stimulatory investigations revealed persistently elevated immunoreactive GH levels. After glucose load, GH was adequately suppressed. It would have been ideal to measure IGF-1 levels but due to nonavailability of this assay here, it could not be done. A similar case of GH insensitivity syndrome was reported earlier(2). GH insensitivity syndrome encompasses a range of etiologies one of which is GH receptor deficiency (GHRD). The reported patients, now exceeding 150, with GHRD are predominantly of Mediterranean origin(3). A remarkable concentration of GHRD was reported from an inbred Caucasian population in southern Ecuador predominantly affecting females(4). Elucidation of molecular mechanisms responsible for Laron syndrome has been rapid, and promises to be helpful in genetic counseling of the affected families.

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Multifocal Chloromas Preceding Acute Myelogenous Leukemia

We report a rare case of multifocal chloromas preceding acute myelogenous leukemia (AML) by a period of three months, the shortest time interval reported so far in the literature. A ten year old boy presented with low back ache since two months followed by progressive weakness of the lower limbs, hypersthesias and urinary incontinence. On clinical examination, the findings

were suggestive of upper motor neuron type of lesion localized to D7 vertebra. The routine laboratory investigations were normal. Peripheral blood smear was unremarkable. Plain skiagrams of the dorsal spine, chest and the skull were normal. A CT myelogram was done which showed a complete block by an extradural soft tissue mass *(Fig. 1)*. Three weeks later the patient noticed gradually increasing bilateral proptosis. CT scan of the orbits showed a retrobulbar soft tissue mass in the left orbit *(Fig. 2)* with bilateral enlarged extraocular muscles. CECT of the brain done at the same time revealed bilateral extra-axial masses *(Fig.* 3). Perhpieral blood smear at this stage was again unremarkable. FNAC of the left retrobulbar mass revealed blast cells followed by bone marrow study which showed 20% myeloblasts.

Chloromas are observed in only 1-9% patients with AML(l); the first case was described by King in 1853(2) and is commonly seen in younger patients. These can involve any site and can occur coincidental, antedate or follow the development of AML by months to years(4). The common sites of involvement are orbits, paranasal sinuses, spine, thorax, thyroid, breast, bowel and pelvic organs(5). The etiopathogenesis of chloromas is not yet clear, however, it is suggested that chloromas represented multifocal foci of extramedullary malignant granulopoiesis and show cells more immature than those in peripheral blood and are quite frequently associated with the absence of blast cells in the peripheral smear(l). The usual time interval between the development of chloroma and AML varies between 5-16 months; to our knowledge this is the

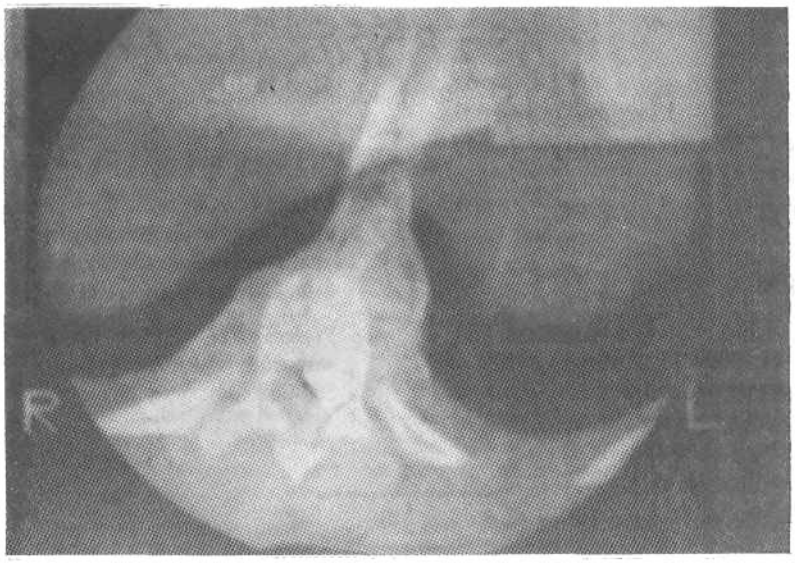

Fig. 1. CT myelogram of dorsal spine showing extradural mass with compression of cord.

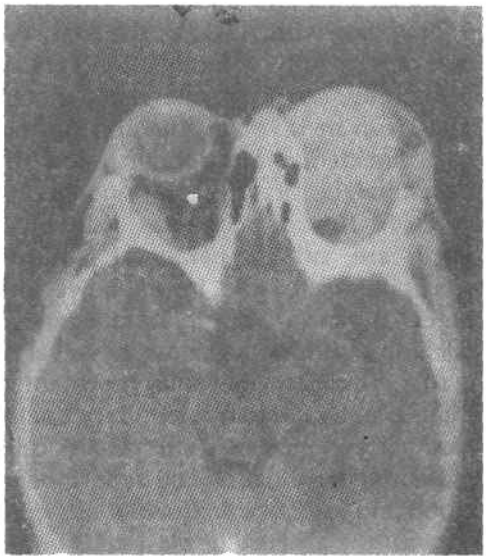

Fig. 2. Enhanced CT showing retrobulbar mass with proptosis left side.

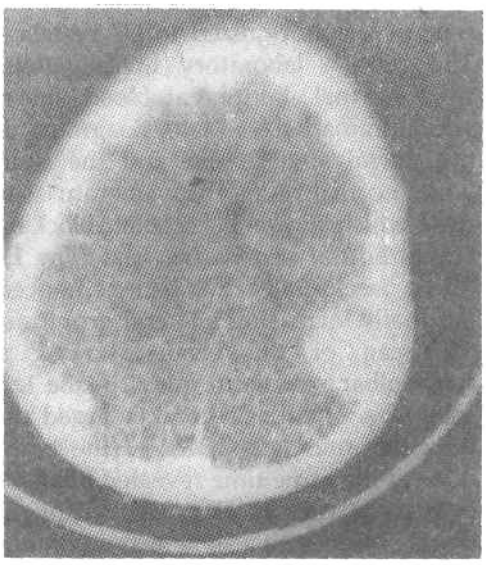

Fig. 3. Bilateral extra-axial masses seen on contrast enhanced CT.

first case with the chloroma preceding the development of AML by only three month period.

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