In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: February 19, 2020

JAMES F. DUNN,	*	PUBLISHED
Petitioner,	*	No. 16-1506V
v.	*	Special Master Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Tetanus-Diphtheria-Acellular Pertussis Vaccine ("Tdap"); Varicella Zoster Virus ("VZV") Infection; Meningoencephalitis; Reactivation; Alternative Factor Unrelated to Vaccine
Respondent.	*	

Jeffrey A. Golvash, Brennan, Robins & Daley, P.C., Pittsburgh, PA, for petitioner.
Darryl R. Wishard, United States Department of Justice, Washington, DC, for respondent.

DECISION[1]

I. INTRODUCTION

On November 14, 2016, James F. Dunn ("petitioner") filed a petition under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"),[2] 42 U.S.C. § 300aa-10 et seq. (2012), alleging that as a result of receiving a Tetanus-diphtheria-acellular pertussis

[1] Because this decision contains a reasoned explanation for the undersigned's action in this case, the undersigned intends to post this decision on the website of the United States Court of Federal Claims, in accordance with the E- Government Act of 2002, 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** As provided by Vaccine Rule 18(b), each party has 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b).

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2012). All citations in this decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

("Tdap") vaccine on December 2, 2014,[3] he developed meningoencephalitis. Petition at Preamble; Joint Prehearing Submission ("Joint Sub."), filed May 23, 2019, at 1 (ECF No. 59).

Petitioner asserts that the Tdap vaccination he received in December 2014, caused an "immune-mediated inflammatory response, such as occurs with normal antibody production post-Tdap vaccination" resulting in meningoencephalitis. Petitioner's Prehearing Submission at 6 (ECF No. 53). Respondent argues against awarding compensation, stating that petitioner failed to provide preponderant evidence that his illness was caused by the Tdap vaccine. Respondent's Report at 9. Respondent also contends that petitioner's meningoencephalitis was caused by a varicella zoster virus ("VZV") infection, an alternative factor, unrelated to the Tdap vaccine. Respondent's Pre-Hearing Brief at 1 (ECF No. 58).

Petitioner suffered a very serious and significant illness which required hospitalization in an intensive care unit. He suffered respiratory failure requiring intubation and ventilation, and other sequela, which had a profound impact on his life, for which the undersigned extends her sympathy. However, after carefully analyzing and weighing all of the evidence and testimony presented in this case in accordance with the applicable legal standards, the undersigned finds that petitioner is not entitled to compensation.

Even assuming that petitioner provided preponderant evidence of causation, the undersigned finds that respondent proved by preponderant evidence that petitioner's meningoencephalitis was caused by VZV infection reactivation, an alternative factor unrelated to his Tdap vaccination. Therefore, petitioner is not entitled to compensation, and his case must be dismissed.

II. PROCEDURAL HISTORY

Petitioner, James F. Dunn, filed for compensation under the National Vaccine Injury Compensation Program on November 14, 2016. Petitioner alleged that he developed viral encephalopathy/aseptic meningitis, which was caused-in-fact by the Tdap vaccination he received on December 2, 2014. Petition at Preamble. Petitioner alleged that he "suffered from the residual effect and/or complication from his viral encephalopathy/aseptic meningitis for more than six (6) months." Id. at ¶ 14. Petitioner filed medical records on January 9, 2017. Petitioner's Exhibits ("Pet. Exs.") 2(a)-5(b). Respondent filed his Rule 4(c) Report on March 8, 2017, recommending against compensation. Respondent's Report at 9 (ECF No. 12).

Petitioner filed additional medical records on May 5, 2017. Pet. Exs. 6-8. On August 28, 2017, petitioner filed the expert report of Dr. George Small, a neurologist, and respondent filed the expert report by Dr. Subramanian Sriram, a neurologist, on November 9, 2017. Pet. Ex. 9; Respondent's Exhibit ("Resp. Ex.") A. Petitioner filed a responsive expert report from Dr. Small on January 9, 2018. Pet. Ex. 10.

[3] The parties later stipulated that petitioner received the Tdap vaccine on either December 2, or December 4, 2014. Joint Sub., filed May 23, 2019, at 1 (ECF No. 59). For purposes of this decision, December 2, 2014 will be referred to as the date of vaccination.

