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INHIBITION OF ANTIBODY FORMATION IN INTRAUTERINE VARICELLA VIRUS INFECTION

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Summary. - Serological and clinical follow-up of 35 pregnancies complicated with varicella-zoster virus (VZV) infection showed insufficient antibody response in 2 children with undetectable IgG levels in ELISA and indirect haemagglutination (IHA) test. Secondary infection with VZV at the age of 4 1/2 years of the first child and inapparent infection at the age of 4 years and 3 months in the second child born with congenital varicella lead to seroconversion against VZV. None of the children in question developed acquired varicella primoinfection. We believe that a temporary specific inhibition of antibody formation was caused by transplacentar transmission of maternal antibodies into foetal circulation.

Key words: varicella zoster virus; intrauterine infection; inhibition of antibody formation

In the varicella-zoster virus (VZV) research laboratory of the Institute of Hygiene and Epidemiology in Prague we collected 35 cases of terminated pregnancy complicated with varicella. The newborn children were followed for at least several months or even 14 years and in some of them the clinical follow-ups are still being continued. This long-term study allowed to identify 2 children with extraordinary serological and clinical features.

The first child was a healthy girl, born from mother with serologically confirmed varicella in 24 th week of pregnancy. The child was repeatedly seronegative by 16 and 37 months (i. e. at intervals when no passively transferred antibody could be present), the indirect HA antibody titre being < 8 and the IgG ELISA titres ≤ 40. Further serological examination at the age of 10 years showed indirect HA titre of 256 to VZV in the absence of HSV antibody; the ELISA IgG titre to VZV was 640. Retrospective analysis of the anamnestic data showed that the child had herpes zoster in the age of 53 months which healed leaving deep scars and depigmentations in the dermatomas Th2 and Th3. The child has never before experienced varicella. All other intercurrent infections were tolerated without difficulties.

The second child was born from a mother who developed varicella 11 days

Table 1. Results of serological and clinical follow-up in children with maternal varicella

Child No.	Varicella infection of the mother	Age of the child	IHA titre*	ELISA IgG titre**	VZV infection of the child
1	24 weeks of pregnancy	16 months	<1 : 8	<1 : 40	
		37 months	<1 : 8	1 : 40	
		53 months			Zoster at Th2 and Th3
		10 years	1 : 256 <1 : 8 (HSV)	1 : 640	
2	11 days before labor (normal date)	cord blood	1 : 512 - 1024		Congenital varicella
		34 months	<1 : 8 - 1 : 8	1 : 400	
		51 months	1 : 256	1 : 1600	

* seronegativity at levels of 1 : < 8
** seronegativity at levels 1 : ≦ 40 to 1 : 400

before labor. On the day of labor the newborn had 2 typical eflorescences on its skin and during further two days a typical rash appeared. However, the course of this congenital varicella was very mild. The indirect HA VZV antibody titre in the umbilical cord blood was 512 - 1024. By the age of 34 months the IHA antibody titre was ≦ 8 and the ELISA IgG titre 400 (threshold levels) but by 51 months the VZV antibodies titred 256 and 1600, respectively (Table 1). No further VZV associated eflorescences developed and the overall state of the child was very good.

We believe that intrauterine VZV infection elicited in abovementioned children an antibody response which, after elimination of maternal antibody, was undetectable or nearly undetectable. But development of herpes zoster in the first child or an inapparent secondary VZV infection in the second caused clearcut seroconversion. Primary zoster was not yet described in a seronegative child and it seems improbable in relation to the pregnancy complicated with apparent varicella.

Foetuses may develop temporary inhibition of antibody formation to certain antigens which had been transferred across the placenta (Miller, 1980). The phenomenon is explained by the presence of maternal IgG in foetal and newborn circulation. Assuming that VZV might have such effect, we believe that our surprising results were caused by inhibition of active antibody formation associated with intrauterine VZV infection. In some cases of maternal varicella intrauterine disease can be excluded only when an acquired varicella

primoinfection occurs in the child but not according to the negative VZV serology.

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