Asia Pacific Society for Immunodeficiencies (APSID)

APSID Spring School, Hong Kong

28th – 29th April 2016

28th April 2016 (Thursday) APSID Spring School
29th April 2016 (Friday) APSID Spring School
Inauguration Ceremony and Reception for APSID Congress
30th April 2016 (Saturday) APSID Congress
1st May 2016 (Sunday) APSID Congress

REGISTRATION FORM

Please type or	print your name and address in CAPITAL letters	
Name:		
Family Name		ven name
Institution	:	
Specialty:	General paediatrics / Immunology / Allergy / Haematology / Oncology/ Others (please specify)	
Current po	st:	
Year of gra	duation from medical school:	
Mailing Ad	dress:	
Dietary red	quest: Vegetarian / Halal / No pork e presentation:	
Supervisor		
Name:		·
Institution:	,	
Subspecialt	ty:	
	ormation of the supervisor	
Telephone	No:	
Email:		

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Abstract (maximum 400 words, please type in the following space; where necessary, one figure in JPEG / TIFF format is permitted. Please see an example on page 3)
Please identify key issues for discussion (maximum three): 1.
2.
3.
Please provide a personal statement what you want to achieve in the APSID Spring School and how to promote care for immunodeficient children in your country (please type in the following space, maximum 150 words).

Please return the completed registration form to the APSID Spring School Secretariat via e-mail (apsidsch@hku.hk), by 29th February 2016.

If you require a visa to travel to Hong Kong SAR, China, please do so early.

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<Example of abstract submission>

Pyrexia of unknown origin in a 1-year-old girl

Presenter: Dr MAO, Huawei

Supervisor: Professor LAU, Yu-Lung

Abstract

In November 2014, a 1-year-old girl presented to us with recurrent fever for 3 months. The patient had lymphopenia (ALC 800/ul with 12.6% CD3T, 5.95% CD3CD4T, 1.82% CD3CD8T, 54.26% CD19B and 35.5% CD56 NK cells) and low immunoglobulin level. Lymphocyte subset analysis showed T-B+NK+ phenotype. Target gene IL7Ra sequencing was performed while no mutation was identified. Exome sequencing of customerized PID-related genes was offered and in progress. The working diagnosis was combined immunodeficiency. Extensive microbiology workups were done with no positive result, except high copies of EBV DNA load were detected in the blood and nasopharyngeal aspirate. Inflammatory markers including CRP and PCT were mildly increased. Serial CXR and CT thorax suggested inflammatory infiltration and progressive space-occupying opacities. Lung biopsy was offered but refused by the parents. Broad-spectrum antibiotics and antifungals were initiated. Isoniazid and rifampicin were also started as the patient received BCG vaccination. However the fever persisted. She developed left-sided convulsion. MRI brain showed cerebellar lesion. Finally lung biopsy was performed and histology indicated the presence of AFB, while immunohistochemistry staining revealed EBV-associated B cell lymphoproliferation, and features of progression to diffuse large cell lymphoma. Antibiotics and anti-fungal drugs were stopped. Instead, ethambutol and clarithromycin were added. The fever came down gradually. Chemotherapy with rituximab plus COP was further started for the treatment of lymphoproliferative disease. After three cycles of chemotherapy, EBV was not detected and repeated CT thorax showed the size of lung lesion was reduced. Bone marrow transplantation option was offered to the parents and a full matched sibling was identified. Now the patient is stable, and waiting for the transplantation after completion of chemotherapy.

Key issues for discussion

- 1. Would she need any conditioning for the transplantation?
- 2. What is the nature of the cerebellar lesion, mycobacterial disease or B cell lymphoproliferative disease, or both?