Idiopathic Oesophageal Ulcer in A HIV Positive Individual with Immunological and Virological Failure

Dr Samir Kumar Rama¹, Dr Dipankar Chakraborty²

¹² (Assam Rifles Composite Hospital, Shokhuvi, Dimapur, Nagaland, India, 797115)

Abstract: HIV infection of the oesophagus is one of the cause of oesophageal ulcer in HIV positive patient with odynophagia and retro- sternal chest pain. We report a case of idiopathic oesophageal ulcer in a HIV positive individual with immunological and virological failure on first line HAART who responded symptomatically and endoscopically to oral steroid, adequate nutrition and second line HAART. Recurrence of ulcer was not seen on six month follow up due to adequate immunological recovery and virological suppression on second line HAART.

Keywords: HAART, HIV, Idiopathic Oesophageal Ulcer, Immunological Failure, Virological Failure

I. Introduction

Oesophageal ulceration is a recognised manifestation of HIV infection. Oesophageal ulcers occurs at some point in HIV disease during advanced immunosupression^[1]. Ulceration occurs usually in mid to lower third of oesophagus^[1]. The most common oesophageal symptoms are odynophagia, dysphagia and retro- sternal chest pain. HIV associated oesophageal disease commonly includes candidiasis, CMV, HSV, TB, Kaposi Sarcoma, lymphoma and idiopathic ulceration. Definitive diagnosis of HIV associated oesophageal disease requires upper GI endoscopy with biopsy. The treatment of idiopathic oesophageal ulceration includes treatment of the underlying HIV induced immune suppression along with steroids^[2,3]

II. Case Report

A 42 years old male detected to be HIV positive during voluntary testing and counselling in 2008. He was in WHO Stage-I and there was no past history of opportunistic infection. His CD 4 count was 381 cells/cumm and was on yearly follow up at the ART Centre. In Sep 2010 during follow up his CD 4 Count was found to be 169 cells/cumm and was started on Highly Active Anti Retroviral Therapy (HAART) (AZT+ 3TC+ NVP) with Septran prophylaxis. Due to adherence to HAART less than 95% his CD 4 count fell down to 147 cells/cumm in Feb 2011. After counselling he was continued on first line HAART with six monthly follow up showing immunological recovery as his CD 4 count increased to 318 cells/cumm in May 2012. Despite adherence more than 95% his CD 4 count fell down in Oct 2013 to 160 cells/ cumm, also he started developing odynophagia to solid food and retro-sternal chest pain intermittently with progression in symptoms. He presented to us in Jan 2014 for follow up and was evaluated for odynophagia and immunological failure despite adherence to first line HAART more than 95%. General examination did not reveal any oral candidiasis and systemic examination was unremarkable. Upper GI endoscopy was carried out and a single ulcer was found in oesophagus 28 to 30 cm from incisor, the margins of the ulcer was not raised and there was no point of bleeding. Endoscopic oesophageal biopsy was obtained from the representative area of ulcer which on histopathological examination revealed granulation tissue with extensive area of suppurative necrosis. There was no evidence of malignancy in any of the four sections obtained for HPE. CMV and HSV was ruled out and MTB PCR was negative. Candida was also ruled out on fungal culture of the specimen obtained. His haematological and biochemical parameters were within normal limit. Viral load for HIV was 71019 copies/ ml and CD 4 count was 99 cell/ cumm. Immunological and virological failure was detected in the individual with idiophatic oesophageal ulcer causing odynophagia. The individual was started on second line HAART with combination of TDF, Lopi/rito, 3TC along with septran prophylaxis. He was also started on prednisolone 40mg Once daily for a week and thereafter tapered 10mg weekly over 04 weeks.

There was marked improvement in the symptoms with repeat viral load on 03 monthly follow up which was found to be 277 copies/ ml and CD 4 count 254 cells/cumm. Repeat relook Upper GI endoscopy was unremarkable with the raw ulcerative area which was seen in endoscopy before have completely healed up. This subjective and objective improvement of Signs and Symptoms in the individual made us to report this case of idiopathic ulcer in HIV positive which developed during his immunological and virological failure on first line HAART. Successful recovery in immune status with viral suppression on second line HAART along with short course of steroid have helped in healing up the idiopathic oesophageal ulcer in the individual.

During his last follow up in Dec 2014 there was no recurrence of signs and symptoms and his CD 4 count was found to be 312 Cells/ cumm and viral load was less than 25 copies/ ml.

III. Discussion

The oesophagus is a frequent site of infection by opportunistic infections and three pathogens candida, CMV and HSV causes the majority of them in addition although obscure in pathogenesis HIV associated oesophageal ulceration is an important disease condition. Oesophageal ulceration occurs with marked immunosupression of CD 4 count less than 100 cells/ cumm^[1] as found in our case where CD 4 count was 99 cells/ cumm and viral load 71019 copies/ ml on presentation due to immunological and virological failure on first line HAART. HIV associated oesophageal ulceration is considered and should be treated with systemic steroids which is effective in symptomatic and endoscopic healing of idiopathic ulcers^[2,3]. Study have shown that symptoms disappear during an average period of 8.3 days from presentation^[4]. In our case after ruling out the opportunistic infection as the cause of ulcer oral prednisolone was started with symptomatic improvement after 10 days of initial presentation.

IV. Conclusion

Oesophageal ulceration is a recognised manifestation of HIV infection. Oesophageal ulcer occurs at some point in HIV disease during severe immunosupression so it is important to consider it as a differential diagnosis in a HIV positive individual presenting with odynophagia and retro- sternal chest pain. Management of oesophageal ulcer in HIV positive individual is etiology dependent and effective. If no infective etiology is found HIV induced idiopathic oesophageal ulcer should be considered which can be effectively managed with inducing ulcer healing, preventing ulcer recurrence, analgesia and maintaining adequate nutrition. In our case report systemic steroid was effective in inducing healing of the ulcer and the recurrence was prevented with effective immunological recovery along with successful viral suppression by starting second line HAART.

References

- [1]. D P Epstein, M Locketz, Oesophageal ulceration in HIV infected patients, South Asian Medical Journal, Feb 2009, Vol. 99, No.2
- [2]. Wilcox CM, Esophageal disease in the acquired immunodeficiency syndrome: etiology, diagnosis, and management, American Journal of Medicine, 1992; 92: 412-421.
- [3]. Kotler DP, Reka S, Orenstein JM, et al, Chronic idiopathic ulceration in the acquired immunodeficiency syndrome. Journal of Clinical Gastroenterology 1992; 15(4): 284-290.
- [4]. Levine MS, Loercher G, Katzka DA, Herlinger H, Rubesin SE, Laufer I, Giant human immunodeficiency virus-related ulcers in the esophagu, Radiology, 1991 Aug;180(2):323-6