: Lab parameters before and 1 week and 3 months after initiation of therapy.

<table>
<thead>
<tr>
<th>Lab parameter on presentation</th>
<th>Lab parameters 1 week later</th>
<th>Lab parameters 3 months later</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>122×10^9/L</td>
<td>70.6×10^9/L</td>
</tr>
<tr>
<td>PLT</td>
<td>1,254×10^9/L</td>
<td>762×10^9/L</td>
</tr>
<tr>
<td>Hb</td>
<td>11.1 g/dL</td>
<td>11 g/dL</td>
</tr>
<tr>
<td>Differential WBC count</td>
<td>Shift to the left with absolute basophilia</td>
<td>Qualitative PCR analysis for bcr-abl fusion gene</td>
</tr>
<tr>
<td>Liver &amp; kidney functions</td>
<td>Normal</td>
<td>JAK2V617F mutation</td>
</tr>
<tr>
<td>Serum uric acid</td>
<td>6.4 mg/dL</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Ophthalmologic examination on presentation.

<table>
<thead>
<tr>
<th>OD</th>
<th>OS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual acuity</td>
<td>Counting fingers (CF) at 10 cm</td>
</tr>
<tr>
<td>IOP (mmHg)</td>
<td>26</td>
</tr>
<tr>
<td>Cornea</td>
<td>Clear cornea</td>
</tr>
<tr>
<td>Anterior Chamber</td>
<td>Normal depth and content</td>
</tr>
<tr>
<td>Iris</td>
<td>Extensive rubeosis iridis and posterior synchia</td>
</tr>
<tr>
<td>Gonioscopy</td>
<td>360 degrees of peripheral anterior synchia (PAS) with neovessels in the angle</td>
</tr>
<tr>
<td>Fundus</td>
<td>Totally cupped optic disc with neovascularization of the disc (NVD)</td>
</tr>
<tr>
<td>Remarks</td>
<td>Superonasal scarred flat bleb</td>
</tr>
</tbody>
</table>

Figure 1: (A,B) Rubeosis iridis and posterior synechia. (C,D) Peripheral Anterior Synechia (PAS) (gonioscopic view).

(Dasatinib, Bristol Myers Squibb) tablets, 100 mg daily. Monthly CBCs were normal thereafter.