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## Comparison of Characteristics of Patients with West Nile Virus or St. Louis Encephalitis Virus Neuroinvasive Disease During Concurrent Outbreaks, Maricopa County, Arizona, 2015

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### Abstract

West Nile virus (WNV) and St. Louis encephalitis virus (SLEV) are closely related mosquito-borne flaviviruses that can cause neuroinvasive disease. No concurrent WNV and SLEV disease outbreaks have previously been identified. When concurrent outbreaks occurred in 2015 in Maricopa County, Arizona, we collected data to describe the epidemiology, and to compare

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Author Disclosure Statement

No conflicting financial interests exist.

Supplementary Material

Supplementary Table S1

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features of patients with WNV and SLEV neuroinvasive disease. We performed enhanced case finding, and gathered information from medical records and patient interviews. A case was defined as a clinically compatible illness and laboratory evidence of WNV, SLEV, or unspecified flavivirus infection in a person residing in Maricopa County in 2015. We compared demographic and clinical features of WNV and SLEV neuroinvasive cases; for this analysis, a case was defined as physician-documented encephalitis or meningitis and a white blood cell count  $>5$  cells/mm<sup>3</sup> in cerebrospinal fluid. In total, we identified 82 cases, including 39 WNV, 21 SLEV, and 22 unspecified flavivirus cases. The comparative analysis included 21 WNV and 14 SLEV neuroinvasive cases. Among neuroinvasive cases, the median age of patients with SLEV (63 years) was higher than WNV (52 years). Patients had similar symptoms; rash was identified more frequently in WNV (33%) neuroinvasive cases than in SLEV (7%) cases, but this difference was not statistically significant ( $p = 0.11$ ). In summary, during the first known concurrent WNV and SLEV disease outbreaks, no specific clinical features were identified that could differentiate between WNV and SLEV neuroinvasive cases. Health care providers should consider both infections in patients with aseptic meningitis or encephalitis.

## Keywords

encephalitis; West Nile virus; St. Louis encephalitis virus; neuroinvasive; outbreak

## Introduction

WEST NILE VIRUS (WNV) and St. Louis encephalitis virus (SLEV) are closely related mosquito-borne flaviviruses (Petersen et al. 2013, Curren et al. 2018). The majority of WNV and SLEV infections are asymptomatic, but can also result in nonspecific febrile illness or neurologic disease. When neuroinvasive disease occurs, it typically presents as encephalitis, meningitis, or acute flaccid paralysis. Older persons and those with certain underlying medical conditions are at higher risk for developing neuroinvasive disease (Jean et al. 2007, Lindsey et al. 2012).

Both viruses are endemic throughout much of the United States and share the same transmission cycle, including *Culex* species mosquito vectors and avian hosts. SLEV was first identified in St. Louis, Missouri, in 1933, and  $>50$  outbreaks were reported in the United States during the next 70 years (Lumsden 1958, Kopp et al. 2013). SLEV disease incidence has been substantially lower than WNV disease incidence since WNV was first identified in the United States in 1999 (Reimann et al. 2008). However, isolated cases and limited outbreaks of SLEV disease still occur sporadically in the United States.

WNV disease and SLEV disease are both nationally notifiable conditions in the United States (CDC 2018). During 2010–2014, a total of 537 patients with WNV disease and only one patient with SLEV disease were reported to Arizona Department of Health Services (ADHS). However during 2015, by the end of July, seven patients with SLEV disease had been identified among Maricopa County residents; 22 patients with WNV disease also had been reported (Venkat et al. 2015). No outbreaks involving the two viruses circulating in the same location at the same time had previously been identified. An investigation was initiated

to ascertain the magnitude and describe the epidemiology of the outbreak in Maricopa County, and compare the demographic and clinical features of patients with SLEV or WNV neuroinvasive disease.