On February 5, 2018, respondent filed a second expert report from Dr. Sriram, including responses to questions the special master posed during the January 25, 2018 Status Conference and responses to Dr. Small. Resp. Exs. E, M. During this period, the parties discussed settlement of this matter but were unable to resolve the case informally. Respondent's Status Report, filed Mar. 7, 2018 (ECF No. 40). Petitioner then filed responses to questions the special master posed and a responsive expert report from Dr. Small on May 18, 2018. Pet. Exs. 13, 19. Both parties filed medical literature referenced by their respective experts.

Petitioner filed additional medical records on October 15, 2018. Pet. Ex. 21. On October 19, 2018, respondent filed a supplemental expert report from Dr. Sriram. Resp. Ex. N. Petitioner filed a third expert report from Dr. Small on April 25, 2019, and respondent filed another expert report from Dr. Sriram in response on May 23, 2019. Pet. Ex. 22; Resp. Ex. P.

The parties filed a joint stipulation of facts on May 23, 2019, in which they agreed that the petitioner received the Tdap vaccination in December 2014 (either on December 2, 2014 or December 4, 2014) in the United States, and that he was diagnosed with meningoencephalitis in December 2014. Joint Sub., filed May 23, 2019, at 1 (ECF No. 59). The parties disagreed on the significance of the VZV test performed on the petitioner during his admission at Allegheny General Hospital ("AGH") in December 2014. Id. at 2.

An entitlement hearing was held on June 26, 2019 in Pittsburgh, Pennsylvania. Dr. Small testified on behalf of the petitioner. Dr. Sriram testified on behalf of respondent. Post-hearing, petitioner and respondent both continued to file additional exhibits, including medical literature and expert reports until the record was closed on November 26, 2019. Petitioner filed a fourth expert report from Dr. Small on November 15, 2019. Pet. Ex. 38. Respondent filed a final expert report from Dr. Sriram on November 26, 2019. Resp. Ex. S.

The matter is now ripe for adjudication.

III. ISSUES TO BE DECIDED

The parties dispute causation.[4] Petitioner asserts that his Tdap vaccination caused him to suffer meningoencephalitis and maintains that he has proven by preponderant evidence the standards articulated in Althen. See Althen v. Sec'y of Health & Human Servs., 418 F.3d 1274, 1280 (Fed. Cir. 2005), Joint Sub., filed May 23, 2019, at 2 (ECF No. 59). Respondent disagrees. Moreover, respondent contends that even if petitioner has met his burden under Althen, there was an alternate cause for petitioner's illness, unrelated to his vaccination. Respondent "contends that petitioner's VZV infection was the likely cause of his meningoencephalitis." Id.

[4] In addition, the respondent disagrees with the tentative findings set forth in an Order filed April 6, 2017, as to whether petitioner's evidence meets the severity requirements under the Act. See 42 U.S.C. §§ 300aa-11(c)(1)(D)(i), (iii); Joint Sub., filed May 23, 2019 at 2 (ECF No. 59). This decision makes that tentative finding not relevant. The parties also dispute the significance of petitioner's testing regarding VZV infection. That issue will be discussed in the context of the experts' opinions and causation analysis.

For purposes of this decision, the undersigned assumes that petitioner has proven his case, and that the burden then shifted to respondent to show that a factor unrelated to the vaccination caused petitioner's meningoencephalitis. Therefore, the causation analysis relates only to respondent's theory of causation and does not include an analysis of petitioner's causation claim.

IV. FACTUAL SUMMARY

A. Summary of Facts

In his Prehearing Memorandum, petitioner sets forth a summary of facts which the undersigned finds generally accurate, with some additions and explanations. Petitioner's Prehearing Memorandum, filed Apr. 25, 2019, at 1-4 (ECF No. 53). The summary below is largely derived from petitioner's summary.[5]

At the time petitioner received the vaccination at issue in this case, he was forty-four years old, active, and working. On November 14, 2013, petitioner weighed 348 pounds, and was diagnosed as morbidly obese. His blood pressure was elevated at 137/97. Pet. Ex. 2(a) at 1, 2, 175. Petitioner's past medical history was significant for ocular stroke,[6] obstructive sleep apnea, post-traumatic stress disorder, and hyperlipidemia. Pet. Ex. 4(a) at 118, 123, 126.

