photosensitivity/photo toxicity. The photo-onycholysis thus seen is often painful and is usually associated with photodermatitis.

Damage to the supporting structures, disruption of the cementing substance binding the nail plate to the surrounding structures or formation of granular layer, which is otherwise absent in a normal nail bed and nail matrix, will produce onycholysis.\textsuperscript{[1]} Trauma from physical factors, manicuring, foreign body implantation or chemicals like solvents, various nail cosmetics and immersion in alkaline soap water etc. will cause onycholysis in occupation-related cases. Psoriasis, lichen planus, eczemas, pemphigus, Darier’s disease, leprosy, scleroderma, bacterial, fungal or viral infections and congenital ectodermal defects are some of the dermatological diseases associated with onycholysis. It may also be secondary to general medical conditions like hypo- or hyperthyroidism, porphyrias, syphilis, peripheral ischemia, bronchiectasis, anemia and pregnancy. Drugs like tetracyclines, psoralens, fluoroquinolones and phenothiazines may produce photoonycholysis while adriamycin, bleomycin, 5 – fluorouracil, cephaloridine and cloxacillin cause onycholysis through toxicity to nail matrix or nail bed. Benign or malignant tumors underneath the nail plate may also present clinically with onycholysis. However, most cases of onycholysis remain idiopathic.

Roxithromycin, a new semisynthetic, long acting, acid stable macrolide has antibacterial spectrum similar to that of erythromycin. It has a low potential for hepatotoxicity or gastrointestinal side effect unlike older macrolides. Transient deafness, allergic reaction are seen uncommonly and association with pancreatitis is questionable.\textsuperscript{[5,6]} It is not reported to have any photosensitizing potential. Its side effects are either immune mediated or occur due to primary parenchymal toxicity.

The exact pathogenesis of onycholysis in our patient due to roxithromycin is unclear. Absence of skin photosensitivity and nail tenderness excludes photo-onycholysis. It appears to be due to toxicity to nail bed/ nail matrix similar to what is described for cephaloridine and cloxacillin.\textsuperscript{[1]} Excessive concentration of roxithromycin under the free edge of nail due to its prolonged intake perhaps resulted in direct parenchymal toxicity to nail matrix/nail bed manifesting clinically as onycholysis. In spontaneous onycholysis patients, an adverse reaction to systemic drug must be suspected.

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Hydroxyurea induced non-healing leg ulcer

Sir,

Hydroxyurea is a commonly used chemotherapeutic agent in chronic myeloid leukemia in India. Rarely its long term use can lead to leg ulceration. Such an ulcer generally does not respond to the usual measures but heals when hydroxyurea is stopped.

A 40 year old man, case of chronic myeloid leukemia (CML) on therapy with hydroxyurea for over two years, presented with a non-healing ulcer on the right foot near the lateral malleolus. There was no history of trauma at the site of the ulcer. He was not a diabetic and there was no family history of diabetes.

Letters to Editor
Two years back the patient had presented with weakness and fatigue of two months duration. At that time, splenomegaly was detected; the spleen was palpable six centimeters below the costal margin in the left midclavicular line. The liver was palpable two centimeters below the costal margin in the right midclavicular line. Systemic examination was otherwise normal.

On investigation the hemoglobin was 9 g%, the total leukocyte count was 66,000/cmm, and the differential count was: promyelocytes 10%, metamyelocytes 8%, myelocytes 12%, basophils 5%, eosinophils 4%, neutrophils 41%, and lymphocytes 20%. Bone marrow examination was suggestive of chronic myeloid leukemia in the chronic phase. Cytogenetics was positive for Philadelphia chromosome in all the metaphases grown from the bone marrow cells. Hepatomegaly and splenomegaly were seen on ultrasound examination of the abdomen. X-ray chest, ECG, and serum biochemistry were normal.

The patient was diagnosed as a case of chronic myeloid leukemia (CML) and was started on hydroxyurea. During this period, the dose of hydroxyurea varied from 2 g/day to no drug at all depending on whether the white cell count was less than 5000/cumm.

This ulcer gradually enlarged to 4x3 centimeters with a necrotic base and was associated with pain. There was no purulent discharge. The patient was started on an oral antibiotic and local dressing was done regularly. The ulcer did not respond to these measures. An X-ray of the left ankle and culture of a swab from the ulcer were normal. He also received injection GM-CSF locally with no response.

