Clinical Markers and Risk for CSF HSV DNA Persistence in Neonatal Disseminated and Central Nervous System Infection

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Introduction

es diagnosed annually with HSV
S, >30% have CNS disease. Twenty-
yclovir therapy is the recommended
at lumbar puncture indicated to confirm
tion. For those with positive PCR
the end of the therapy (pHSV),
ment is indicated until negative PCR
l. Risk factors for and clinical
SV are lacking.

Objective

neonatal HSV from 2003-2014 at our
ren's hospital and define clinical and
s associated with pHSV.

Methods

ns of age with possible HSV infection
m an ID tracking list. Clinical,
tcome data was collected on those with
F HSV PCR result – those with negative
CR result and those with pHSV were
CR testing was performed in the
Hospital Microbiology Lab. Descriptive
ized for analysis.

Results

90 charts were reviewed. 46 neonates had HSV infection, 20 with SEM, 9 with CNS disease, 17 with disseminated disease (12 with CNS disease). Of those with HSV in the CSF, 3 were excluded. 4/18 patients had pHSV and were treated for ≥28 days. Baseline characteristics and lab work were similar in both groups.

Table 1: Characteristics of patients with pHSV at admission

	Case 1	Case 2	Case 3	Case 4
Classification of HSV Disease	D-CNS	CNS	D-CNS	CNS
HSV Type	HSV-II	HSV-II	HSV-II	HSV-II
Liver Dysfunction*	Yes	No	Yes	No
Thrombocytopenia∫	Yes	No	Yes	No
Seizures	No	Yes	No	Yes
Age at onset of illness (d)	11	37	4	13
Symptoms prior starting acyclovir (d)	3	0	1	2

* = ALT greater than 2 times the normal range
∫ = Platelet count < 150 x 10^3/μL

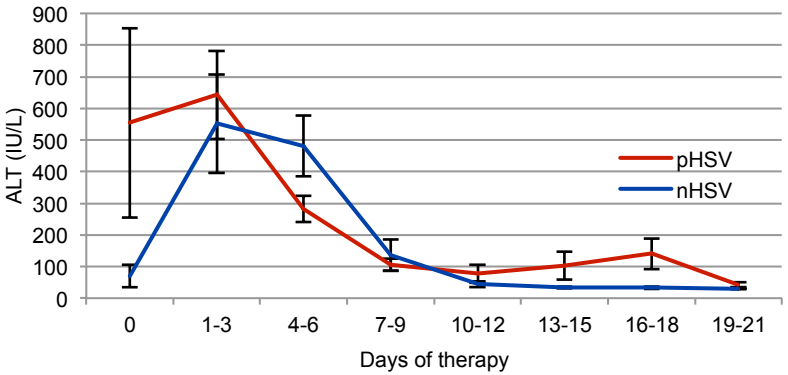

Figure 1: Values of AST over the course of hospitalization

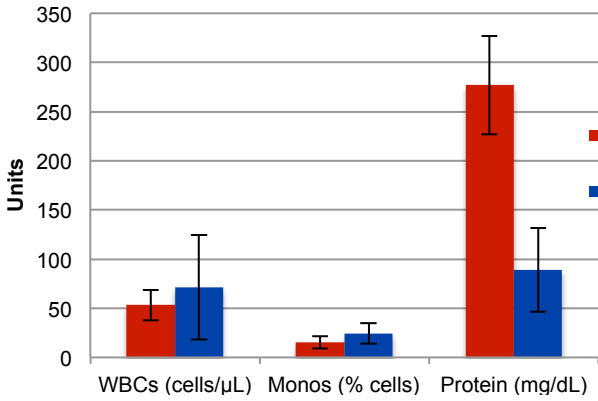

Figure 2: CSF studies from second lumbar puncture in infants with HSV CNS dise

Table 2: Neurodevelopmental outcomes

Characteristic	pHSV (n=4)	Negative DNA, n
Seizure activity after 14 days of acyclovir treatment	2 (50%)	0 (0%
Cortical blindness	2 (50)	0 (0
Motor delay	3 (75)	2 (15
Developmental delay	3 (75)	3 (23
Moderate-to-Severe Impairment∫	3 (75)	3 (23

* = one patient died shortly after completing therapy, so n=14 for this value
∫ = according to a protocol developed by RJ Whitley, MD and the NIAID CA

Conclusions

Infants with pHSV had similar presentatio
those who were negative at the end of th
severe disease during the clinical and lab
was noted in pHSV infants, characterized
transaminitis, persistent seizure activity, a
CSF protein at the end of therapy. Worse
developmental outcomes were noted in p