On December 2, 2014, petitioner received a Tdap vaccine at the Veteran's Administration Hospital ("VA"). Pet. Ex. 3. On or about December 12, 2014, he began to experience headaches. Petition at ¶ 4. Approximately on December 18, 2014, petitioner began to have flu-like symptoms including fever, chills, headache, and mild nausea. Id.; Pet. Ex. 5(b) at 298. On December 19, 2014, petitioner awoke feeling warm, fatigued, and generally not himself. Petition at ¶ 5; Pet. Ex. 4(a) at 8, 118, 123, 126, 129. Petitioner went to work but left work early to return home. Petition at ¶ 5; Pet. Ex. 4(a) at 8, 118, 123, 126, 129. When petitioner's wife arrived home, petitioner was unable to respond to questions or commands. Petition at ¶ 5; Pet. Ex. 4(a) at 8, 118, 123, 126, 129. She dialed 911 for emergency medical assistance and petitioner was taken by ambulance to AGH for evaluation and treatment. Petition at ¶ 5; Pet. Ex. 4(a) at 8, 118, 123, 126, 129.

When petitioner arrived at the emergency room, he was nonverbal and nonresponsive, could open his eyes to painful stimuli, but was otherwise encephalopathic. Pet. Ex. 4(a) at 8, 118, 121, 123. CT and MRI of head and chest X-ray were unremarkable. Due to his altered mental status ("AMS"), a lumbar puncture ("LP") was performed. Pet. Ex. 4(a) at 8, 118-19, 121, 123, 126-27. Cerebral spinal fluid ("CSF") analysis revealed elevated white blood cells of 16 and elevated protein of 63 and glucose of 83. Pet. Ex. 4(d) at 741. Bacterial antigens and Gram stain were negative. Polymerase chain reaction ("PCR") for herpes simplex virus ("HSV")

[5] Petitioner's summary for events occurring after November 2015 is not included here.

[6] A brain MRI done October 23, 2013 showed small old lacunar infarct in the right superior frontal periventricular white matter. Pet. Ex. 2(a) at 21. On October 25, 2013, petitioner was diagnosed with central retinal artery occlusion and acute vision loss of his left eye. Id. at 48.

and VZV were also negative. Id. at 741-42. Influenza type A, B, and A-H1N1 were all negative. Id. Cryptococcal antigen was negative. Pet. Ex. 4(a) at 8, 118-19, 126-27; 129-31; Pet. Ex. 4(d) at 741-42.[7] Blood cultures were also negative. Petitioner was started on empiric antiviral and antibiotic therapy with acyclovir, ceftriaxone, and vancomycin. Pet. Ex. 4(a) at 8, 118, 126, 129. He was transferred to ICU for continued evaluation and treatment. Id. at 126. Impression was altered mental status likely secondary to viral encephalitis/aseptic meningitis. Id. at 127.

Petitioner was seen by an infectious disease specialist on December 20, 2014. He was observed to be nonresponsive and otherwise encephalopathic. Pet. Ex. 4(a) at 119. He had no evidence of skin rash. Id. Assessment was encephalopathy secondary to toxic ingestion versus nicotine toxicity versus viral encephalitis, possible meningoencephalitis, abnormal cerebrospinal fluid, and hypoxia. Id. at 21, 119. The infectious disease specialist suspected abnormal CSF findings were reactive in nature as petitioner did not have any evidence of infection. Id. at 119. Petitioner was also seen by a neurologist on December 20, 2014. Id. at 130. Temperature was 37.8 ºC and the petitioner was noted to be confused, nonverbal, unable to track, exhibiting non-purposeful movements but without seizures, and there was no evidence of skin rash. Id. The neurologist assessment was altered mental status, likely viral encephalitis considering the CSF. Id. at 130-31.

Petitioner remained in the ICU through December 24, and was nonresponsive, unable to follow commands, unable to verbalize, hyper-reflexive, and encephalopathic. Pet. Ex. 4(a) at 25-28. Due to acute respiratory failure, he underwent bronchoscopy on December 23, 2014. Id. at 132. A biopsy of the lung Gram stain, bacterial culture, and viral culture were all negative. Pet. Ex. 4(d) at 761. Following his bronchoscopy, petitioner continued to be followed and monitored in the ICU. Petitioner remained encephalopathic as he continued to be nonverbal without showing any signs of understanding. Pet. Ex. 4(a) at 44, 140. His differential diagnosis was encephalitis/aseptic meningitis.[8] Id. at 37, 47. On December 23, 2014, a bilateral rash was subsequently observed and swabs were sent for testing.[9] Id. at 36, 140. He had a PICC line inserted on December 24, 2014 for fluid and medications. Id. at 42. Petitioner remained nonresponsive and noncommunicative with intermittent body tremors.

On December 25, 2014, petitioner's mental status began to improve. He awoke on that day and was alert and oriented as to time, place, and person. The antivirals and antibiotics were discontinued. Pet. Ex. 4(a) at 140.

