Austin Journal of Cancer and Clinical Research

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Austin Publishing Group

Case Report

# Coexistence of Monoclonal Gammopathy of Undetermined Significance with Essential Thrombocythemia

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**Received:** May 13, 2015; **Accepted:** June 08, 2015; **Published:** July 24, 2015

**Abstract**

We found an interesting case about the patient coexistence of monoclonal gammopathy of undetermined significance (MGUS) and essential thrombocythemia (ET). After this patient has been diagnosised essential thrombocythemia five years, he suffered hyperglobulinemia. We analysis the relationship of this two diseases and review. It is known that the IL-6 can stimulate megakaryocyte proliferation *in vitro* and raise the counts of platelet *in vivo*, but pathogenesis of the two diseases about whether have the same common cellular origins needs to be explored. Coexistence of monoclonal gammopathy of undetermined significance with essential thrombocythemia in the same patient is rare and we believe that this study is of interest to us.

**Keywords:** Monoclonal gammopathy of undetermined significance; Essential thrombocythemia; IL-6; Pathogenesis; Megakaryocyte proliferation

## Case Presentation

A sixty-six year old Chinese male patient presented in January 2007 with paroxysmal dizziness and double lower limbs weakness. Physical exam was unremarkable with no palpable liver, spleen or lymph nodes, the god clear language, understanding and directional force was normal, memory and computing ability declined, right hemianopsia, bilateral pupil the same size, diameter was 3mm the light reflex sensitivity, the limbs muscle tension was normal, the limbs muscle strength was normal. Auxiliary examination: Head CT reported the border area of the right parietal lobe, temporal lobe, Occipital lobe Cerebral infarction, the left lateral Ventricle cerebral infarction; Carotid artery ultrasonic prompted the right subclavian atherosclerotic plaques; Routine blood tests prompted normal hemoglobin, white blood cell counts, a platelet count of 795-1055×$10^9$/l; Globulin and renal function were normal; tumor markers were normal; anti nuclear antibody spectrum was normal; thyroid function was normal; bone marrow biopsy and aspirate revealed non-fibrotic megakaryocytic hyperplasia. There was no evidence of monoclonal protein spike on serum and urine protein analysis, diagnosed essential thrombocythemia. Therapy with hydroxyurea (1000mg/day) was initialed and continued for six years following which the patient was put on aspirin (100mg/day). Until October 2013, the patient was found globulin raised on following, Immune fixation electrophoresis found monoclonal protein spike on serum and urine protein analysis, was IgA-κ(IgA1070mg/dl, normal 82-453mg/dl, and λ1080mg/dl, normal 629-1350mg/dl); Bone marrow biopsy revealed non-fibrotic megakaryocytic hyperplasia and immature plasma cells (8%); one marrow immunophenotype showed CD38 (+), CD138 (+), CD45 (-) cells was 2.8 percentage of nucleated cells. The patient had no JAKV617F. Fluorescence in situ hybridization (FISH) found Routine blood tests: WBC 8.0×$10^9$/l, Hb137g/l, plt 410×$10^9$/l, CRP5.0mg/l, ESR75mm/h, β2 microglobulin was 3.36mg/l (normal 1-3mg/l), renal function and blood calcium were normal. The radiological evaluations of his skeleton were not found lytic or sclerotic bone changes. The patient was diagnosed as monoclonal gammopathy of undetermined significance with essential thrombocythemia, suggested him continuing to oral hydroxyurea and following up.

## Discussion

Coexistence of monoclonal gammopathy of undetermined significance and essential thrombocythemia in the same patient is rare [1-3]. The cytokine interleukin-6 (IL-6) may lead to the occurrence of monoclonal gammopathy of undetermined significance and essential thrombocythemia happening in the same patient [4,5]. It is known that IL-6 is a potent human myeloma-cell growth factor and overproduction of this cytokine is considered to be an important component in the pathogenesis and progression of malignant plasma cell disease [6]. It is known that the IL-6 can stimulate megakaryocyte proliferation in vitro and raise the counts of platelet in vivo [4], but pathogenesis of the two diseases about whether have the same common cellular origins needs to be explored.

According to the literature [7], the standard approach to treat patients with coexistent MGUS and MPN is to treat the MPN and monitor for MGUS progression on a 3- to 4-month basis. We also treat this patient's only use oral hydroxyurea to control the platelet count and follow up the MGUS.

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**Citation:** Zhong Y and Zhang J. Coexistence of Monoclonal Gammopathy of Undetermined Significance with Essential Thrombocythemia. Austin J Cancer Clin Res 2015;2(6): 1049.

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Austin J Cancer Clin Res - Volume 2 Issue 6 - 2015
**ISSN : 2381-909X** | www.austinpublishinggroup.com


**Citation:** Zhong Y and Zhang J. Coexistence of Monoclonal Gammopathy of Undetermined Significance with Essential Thrombocythemia. Austin J Cancer Clin Res 2015;2(6): 1049.