## Materials and Methods

### Case definitions and classifications

A case was defined as a clinically compatible illness and laboratory evidence of WNV, SLEV, or unspecified flavivirus infection in a resident of Maricopa County during 2015. A clinically compatible illness was defined according to the Council of State and Territorial Epidemiologists national arboviral disease neuroinvasive and nonneuroinvasive case definitions (CDC 2015). Neuroinvasive disease is defined as encephalitis, meningitis, acute flaccid paralysis, or other acute signs of central or peripheral neurologic dysfunction, as documented by a physician. Nonneuroinvasive disease is defined as fever and absence of neuroinvasive disease. Based on laboratory results, we classified cases as confirmed WNV or SLEV infection, probable WNV or SLEV infection, or unspecified flavivirus infection (Table 1). These classifications were used for the purposes of the outbreak and differed from routine Arizona arboviral disease case classifications.

We used a stricter definition for clinically compatible illness when comparing WNV and SLEV neuroinvasive cases. A clinically compatible illness was defined as encephalitis or meningitis as documented by a physician, and a white blood cell count  $>5$  cells/mm<sup>3</sup> in cerebrospinal fluid (CSF).

### Case finding and enhanced surveillance

We performed four activities to enhance case finding. First, ADHS circulated a statewide health advisory to notify health care providers of the concurrent WNV and SLEV outbreaks and remind them to report suspected cases for further investigation and testing. Second, we contacted three major local commercial laboratories who agreed to forward WNV immunoglobulin M (IgM)-positive samples to the Arizona State Public Health Laboratory (ASPHL) for additional WNV and SLEV testing. Third, presentations on the epidemiology, clinical manifestations, and diagnosis of WNV and SLEV infections were performed at 15 health care facilities in Maricopa County. Finally, a large hospital chain with 15 hospitals in Maricopa County agreed to send to ADHS a sample aliquot from any patient for whom WNV testing was ordered for additional WNV and SLEV testing.

Local and county public health department staff requested medical records and interviewed patients to collect clinical and demographic data following the normal public health procedures for suspected WNV or SLEV cases. Information gathered and laboratory results were entered into the Arizona Medical Electronic Disease Surveillance Intelligence System. All comorbidities identified in the medical record were included. The presence or absence of symptoms and signs were determined by medical record review upon notification of a case, and patient or proxy interview; if not captured using either of these methods, the symptom or sign was assumed to be absent. These activities were considered routine public health surveillance activities and Institutional Review Board review was not required.

## Laboratory methods

WNV IgM antibody testing on serum and CSF samples was initially conducted at commercial laboratories or ASPHL using enzyme-linked immunosorbent assays (ELISA). As there was no commercially available SLEV test during the outbreak, all SLEV IgM antibody testing was performed at ASPHL using an ELISA (Martin et al. 2000). Because antibodies against WNV and SLEV can crossreact on standard diagnostic tests, confirmatory neutralizing antibody testing using a plaque reduction neutralization test (PRNT) was conducted on any sample with WNV or SLEV IgM antibodies and sufficient remaining sample quantity. PRNT was conducted at the Centers for Disease Control and Prevention (CDC) Arboviral Diseases Branch laboratory in Fort Collins, Colorado using methods previously described (Lindsey et al. 1976)]. Molecular testing was conducted commercially or at CDC by nucleic acid amplification tests.

## Descriptive and statistical analyses

We analyzed case demographic and clinical information and reported results as frequencies and proportions for categorical variables, and as median values and ranges for continuous variables. We compared confirmed and probable WNV and SLEV neuroinvasive case characteristics using Fisher's exact test for categorical data and a Mann/Whitney U test for continuous variables using SAS<sup>®</sup> version 9.3 (SAS Institute Incorporated, Cary, NC). Statistical significance was defined as a 2-sided  $p < 0.05$ .

## Results

### Outbreak description

Eighty-two cases were reported among residents of Maricopa County, including 39 WNV, 21 SLEV, and 22 unspecified flavivirus cases. Median age of patients was 57 years (range 21–89 years). Disease onset dates ranged from May 6 to October 23, 2015, and 61 (74%) cases occurred during July–September. Both WNV and SLEV cases occurred throughout the outbreak period and were widely distributed without geographic clustering (Fig. 1). Among the 39 WNV cases, 37 were confirmed and 2 were probable cases. Among the 21 SLEV cases, 19 were confirmed and 2 were probable cases. A total of 56 neuroinvasive cases (23 WNV, 18 SLEV, and 15 unspecified flavivirus cases) and 26 nonneuroinvasive cases (16 WNV, 3 SLEV, and 7 unspecified flavivirus cases) were identified.