[7] Urine toxicology screen was positive for benzodiazepines and cannabinoids. It was noted that petitioner used cannabis daily and that he vaped nicotine. Pet. Ex. 4(a) at 8; Pet. Ex. 4(d) at 739.

[8] Aseptic meningitis is defined as "any of several mild types of meningitis, most of which are caused by viruses." Dorland's Illustrated Medical Dictionary 1117 (33rd ed. 2020).

[9] Additional details about petitioner's rash and swab testing are set forth below in section ii: "Additional Facts Regarding Rash and Diagnostic Testing."

Given his continued improved mental state, petitioner was discharged from the hospital on December 27, 2014 with instructions to follow up with his primary care physician. Pet. Ex. 4(a) at 62, 140. At the time of discharge, petitioner's differential diagnosis was altered mental status secondary to encephalitis/aseptic meningitis and acute respiratory failure secondary to encephalitis/aseptic meningitis. Id. at 57, 62. On December 26, 2014, the infectious disease physician wrote, "encephalopathy resolved—suspect nicotine overdose initially followed by withdrawal." Id. at 60. The discharge summary dated December 27, stated "leukocytosis concerning for viral etiology" and PCR for VZV and HSV were negative. Id. at 140. (The reference to PCR here applies to CSF.) The discharge note does not reference the PCR swab test for petitioner's rash. See Pet. Ex. 4(d) at 758. At the time of discharge, petitioner had word-finding difficulty, general fatigue, and some short-term memory lapses. Petition at ¶ 8.

i. Subsequent Care

On January 13, 2015, petitioner was seen in follow-up by his primary care physician, Derek Pae, M.D., at the VA. Dr. Pae noted petitioner's recent hospitalization for altered mental status and presumptive diagnosis of viral encephalitis. Pet. Ex. 5(b) at 303-04. Petitioner reported a depressed mood, cognitive/short term memory deficiency, and that he generally felt "clouded," which affected his ability to function. Id. Dr. Pae recommended further consult and evaluation by an infectious disease specialist. Id.

Petitioner was seen by Jae Ho Hong, M.D., an infectious disease specialist at the VA, on January 28, 2015. Pet. Ex. 5(b) at 296. During the visit, Dr. Hong noted, "Most likely the cause was viral encephalitis. However, on reviewing the chart, he received Tdap vaccine on the beginning of December. . . . Post-vaccination ADEM [acute disseminated encephalomyelitis] has been associated with several vaccines such as rabies, diptheria?tetanus? . . . . Anyway, he is clinically improving and no need for further testing or treatment." Id. at 297. Subsequently on February 4, 2015, Dr. Hong prepared an allergy and immunology adverse event note. In the adverse event note, Dr. Hong reported that petitioner had a Tdap vaccine on December 2, 2014 and that petitioner was hospitalized on December 19, 2014 with altered mental status and aseptic meningitis.[10] Id. at 296, 357.

On March 24, 2015, petitioner was again seen by Dr. Pae. Petitioner continued to have short-term memory lapses and lethargy. Pet. Ex. 5(b) at 292-93. The short-term memory lapses impacted petitioner's ability to perform everyday living activities. Id. Dr. Pae suspected that petitioner's cognitive defects and short-term memory loss was caused by his hospitalization and worsened by depression. Id. Dr. Pae prescribed an antidepressant (sertraline). Id. at 292-93, 364. On May 12, 2015, petitioner presented to Dr. Pae in follow-up. Petitioner's cognitive defects, memory loss, and depression had improved since taking sertraline. Pet. Ex. 5(a) at 95.

[10] Adverse Event Note states: "PT was admitted to AGH on 12/19/2014 with AMS. LP shows aseptic meningitis result. Had Tdap vaccine on 12/2/14." Pet. Ex. 5(b) at 357.

Petitioner next saw Dr. Pae on November 10, 2015. Pet. Ex. 5(a) at 93. Dr. Pae wrote: "I am [] concerned because after his hospitalization for encephalitis, no known cause found and per ID [infectious disease], most likely 2/2 [secondary to] tetanus vaccine. He is understandably reluctant about future vaccines." Id. at 94.

On November 25, 2015, petitioner sought emergency care for fever and chills. His prior medical history noted his previous hospitalization for encephalitis, which may have been secondary to Tdap vaccine. Petitioner was discharged the next day with his symptoms resolved. Pet. Ex. 5(a) at 20-21.

