Reviewer's report

Title: A patient with hypereosinophilic syndrome with coagulation abnormality, who relapsed without eosinophilia: a case report

Version: 1 Date: 19 September 2011

Reviewer: Hao-Wei Teng

Which of the following best describes what type of case report this is?: An unexpected association between diseases or symptoms

Has the case been reported coherently?: Yes

Is the case report authentic?: Yes

Is the case report ethical?: Yes

Is there any missing information that you think must be added before publication?: Yes

Is this case worth reporting?: Yes

Is the case report persuasive?: Yes

Does the case report have explanatory value?: Yes

Does the case report have diagnostic value?: No

Will the case report make a difference to clinical practice?: No

Is the anonymity of the patient protected?: Yes

Comments to authors:

General comments

The author reports a patient with L-HES who manifested with a coagulation abnormality. Glucocorticoids effectively suppressed hypereosinophilia and corrected the coagulation abnormality. However, excessive subsequent tapering of the glucocorticoid brought about a relapse of the coagulation abnormality, but not the eosinophilia. They also present the change of cytokines at diagnosis and relapse. This cases report is interesting with abundance of observation, like cytokines, but they should answer the following (major) critical problems regarding the criteria for HES relapse before acceptance.

Major criticisms (Revisions necessary for publication):
1. The eosinophil count is normal at relapse. According to author’s criteria 1, elevated eosinophil count is needed to confirm the relapse. How can authors say HES relapse? What criteria did authors use or how did you explain?

Minor criticisms:

1. In the section of “Differential diagnoses for coagulopathy”, Authors say the “mixed aPTT “test disclosed the underlying of FVIII deficiency is related to deficiency of FVIII (consumption). Could authors provide the laboratory data in detail (CA and mixed aPTT test(0 and 2 hr) and did author did the mixed aPTT at relapse? Also the IgAM data is needed? Another issue is that author say the FVIII deficiency was related to thrombotic events of HES, where is the thrombosis? If it is related to thrombosis, we can find “diffuse lower coagulation factors level, not only FVIII only”

2. Hypereosinophilic syndrome (HES) was one myeloproliferative disorder (MPD) defined in the 2001 World Health Organization (WHO) classification of myeloid neoplasm. The 2008 version reclassified molecularly characterized clonal eosinophilia as a myeloid neoplasm associated with eosinophilia and abnormalities of PDGFRα, PDGFRβ or FGFR1. HES patients with abnormalities of PDGFRα and PDGFRβ typically respond well to the tyrosine kinase inhibitor, imatinib mesylate. It had better did the gene test for this patient.

3. In the case presentation section, according to history, patient has HES and artery thrombosis in 5 yrs ago without Tx. “5-yr HES did not converted to target organ damage, it is rare.

4. In the discussion section, Fig g-I differ from Fig j-k? is any discussion for this observation?

5. For image, the abdo. CT with contrast is better to showed the LAPs.

Quality of written English: Acceptable

Declaration of competing interests:

no any competition