### Comparison of WNV and SLEV neuroinvasive cases

Twenty-one WNV neuroinvasive and 14 SLEV neuroinvasive cases in 35 patients met the criteria for inclusion in the comparative analysis (Table 2 and Supplementary Table S1). The excluded neuroinvasive cases included two patients with WNV infection and four patients with SLEV infection who did not have lumbar puncture performed for unspecified reasons.

Patient demographics. Among the 35 neuroinvasive cases included in the comparative analysis, WNV neuroinvasive cases were in patients with a median age of 52 years and SLEV cases were in patients with a median age of 63 years ( $p = 0.07$ ); only 4 (19%) of 21 WNV cases were in patients  $\geq 60$  years of age compared with 8 (57%) of 14 SLEV cases ( $p = 0.03$ ). Thirteen (62%) WNV cases and 11 (79%) SLEV cases were in males ( $p = 0.46$ ).

The majority of both WNV and SLEV neuroinvasive cases were in White Non-Hispanic patients (71% and 64% of WNV and SLEV cases, respectively).

**Clinical characteristics.**—Fever was the most common clinical finding, reported in 20 (95%) of 21 WNV neuroinvasive cases and 13 (93%) of 14 SLEV neuroinvasive cases. Other common symptoms or signs included headache, nausea, vomiting and/or diarrhea, neck pain, fatigue, and photophobia; the percentage of patients with WNV or SLEV infection with each symptom or sign was not significantly different (Table 3). Rash was reported in 7 (33%) WNV cases compared with 1 (7%) SLEV case ( $p = 0.11$ ). The median CSF white blood cell count was 110 cells/mm<sup>3</sup> (range, 14–4750) among 21 WNV neuroinvasive cases and 66 cells/mm<sup>3</sup> (range, 6–259) among 11 SLEV neuroinvasive cases ( $p = 0.08$ ) in which values were recorded (Table 4).

**Comorbidities.**—Among the 21 WNV neuroinvasive cases, 12 (57%) were among patients with at least one comorbidity documented; the most common conditions among the 12 patients were hyperlipidemia (67%), hypertension (33%), and cancer or history of cancer (25%). Additionally, 13 (93%) of 14 patients with SLEV neuroinvasive disease had at least one comorbidity. The most common comorbidities among these 13 patients were hypertension (69%), heart disease (54%), and diabetes mellitus (31%).

**Clinical course and outcomes.**—All patients were hospitalized and one patient died during hospitalization (Table 2). The death was in a white female >70 years of age with WNV encephalitis who had multiple comorbidities, including rheumatoid arthritis, hyperlipidemia, emphysema, and hypothyroidism.

## Discussion

The first known concurrent outbreaks of WNV and SLEV, two closely related flaviviruses, occurred in Maricopa County, Arizona in 2015. We found no apparent differences in clinical characteristics among 21 neuroinvasive WNV cases compared with 14 neuroinvasive SLEV cases.

The reason for the concurrent disease outbreaks in 2015 is unknown. Both WNV and SLEV are maintained in nature in mosquito/bird/mosquito transmission cycles, and changes in both *Culex* mosquito vector and avian host populations, linked to environmental factors, likely produced conditions favorable for transmission (Diaz et al. 2018). A factor contributing to reemergence of SLEV in Arizona is thought to be introduction of a new strain of SLEV closely related to an epidemic strain from Argentina (Diaz et al. 2018).

Although there were no significant differences in clinical characteristics of WNV and SLEV neuroinvasive cases, we found a higher proportion of SLEV neuroinvasive cases among persons ≥60 years of age compared with WNV neuroinvasive cases. Similar to our results that showed 57% of all SLEV neuroinvasive cases among persons ≥60 years of age, some previous studies have also suggested ~60% of SLEV neuroinvasive cases occur in persons in this age group (Powell and Blakey 1977). However, in contrast, reports from the U.S. National Notifiable Disease Surveillance System and most SLEV studies have shown 30–