Petitioner was seen in follow-up by Dr. Omran on December 2, 2015. Petitioner's past medical history was significant for "encephalitis in 2014, temporal link to tetanus vaccine" and "adverse reaction/allergy to Tdap". Pet. Ex. 5(b) at 191-92. At that time, petitioner was still taking sertraline for his depression. Id.

On May 6, 2016, petitioner was seen by a rheumatologist at the VA. His prior medical history was significant for aseptic encephalitis/meningitis that may be Tdap vaccine related. Pet. Ex. 5(b) at 99. Petitioner was still taking sertraline. Id. By July 13, 2016, approximately one and half years after his initial hospitalization, initial mental slowing, progress notes reflect that petitioner's cognitive/memory impairment had resolved. Id. at 119.

ii. Additional Facts Regarding Rash and Diagnostic Testing

In addition to the facts set forth above, the following facts are relevant and pertinent.

On the date of vaccination, petitioner presented to Dr. Pae on December 2, 2014, for a routine follow-up visit. On that date, Dr. Pae noted that Mr. Dunn had "a truncal rash which does not itch or cause pain." This problem had been treated in the past with medication. Pet. Ex. 5(b) at 371. Dr. Pae also documented that petitioner had "another separate lesion on his right arm which is different and itchy. He has always had 'skin problems' in the past but this is new." Id. Dr. Pae diagnosed Mr. Dunn's truncal rash as "suspect[ed] tinea corporus" (fungus) and the lesion on his right arm as eczema. Id. Mr. Dunn received the Tdap vaccine at this visit. Id. at 373.

On December 19, 2014, petitioner was taken to the AGH emergency department with decreased mental status and admitted for evaluation and treatment. Lab work testing included blood work for VZV IgG, which was positive: VZV IgG: >4000.[11] Pet. Ex. 4(a) at 189.

Diagnostic testing was ordered on December 20, 2014, including CSF tests by PCR for a number of viruses, including HSV, VZV, enterovirus, West Nile virus, Lyme Disease, syphilis, human immunodeficiency virus, and others. Pet. Ex. 4(a) at 189. The results of the PCR tests on

[11] Petitioner's result was greater than 4,000 units. A positive result is generally greater than 165 units. Pet. Ex. 4(d) at 746.

the petitioner's cerebrospinal fluid were ultimately reported back as negative, including the test for VZV.[12] Id.; Pet. Ex. 4(d) at 741-42.

On December 23, 2014, an infectious disease physician documented the presence of skin vesicles on petitioner's left chest and left upper extremity where the cardiac monitor pad and BP cuff had been placed. Pet. Ex. 4(a) at 36. The vesicles looked like "contact dermatitis" as they were not in a dermatomal distribution. Id. "However, given possible viral meningitis/encephalitis, shingle/herpetic lesions should be ruled out." Id. "Blister in LUE [left upper extremity] and chest—not typical appearance of shingles but [] and fever, droplet precautions given that patients next room are immunosuppressed. Skin lesions in VZV encephalitis may develop after AMS even during tx [treatment]." Id. 35.

The physician ordered droplet isolation, and a PCR swab test from the left wrist blister for herpes simplex virus and VZV. Pet. Ex. 4(a) at 35. The physician also ordered to continue IV acyclovir (antiviral) until the cerebrospinal fluid and blister swab results were returned. Id. The PCR of the blister subsequently tested positive for VZV. Pet. Ex. 4(d) at 758.

Petitioner's medical records do not state when the results of the positive VZV blister test were received or whether his physicians were notified of the results. There is no reference to the results in the physicians' progress notes or petitioner's discharge summary. The test results appear in the laboratory reports section of the petitioner's medical records. Pet. Ex. 4(d) at 758. During the hearing, upon review of the lab reports, it was noted that the swab PCR ("miscellaneous fluid") was performed at LabCorp, an outside laboratory. Pet. Ex. 4(d) at 758; Transcript ("Tr.") 170. After the hearing, petitioner obtained the report from LabCorp, which notes that the test was drawn on December 23, 2014 and reported on December 30, 2014. The results were positive for VZV; "varicella zoster virus DNA [was] detected." Pet. Ex. 39 at 2.

iii. Petitioner's Testimony

Mr. Dunn was born October 24, 1970. He testified that as a child he had chickenpox. Tr. 30. He received the Tdap vaccine at issue in this case on December 2, 2014. Tr. 8.

