

2 weeks before the PAIR procedure. She had mild elevation of transaminase levels at time of the procedure, but albendazole was continued for a week. Three weeks later, AST and ALT were 496 and 468 IU/L. ANA titers became positive (1:100; granular pattern). The pattern of liver enzyme derangement in the child is depicted in **Fig. 1**. Liver biopsy showed widespread portal lymphoplasmacytic inflammation with extensive interface (piecemeal) necrosis. Prednisolone and azathioprine were started. Transaminase levels rapidly decreased to normal ranges in two weeks. One month later, she had no complaints; physical examination and laboratory parameters were all normal. The steroid dose was tapered. Four months later, laboratory findings and clinical features were also normal. Afterwards, the dose of azathioprine was also tapered.

AIH can be triggered in susceptible persons by an external factor. Previous data suggest [2] that drug-

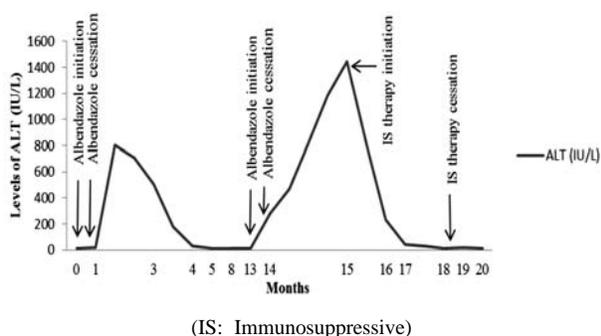


FIG. 1 The pattern of liver enzyme derangement in the patient.

induced AIH makes up a significant proportion, approximately 9%, of AIH cases [1]. Björnsson, *et al.* [2] suggested that a substantial number of patients who were found to develop drug-induced liver injury were diagnosed with AIH during follow-up.

Our patient had transaminitis recurring every time after treatment of albendazole. In the first episode, elevated transaminase levels rapidly returned to the normal ranges following the cessation of albendazole. Also, ANA was negative and IgG level was in normal range. Hence, she was diagnosed as drug-induced hepatotoxicity due to albendazole. AIH was considered during second episode as ANA became positive, IgG level raised, and liver biopsy showed histologic features of AIH. Rapid response to immunosuppressive drugs supported our diagnosis, as well.

To our knowledge, this is the first report of AIH induced by albendazole. We speculate that drug-induced AIH may be prevented by avoiding use of drugs which have previously caused hepatotoxicity in a given patient.

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MMR at 9 Months!!!

A cursory glance at the major changes in the recommendations for immunization by IAP 2014 [1] startled me and made me sit up and take notice. The committee recommends two doses of MMR at 9 and 15 months; no standalone measles dose at 9 months; and no MMR dose at 4-6 years of age. While there is no doubt about the need for two doses of MMR, it is their timing in the recommendation that is questionable.

There is indeed undeniable evidence that both mumps and rubella are also significant problems and this matter

has been addressed beautifully in recent articles [1,2] and in the IAP Guidebook on Immunization 2013-14 [3]. Till the new recommendations became available, we were giving measles vaccine at 9 months and 2 doses of MMR at 15 months and 5 years. This schedule clearly combined the benefit of early protection against measles, coverage of all three diseases as recommended by WHO, and the very important issue of long term protection against mumps and rubella.

In the pre-vaccine era, mumps usually occurred primarily in young children between the age of 5 and 9 years [4]. It is most unusual to see mumps in children younger than below 5 years; so why the haste in completing both doses of mumps by 15 months?

Outbreaks continue to occur even in highly vaccinated populations as a result of vaccine failure and also under vaccination of susceptible persons [5]. Also, though immunity appears to be long lasting, studies from the UK and the recent epidemic in the USA suggest that both antibody levels and vaccine effectiveness may decline, contributing to outbreaks mumps of in older vaccinated populations [4]. This is the basis of giving the second dose of mumps at 5 years, a practice followed all over the world. We must give mumps at 15 months and 5 years or face a massive outbreak of mumps in the older population due to waning immunity.

The case for rubella is equally interesting. Rubella vaccine should not be administered to infants younger than one year of age because persisting maternal antibodies may interfere with seroconversion [6]. Furthermore, a resurgence of rubella and congenital rubella syndrome in 1989-1991 forced the American authorities to introduce a second dose of rubella at 5 years. Once again the effort was to delay the second dose with aim of long lasting protection [7]. If our first dose of rubella vaccine at 9 months does not work and the second dose is given as early as 15 months, we may be staring at a massive outbreak of congenital rubella syndrome in the older population who has a waning immunity as seen in the West many years ago.

In a nutshell, the recommendations have been shortsighted in looking at the immediate seroconversion, and not at the long term immunity and consequences. In the private sector where families tend to follow the immunization schedule religiously, compliance becomes a relative nonissue. In this setting, should I not continue with measles vaccine at 9 months, and MMR at 15 months and 5 years?

MMR at 9 Months: Rushing in Where Others Fear to Tread?

We read with interest the IAP Committee on Immunization (IAPCOI) recommendation on MMR vaccine at 9 months of age [1]. In this context, we studied the 2014 immunization practices across 121 countries, including 4 countries in Africa, 34 in the Americas, 13 in the Eastern Mediterranean region, 51 in Europe, 15 Western Pacific

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countries and 4 South East Asian countries. In none of these countries is MMR given at 9 months, except Mongolia and Thailand. Germany uses it between 11-14 months and others give it after 1 year of age. Japan does not recommend MMR vaccine at any age.

We now know that the age at which MMR is given may have a bearing on adverse effects. On 28 September 2014, CNN announced news about a US Center for Disease Control (CDC) whistle blower - William Thompson [2] who had formerly co-authored a paper, that there is no link between the age of MMR vaccination and subsequent diagnosis of autism [3]. Thompson has now revealed that there was indeed greater risk of autism in