45% of SLEV neuroinvasive disease is in persons  $\geq 60$  years of age (Hopkins et al. 1975, Reimann et al. 2008, Curren et al. 2018). We found only 19% of all WNV neuroinvasive cases occurred in persons  $\geq 60$  years of age, but previous surveillance data have indicated that 45%–50% of neuroinvasive cases caused by WNV occur in this age group (Reimann et al. 2008, Lindsey et al. 2014). Therefore, the importance of our finding of the different comparative age distributions is uncertain. One possible explanation could be a focal area of SLEV transmission in an area of Maricopa County with a higher percentage of people  $\geq 60$  years of age, although this was not supported by the widespread geographical distribution and lack of clustering of WNV and SLEV cases. Our results might also have been affected by the inability to determine the specific infecting virus in 15 (27%) of the 56 neuroinvasive cases, resulting in these unspecified flavivirus cases being excluded from the analysis.

One interesting clinical finding was the higher frequency of rash in WNV cases compared with SLEV cases, with this symptom reported among 33% of 21 WNV cases and only 7% of 14 SLEV cases. No studies have directly compared patients during concurrent outbreaks, but disease-specific descriptions of the clinical presentations of these diseases support a higher frequency of rash in patients with WNV compared with SLEV neuroinvasive disease (Southern et al. 1969, Jones et al. 2002, Sejvar et al. 2003, Bode et al. 2006).

Several limitations should be considered. First, even though this was the largest outbreak of SLEV disease in  $>10$  years, and the first outbreak of WNV and SLEV diseases occurring concurrently, relatively few neuroinvasive cases were available for the comparative analysis (Curren et al. 2018). Asymptomatic to symptomatic infection ratios are high for both WNV and SLEV infections, so patients with clinical disease represent only a small proportion of all infections (Reisen et al. 2008, Petersen et al. 2013). Inability to define the infecting virus because of flavivirus crossreactivity resulted in unspecified flavivirus diagnoses and contributed to the few WNV and SLEV cases for the comparative analysis. Limited case numbers also prevented us from performing multivariate analysis. Second, data were gathered from medical record reviews conducted for surveillance purposes and relevant information might not have been documented in the medical record. Third, clinical information was typically collected soon after hospital admission and the interval between symptom onset and admission could have influenced the likelihood of presence of symptoms and reported clinical findings.

## Conclusions

Given that both WNV and SLEV diseases are caused by closely related flaviviruses, it is not surprising that the comparison of patient demographic and clinical characteristics did not clearly indicate specific features that could reliably differentiate the two infections. Nonetheless, it was useful to confirm these clinical findings during the first recognized outbreak that involved these two viruses circulating in the same location at the same time.

Health care providers should consider both WNV and SLEV infections in the differential diagnosis in patients with aseptic meningitis and encephalitis and obtain appropriate CSF samples, serum samples, or both for laboratory testing. Confirmatory testing at state health departments or CDC will be required to distinguish these flavivirus infections. Although



clinical management for both diseases involves the same approach of supportive care, differentiating both infections is helpful for better understanding of their epidemiology, including any change in disease transmission patterns. Because human vaccines against domestic arboviruses are not available, prevention of arboviral infection depends on local vector control, community and household efforts to reduce vector populations (*e.g.*, removal of standing water), and individual efforts to decrease exposure to mosquitoes (*e.g.*, applying mosquito repellent, wearing protective clothing, and eliminating mosquito breeding sites).