On December 12, 2014, Mr. Dunn started having headaches. Tr. 11. On the evening of December 18, 2014, he had nausea, dizziness, fever, and chills. Id. The next day, December 19, 2014, he felt ill, and left work and drove home at noon. Tr. 12. When he arrived home, he went upstairs to lie down. Id. That is his last memory until December 25, 2014, when he came out of a coma. Tr. 16.

[12] Varicella virus testing described in this decision includes serological antibody testing of peripheral blood, and viral DNA PCR testing of cerebrospinal fluid and of a fluid filled vesicle. Petitioner's VZV IgG test was positive, indicating that he tested positive for antibodies to the varicella virus. See Mosby's Manual of Diagnostic and Laboratory Tests 712-75 (Kathleen D. Pagana & Timothy J. Pagana eds., 6th ed. 2018). Varicella virus DNA proteins can be detected by PCR. Id. at 713. Here, petitioner had PCR analysis of his CSF on December 20, and then of fluid in a vesicle on December 23. The PCR on the CSF was negative. The PCR of the vesicle fluid was positive.

Based on information shared with him by his wife, petitioner testified that on December 19, 2014, when Ms. Dunn arrived home, she found her husband ill, and she called 911. Tr. 12. Mr. Dunn was taken by ambulance to AGH. Id. Mr. Dunn explained that he had not told his wife that he received the Tdap vaccine, and she did not inform petitioner's health care providers of his history of Tdap vaccination when petitioner was taken to the hospital. Tr. 14.

Petitioner was questioned regarding Dr. Pae's medical records, specifically, the entry dated December 2, 2014, which documented that petitioner had a truncal rash. See Pet. Ex. 5(b) at 371. Petitioner explained that he told Dr. Pae that in the past he had taken medication for the rash and it cleared up. Tr. 24. Dr. Pae's records also note that petitioner had a separate lesion on the right arm which was "different and itchy." Id.; Pet. Ex. 5(b) at 371. Dr. Pae stated that Mr. Dunn had skin problems in the past, but this was a new problem. Pet. Ex. 5(b) at 371.

Petitioner testified that he had a history of sensitive skin, and a nickel allergy, along with a long history of skin allergies. Tr. 29. He testified, however, that he had never sought treatment for a painful rash. Tr. 30. Petitioner also testified that he did not have a painful skin rash between December 2, 2014 and December 19, 2014. Tr. 10.

Significantly, Mr. Dunn testified that while he was a patient at AGH, no one ever told him that he tested positive for shingles (VZV). Tr. 31. Further, when he was discharged from the hospital, he was not informed of his positive shingles test or VZV diagnosis. Tr. 21. Petitioner did not become aware that he had a positive PCR test for shingles until his lawyer told him during the pendency of this claim. Id.

iv. Contemporaneous Treating Physician Assessments

During petitioner's admission to AGH in December 2014, he was initially seen by an emergency room physician and then followed by several specialists, including neurology, infectious disease, and while in the ICU, critical care physicians. The following is a chart with the daily assessments charted by these physicians.

Date	Clinical impression	Pet. Ex. 4(a) at
12/19/14	Initial progress note – altered mental status (AMS) encephalitis/meningitis/intoxication . . . intoxication unlikely	9
12/20	Likely viral meningoencephalitis	10
12/20	Most likely infectious	16
12/20	? etiology appears to have viral infection. LP WBC 16	22
12/21	Could be viral meningitis	12
12/21	Etiology unclear ? nicotine toxicity ? viral encephalitis	14
12/21	Possible meningoencephalopathy but also consistent with toxic exposure	15
12/22	Concern for infectious etiology	25
12/22	Infectious vs. encephalopathy	25
12/22	Likely viral encephalitis	32

12/23	ID noted some vesicles left chest, LUE… shingles/herpetic lesions should be ruled out	36
12/23	Aseptic meningitis CT [continue] Acyclovir follow PCR	37
12/24	AMS – unclear etiology most likely viral etiology	50
12/25	AMS – unclear etiology. Likely viral	52
12/25	AMS 2/2 [secondary to] HSV encephalitis ? CSF HSV PCR negative	54
12/26	Encephalitis 2/2 [secondary to] viral ?	57
12/26	Encephalopathy resolved. Abnormal CSF w/ viral pleocytosis	59
12/26	Encephalopathy resolved, suspect nicotine overdose initially followed by withdrawal	60
12/27	AMS secondary to aseptic meningitis	62

B. Expert Qualifications and Opinions

i. Expert Qualifications

a. Petitioner's Expert, Dr. George Allen Small

Dr. George Allen Small is a board-certified neurologist and is the Director of Allegheny General Hospital's EMG Laboratory and Neuromuscular Service. Pet. Ex. 9 at 5-6. Dr. Small is also an Associate Professor of Neurology at Drexel University School of Medicine and at the Temple University School of Medicine. Id. at 5; Tr. 37. Dr. Small received his M.D. from Jefferson Medical College and completed his residency at the Neurological Institute of New York. Pet. Ex. 9 at 5. He also serves as the Program Director for the Clinical Neurophysiology Fellowship. Id. Dr. Small has published numerous articles. Id. at 9-11.

