First Case Of Immune Thrombocytopenia In The Campath Extension Trial (CAMMS03409)

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BACKGROUND

Immune Thrombocytopenia (ITP) is a rare adverse effect caused by the treatment of Relapsing Remitting Multiple Sclerosis (RRMS) with Alemtuzumab (Campath). Secondary ITP is caused by drug exposure or an underlying disease process. Primary ITP occurs in the absence of an identifiable cause.

Drug Induced Secondary ITP occurs while taking the offending agent and often is corrected by discontinuation and brief immunotherapy treatment. Primary ITP usually requires more protracted immune treatment.

The 6 previously reported cases of Alemtuzumab[1] induced ITP in MS patients were delayed, occurring 19 to 39 months after initial exposure and 1 to 15 months after last exposure. When diagnosed and treated promptly, Alemtuzumab-induced Secondary ITP responds readily to oral steroid treatment, as occurred in this case.

OBJECTIVE

To report, what we believe to be, the first case of ITP occurring in the Campath Extension Trial.

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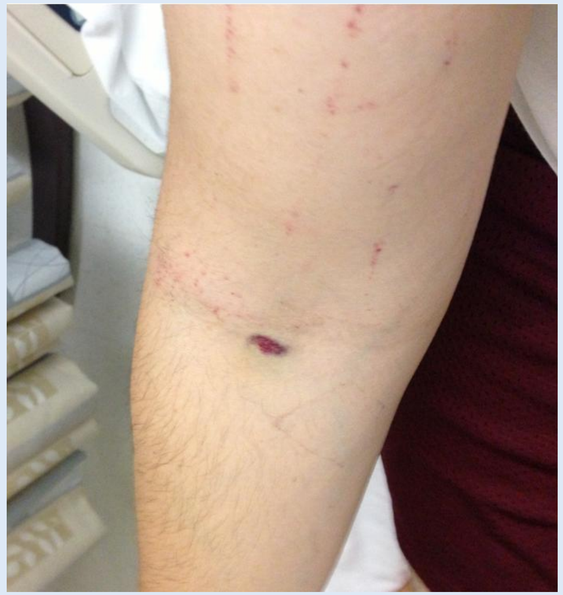

Photograph taken by patient immediately after BP cuff was removed. Note the petechiae caused by the pressure of the cuff.

DISCLOSURE
Dr. Gottesman is a principal investigator in the CAMMS03409 trial

CASE PRESENTATION

A 35 year-old man was diagnosed with RRMS in 2004 and treated with interferon beta-1a (Rebif) which was maintained until 2010 when he entered the CAMPATH trial (CARE MS 2). The past medical history was notable only for asthma and obesity. He received five 12mg intravenous daily doses of Campath in August 2010 and three additional doses in August 2011.

Monthly blood counts were normal until November 2012, when the platelet count was 51K/uL, four days later it fell to 6K/uL. The peripheral smear showed markedly reduced platelets, occasional large platelets, and no obvious giant platelets. ITP was then diagnosed, 27 months after the initial Campath treatment and 15 months after the last infusion.

Upon direct questioning, the patient recalled an episode of mild epistaxis the prior week which he did not consider significant or report.

The examination was notable only for scattered faint lower extremity petechiae; there was no other evidence of bleeding. A large bruise developed underneath the blood pressure cuff after it was removed. There were no findings indicating infection or any other illness. The only other medication was an asthma inhaler which he had used intermittently for several years.

He was started on oral prednisone 100mg daily with normalization of his platelet count within a week to 345K/uL. 25 days later he abruptly terminated a tapering dose of prednisone and his platelets were noted to be 148K/uL. Low dose prednisone treatment was reinstituted with rapid response. He now has been successfully tapered from steroids and has a normal platelet count.

CONCLUSIONS

This case highlights the need for continued laboratory and clinical monitoring to detect the delayed occurrence of ITP following the treatment of MS with Alemtuzumab. This patient had no significant clinical manifestations of thrombocytopenia; the diagnosis was made by protocol mandated monthly blood testing 27 months after the initial Alemtuzumab treatment. The patient responded to oral steroids and recovered fully without sequela.

1. A distinctive form of immune thrombocytopenia in a phase 2 study of alemtuzumab for the treatment of relapsing-remitting multiple sclerosis. Cuker, Coles, Sullivan, Fox, Goldberg, Oyuela, Purvis, Beardsley, Margolin. BLOOD, 8 December 2011. Volume 118, Number 24: 6299-6305.

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