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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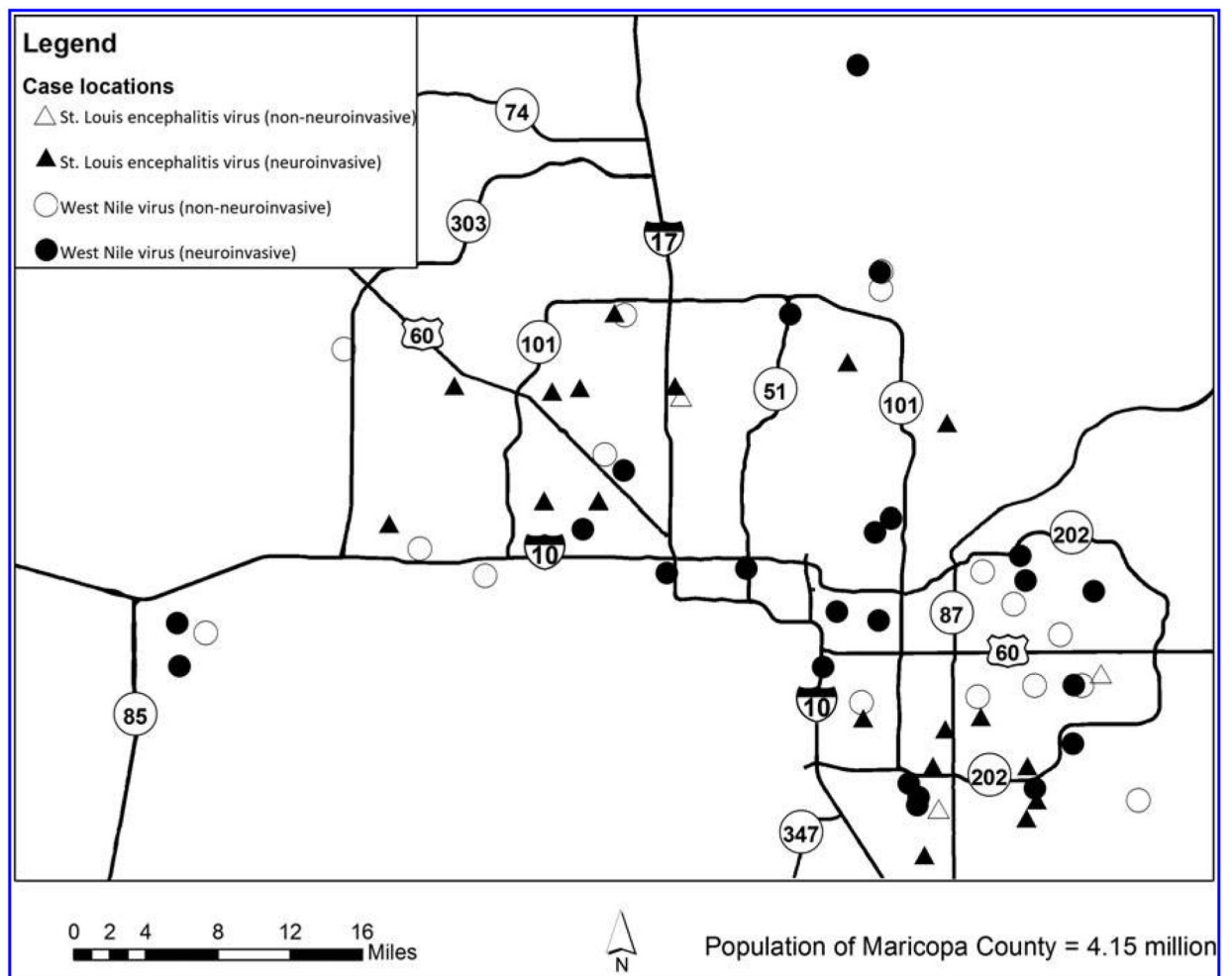
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**FIG. 1.**  
Geographic distribution of neuroinvasive and nonneuroinvasive West Nile virus and St. Louis encephalitis virus cases—Maricopa County, Arizona, 2015.

Laboratory Criteria for Classification of Cases During Concurrent West Nile Virus and St. Louis Encephalitis Virus Disease Outbreaks—Maricopa County, Arizona, 2015