Dr. Small has treated hundreds of patients with encephalitis, and of those, most did not have a recognized or known etiology for their illness. He testified that the most common viral cause is the herpes virus type one (HSV). Tr. 102. Dr. Small has also seen cases of postvaccination encephalitis, but "not very many." Id.

Dr. Small has privileges to practice at AGH, the hospital where petitioner was admitted and received care in December 2014, but he does not recall caring for or treating petitioner. Tr. 111

b. Respondent's Expert, Dr. Subramaniam Sriram

Dr. Subramaniam Sriram is a board-certified neurologist with a focus in neuroimmunology. See Resp. Ex. Q at 2. He obtained a Bachelor of Medicine and a Bachelor of Surgery from the University of Madras in Madras, India. Id. at 1. He then served as an intern and resident at Wayne State University and completed a residency in neurology at Stanford University, where he also served as chief resident and eventually completed a post-doctoral fellowship in neuroimmunology. Id. Currently, Dr. Sriram serves as the William Weaver Professor of Experimental Neurology at Vanderbilt University Medical Center. Id. at 2; Tr. 140.

He also holds a joint appointment as Professor of Pathology, Microbiology, and Immunology. Resp. Ex. Q at 2. Dr. Sriram's clinical practice includes seeing patients two days a week. Tr. 140.

Like Dr. Small, Dr. Sriram has treated patients with meningoencephalitis, including cases caused by VZV. He has also authored a paper about a patient with VZV encephalitis. Resp. Ex. Q at 19; Tr. 142. That patient had CSF findings consistent with VZV, but had abnormal eye movements (ocular findings) and did not present with encephalitis or meningitis. Tr. 142. Of note, two to three weeks later, the patient developed a shingles lesion. Id.

ii. Expert Opinions

a. Petitioner's Expert, Dr. Small[13]

Dr. Small opined that petitioner's meningoencephalitis was due to a reaction to the Tdap vaccine that he received on December 2, 2014. Tr. 73-74, 106. Dr. Small defined meningoencephalitis as altered mental status for greater than twenty-four hours with "objective evidence of inflammation in the brain" and spinal fluid. Tr. 60. He further explained that meningoencephalitis encompasses aseptic meningitis (inflammation of meninges as evidenced by cerebrospinal fluid analysis) and encephalitis (inflammation of the brain as evidence by dysfunction of the brain). Tr. 69. He further explained that aseptic meningitis also suggests that no viral or bacterial organism is found in the spinal fluid. Tr. 70-71.

Petitioner's cerebrospinal fluid showed elevated white blood cells and protein. Pet. Ex. 4(d) at 741. Cultures and PCR testing of the CSF did not reveal a specific virus or bacteria. Tr. 50, 55-56. Dr. Small explained that the CSF was "reactive," meaning that the tests suggested an inflammatory and immunological reaction to some agent. Tr. 52. However, according to Dr. Small, the CSF results ruled out the possibility of active viral or bacterial meningoencephalitis. Tr. 55-57.

With regard to Althen Prong One, Dr. Small posits that the Tdap vaccine causes the following immunological reaction: The bacterial antigen in the vaccine initiates an immune response (T cells interacting with B cells) that results in the production of protective antibodies against the bacterial antigen. Tr. 76; Pet. Ex. 9 at 2. The immune response also results in the production of proinflammatory cytokines, including interleukin 6 (Il-6), and tumor necrosis factor-α (TNF-α). Tr. 76. According to Dr. Small, these cytokines break down the blood brain barrier, which normally protects the brain from noxious material, and allows for inflammation of the central nervous system and brain to occur. Tr. at 77-78. The inflammation causes abnormal function, including altered mental status. Id. Dr. Small testified that the inflammation causes damage to the brain, and the waste products of that damage elevate the protein level in the cerebrospinal fluid. Tr. at 80. The "immunization-promoted inflammatory response becomes exuberant and spills over into the central nervous system." Pet. Ex. 9 at 3.

