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## Severe Immune Thrombocytopenia in a Case of Cervical Carcinoma

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**Abstract:** A case illustrating a rare but clinically important, immune mediated complication occurring in a patient with cervical carcinoma, which is not due to tumor metastasis. A postmenopausal woman with cervical carcinoma had platelet count of  $8000 \mu\text{L}^{-1}$ . There was no splenomegaly and bone-marrow revealed megakaryocytic hyperplasia with no tumor infiltration. Other causes of thrombocytopenia were excluded and platelet count increased on treating with high dose glucocorticoids, suggesting immunologic thrombocytopenia. The possible mechanism can be reckoned to be due to cross reaction of antibodies formed against HPV oncoproteins with platelets.

**Key words:** Cervical, carcinoma, immunologic, thrombocytopenia, glucocorticoids

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### Introduction

Cancer of the uterine cervix is a rare consequence of a common infection with one of the mucosotropic Human Papilloma Viruses (HPVs), most notably HPV types 16 and 18 (Sowjanya *et al.*, 2005). Most of the complications arising due to this cancer are due to metastasis of tumor cells and bleeding from the ulceroproliferative growth on the cervix. This case illustrates a rare but clinically important immune mediated complication, occurring in a patient with cervical carcinoma, which is not due to tumor metastasis.

### Case Summary

A 50 years old woman, para 2 live 2, postmenopausal for 10 years, presented with bleeding *per vaginam* for six months to a university hospital in Southern India, in August 2004. No history suggestive of connective tissue disorders and high risk behavior for sexually transmitted diseases was given. On examination, severe pallor was present, no icterus, cyanosis, clubbing, generalized lymphadenopathy or edema were seen. No petechiae, purpura, mucosal or gum bleeding was present. Vitals were stable. Abdomen was soft, liver and spleen were not palpable and no free fluid was detected in the abdomen. A friable necrotic growth involving cervix was seen and fornices were also involved. Bilaterally, parametrium was involved upto the pelvic wall, with a frozen pelvis. Clinically she was diagnosed as carcinoma cervix stage 3b. The diagnosis was confirmed by a cervical biopsy, which showed squamous cell carcinoma, large cell, nonkeratinising. During her hospital stay, she soaked about three to 10 pads per day. Peripheral smear showed that RBCs were normocytic normochromic with many polychromatic cells and occasional nucleated RBCs. WBCs showed neutrophilic leucocytosis, and platelets were reduced markedly. At admission, her hemoglobin was 2.5 g% and subsequently she received a total of 10 units of packed cell transfusions in a span of 45 days to improve her hemoglobin status. Platelet count at admission was  $16,000 \mu\text{L}^{-1}$ , which further decreased to  $8000 \mu\text{L}^{-1}$  three days later. Bone marrow biopsy revealed hypercellular marrow with megakaryocytic hyperplasia. In addition marrow showed increased lymphocytes. There was no

evidence of tumor infiltration. Ultrasonography of abdomen showed normal sized liver and spleen. Eye fundus examination revealed fundal hemorrhages consistent with thrombocytopenia. Other investigations revealed Fibrin Degradation Products (FDP) negative, HIV negative and LE cell negative. Liver function tests were normal. She was not on treatment with any chemotherapeutic drugs at any time. The counts did not increase despite transfusing four units of platelets and four units of FFP. Then the patient was started on Tab. Prednisolone 40 mg OD for 4 days, and a repeat platelet count showed no improvement. Subsequently, she was given Inj. Methyl prednisolone 1 g OD for 3 days followed by Tab. Prednisolone 40 mg OD. The platelet counts increased to  $100,000 \mu\text{L}^{-1}$ .

### **Discussion**

Thrombocytopenia is caused by one of the three mechanisms - decreased marrow production, increased splenic sequestration or accelerated destruction of platelets. The first two causes are excluded in this patient by bone marrow biopsy which showed increased megakaryocytes, which is again consistent with peripheral accelerated destruction of platelets, and no splenomegaly by USG. As no drugs were given to the patient, drug induced thrombocytopenia is also excluded. Other causes such as DIC, HIV and connective tissue disorders are also excluded. The fact that the patient has responded to high dose of glucocorticoids, and showed increased bone marrow megakaryocytes, suggests that it is an immunologic thrombocytopenia. The possible mechanism can be reckoned to be due to cross reaction of antibodies formed against HPV oncoproteins such as E6 and E7 (Nindl *et al.*, 1994) with the platelets, leading to peripheral destruction of platelets. But this mechanism can be clinically asserted if thrombocytopenia is corrected on cure of the carcinoma. As this patient was not followed up after receiving radiotherapy, it can merely be enunciated that this case exemplify the rare occurrence of two discrete conditions i.e., immunologic thrombocytopenia and cervical carcinoma, in the same patient at a time. Further immunological research is necessitated to ascertain conclusive evidence regarding the relationship between these two conditions.

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