**Table 1.**

Case classification	Laboratory criteria
Confirmed SLEV	<ul style="list-style-type: none"> <li>• Detection in serum of IgM antibodies against either or both viruses and SLEV-neutralizing antibody titers 4-fold higher than WNV-neutralizing antibody titers, OR</li> <li>• Detection in CSF of SLEV IgM antibodies and no detectable WNV IgM antibodies (regardless of serum results), OR</li> <li>• Detection of SLEV RNA by NAAT</li> </ul>
Confirmed WNV	<ul style="list-style-type: none"> <li>• Detection in serum of IgM antibodies against either or both viruses and WNV-neutralizing antibody titers 4-fold higher than SLEV-neutralizing antibody titers, OR</li> <li>• Detection in CSF of WNV IgM antibodies and no detectable SLEV IgM antibodies (regardless of serum results), OR</li> <li>• Detection of WNV RNA by NAAT</li> </ul>
Probable SLEV	<ul style="list-style-type: none"> <li>• Detection in serum of SLEV IgM antibodies and no detectable WNV IgM antibodies</li> </ul>
Probable WNV	<ul style="list-style-type: none"> <li>• Detection in serum of WNV IgM antibodies and no detectable SLEV IgM antibodies</li> </ul>
Unspecified flavivirus	<ul style="list-style-type: none"> <li>• Detection in CSF or serum of either SLEV or WNV IgM antibodies and no testing for the other virus, OR</li> <li>• Detection in CSF or serum of both SLEV and WNV IgM antibodies and no confirmatory neutralizing antibody testing performed, OR</li> <li>• Detection in CSF or serum of SLEV and/or WNV IgM antibodies and &lt;4-fold difference in neutralizing antibody titers</li> </ul>

CSF, cerebrospinal fluid; IgM, immunoglobulin M; NAAT, nucleic acid amplification test; RNA, ribonucleic acid; SLEV, St. Louis encephalitis virus; WNV, West Nile virus.

**Table 2.**

Comparison of Neuroinvasive West Nile Virus and St. Louis Encephalitis Virus Cases—Maricopa County, Arizona, 2015

<i>Characteristic</i>	<i>WNV cases (n = 21)</i>	<i>SLEW cases (n = 14)</i>	<i>p Value</i>
Age, median (range), in years	52 (21–79)	63 (23–84)	0.07
Age group, in years, <i>n</i> (%)			
0–19	0 (0)	0 (0)	
20–39	5 (24)	1 (7)	
40–59	12 (57)	5 (36)	—
60	4 (19)	8 (57)	
Male sex, <i>n</i> (%)	13 (62)	11 (79)	0.46
Race, <i>n</i> (%)			
White, Non-Hispanic	15 (71)	9 (64)	0.72
Other or unknown	6 (29)	5 (36)	—
Hospitalized, <i>n</i> (%)	21 (100)	14 (100)	—
Death while hospitalized, <i>n</i> (%)	1 (5)	0 (0)	1.00

**Table 3.**

Comparison of Symptoms and Signs of Neuroinvasive West Nile Virus and St. Louis Encephalitis Virus Cases  
—Maricopa County, Arizona, 2015

<i>Sign or symptom</i>	<i>WNV cases (n = 21)</i>	<i>SLEV cases (n = 14)</i>	<i>p Value</i>
	<i>n (%)</i>	<i>n (%)</i>	
Fever	20 (95)	13 (93)	1.00
Headache	19 (90)	10 (71)	0.19
Nausea, vomiting, diarrhea	16 (76)	11 (79)	1.00
Neck pain	13 (62)	7 (50)	0.51
Fatigue	12 (57)	11 (79)	0.28
Photophobia	11 (52)	5 (36)	0.49
Altered mental status	9 (43)	9 (64)	0.31
Arthralgia or myalgia	9 (43)	5 (36)	0.74
Rash	7 (33)	1 (7)	0.11
Tremor	6 (29)	6 (43)	0.48
Flaccid paralysis	3 (14)	0 (0)	0.26
Seizure	2 (10)	1 (7)	1.00

Comparison of Cerebrospinal Fluid Parameters of Neuroinvasive West Nile Virus and St. Louis Encephalitis Virus Cases—Maricopa County, Arizona, 2015

**Table 4.**

CSF parameter	Normal range	WNV cases			SLEV cases		
		Median	range	n <sup>a</sup>	Median	range	n <sup>a</sup>
WBC (cells/mm <sup>3</sup> )	0–5	110	(14–4750)	21	66	(6–259)	11
Glucose (mg/dL)	40–70	56	(5–123)	19	59	(45–109)	10
Protein (mg/dL)	15–60	57	(32–387)	19	86	(39–241)	10

<sup>a</sup>Missing results were either because complete lumbar puncture results were not listed in the medical records obtained, or the exact value was not listed (*i.e.*, listed as “high” or “elevated”).

WBC, white blood cell count.