In view of the resistance to various therapies a hydroxyurea induced ulcer was suspected. Hydroxyurea was stopped and the ulcer started healing within twenty days. The patient was then started on busulphan for CML.

Hydroxyurea is a chemotherapeutic agent that inhibits ribonucleotide reductase, an enzyme essential for DNA synthesis. Skin abnormalities have been commonly associated with its long term use, but leg ulceration has been only occasionally reported. The average age of the patient in all series has been above 60 years, suggesting that older patients might be at increased risk of this side effect. The average duration of hydroxyurea intake before developing leg ulceration has been five years, six years and two years six months in different series, but the range in one series was 10-55 months. These ulcers are typically located on the malleolar and/or perimalleolar area,[12] and are usually painful. In one series multiple ulcers were seen in 64% of cases.[2]

The mechanism for ulcer formation is not known. It has been hypothesized that hydroxyurea, being an antineoplastic agent, is more toxic to the actively dividing cells like regrowing edge of a skin ulcer. This leads to ulceration by cutaneous atrophy and impaired wound healing in areas subjected to common trauma. It is well known that hydroxyurea causes a megaloblastic picture in the peripheral blood. It has also been hypothesized that megaloblastic erythrocytes may circulate poorly through the capillary network, leading to decreased oxygenation and impaired healing.[1]

On histopathological examination perivascular lymphocytic infiltration, leukocytoclastic vasculitis, thrombus formation, swelling of endothelial cells and thickening of vascular wall have been observed.[3]

 Withdrawal of hydroxyurea has been consistently associated with healing,[3] and recurrence has been reported when hydroxyurea is reintroduced. Successful treatment of the ulcer without discontinuation of hydroxyurea has also been reported. Granulocyte macrophage colony stimulating factor, prostaglandin E1, pentoxifylline, local wound care and surgical grafting have been used.[3-5]

Hydroxyurea has also been used in psoriasis and the response rate is as high as 70%.[6,7] There are a few reports of hydroxyurea causing leg ulceration when it is used in psoriasis for a prolonged period.[8]

In this case illustrates that hydroxyurea should always be considered as one of the causes for a leg ulcer in a patient on hydroxyurea.
Sixty CSWs working around Raipur were counseled by a medical officer and investigated for the presence of HIV, HbsAg and VDRL test. Surface antigen for hepatitis B (HbsAg) was determined by latex agglutination method while antibodies to HIV were detected by dot immuno-assay for the detection of antibodies to HIV 1 and 2.

The age of the CSWs ranged from 19 – 44 years (mean 29.75 ± 6.71 years). About 88% of CSWs preferred to work during 8 pm-12 pm while 12% were working during the day. The average duration of sexual exposure was 5.86 ± 2.32 years. Fourteen (23.3%) CSW’s showed positive VDRL test while the seroprevalence of HIV and HbsAg was 5% and 8.33% respectively (Table 1).

In 1988, the seroprevalence of HIV amongst CSWs was so low that only one HIV seropositive CSW was found out of 701 CSWs examined[4] but now it is well documented from all parts of the world that CSWs have a very high incidence of HIV and HBV infection. Thus they are a major reservoir and source of transmission. The HIV prevalence in CSWs in some of the big cities in India has ranged from 0.5% to 69%. The prevalence rate of 5% of HIV positivity at Raipur is quite high. This may be because the major business connection of Raipur is Mumbai. Again, this indicates that a large scale epidemic is now looming around in this region and this requires urgent educational measures to be taken for all including the CSWs.

## HIV and HbsAg seroprevalence in commercial sex workers in Raipur (Chhattisgarh) area

Sir,

Heterosexual intercourse is the dominant mode of HIV transmission in most parts of the world. Commercial sex workers (CSWs) are associated with high incidence of STDs. HIV seroprevalence in CSWs in Mumbai had crossed 69% in 1995.[1] HIV and hepatitis B virus (HBV) share a common mode of transmission i.e. sexual, parenteral and perinatal. In India, about 4.7% of adult population was found to be infected with HBV. In STD clinic attendees in Mumbai, prevalence rate was 8.8%[2] while in Tirupati, HbsAg seroprevalence in female CSWs was 24% and in STD patients it was 13.6%.[3]

### REFERENCES