[13] Petitioner filed six expert reports authored by Dr Small. Pet. Exs. 9, 10, 13, 19, 22, 38.

Dr. Small cited several medical articles in support of his proposed causal mechanism. He cited the Kashiwagi[14] article to support his position that cytokines IL-6 and TNF-α are produced in response to the DPT, Hib, and PCV vaccines and that "brain cells can be abrogated by inflammation." Tr. 81-84; see Pet. Ex. 25 at 3. Dr. Small explained that the Rochfort[15] article also describes how cytokines IL-6 and TNF-α cause a breakdown of "tight junctions" that keep bacteria and toxins away from the central nervous system. Tr. 87-88; Pet. Ex. 26. Dr. Small testified that "substances get into parts of the brain where they shouldn't be" due to the presence of these cytokines. Tr. 88.

Dr. Small also cited Hiraiwa-Sofue,[16] a case report of a child who had encephalitis caused by a pertussis infection. Tr. 89; Pet. Ex. 27 at 3. The child had a seizure and encephalopathy eighteen days after the onset of a pertussis infection. MRI showed "marked demyelination." Pet. Ex. 27 at 1. There was no evidence of direct infection of the pertussis bacteria in the central nervous system. The authors suggested several possible mechanisms, including immune mediation, proinflammatory cytokine production, pertussis toxin effect on the blood brain barrier, and inflammation of the central nervous system. Id. at 3. The authors also stated that proinflammatory cytokines were "associated with the development of acute encephalitis." Id. They also noted that levels of IL-6 and another cytokine, IL-10, were higher in the CSF than in the blood, suggesting they played a role in the cause of encephalitis. Id.

Another article cited by Dr. Small is Aydin,[17] a case report of an infant who presented with seizures and abnormal neurological findings six days after receipt of the whole cell DTP vaccination. Pet. Ex. 28. The child was diagnosed with acute necrotizing encephalopathy. Possible causal mechanisms included breakdown of the blood brain barrier, "alteration of vessel wall permeability," or "vessel wall necrosis." Pet. Ex. 28 at 2. The authors hypothesized that the vaccine caused increased levels of IL-6 and TNF-α which may have altered "vessel wall permeability and local breakdown of the [blood brain barrier]." Id.

Based on the articles cited, Dr. Small testified that an active infection with bacteria is not required in order to have meningoencephalitis; cytokines may be elevated in the cerebrospinal

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[14] Yasuyo Kashiwagi et al., Production of Inflammatory Cytokines in Response to Diphtheria-Pertussis-Tetanus (DPT), Haemophilus Influenzae Type b (Hib), and 7-Valent Pneumococcal (PCV7) Vaccines, 10 Hum. Vaccines & Immunotherapeutics 667 (2014).

[15] K.D. Rochfort et al., Downregulation of Blood-Brain Barrier Phenotype by Proinflammatory Cytokines Involves NADPH Oxidase-Dependent ROS Generation: Consequences for Interendothelial Adherens and Tight Junctions, 9 PLoS One e101815 (2014).

[16] Ayako Hiraiwa-Sofue et al., Pertussis-Associated Encephalitis/Encephalopathy with Marked Demyelination in an Unimmunized Child, 320 J. Neurological Sci. 145 (2012).

[17] Hale Aydin et al., Acute Necrotizing Encephalopathy Secondary to Diphtheria, Tetanus Toxoid and Whole-Cell Pertussis Vaccination: Diffusion-Weighted Imaging and Proton MR Spectroscopy Findings, 40 Pediatric Radiology 1281 (2010).

fluid which cause encephalopathy.[18] Tr. 89-91. According to Dr. Small, it is the promotion of cytokines, not bacteria, that breaks down the blood brain barrier, causing meningoencephalitis. See Tr. 97.

Next, Dr. Small opined regarding Althen Prong Two, the logical sequence of cause and effect—how the Tdap vaccine caused petitioner's meningoencephalitis. Dr. Small testified that prior to vaccination, the petitioner was generally healthy. Petitioner received the vaccine on December 2, 2014, and approximately fifteen to sixteen days later became lethargic and unresponsive. Tr. 99; Pet. Ex. 9 at 3. He was admitted to the hospital where diagnostic testing of his cerebrospinal fluid showed a "clear aseptic meningitis picture" and he was diagnosed with meningoencephalitis. Tr. 99. Again, according to Dr. Small, aseptic meningitis indicates an inflammatory reaction in the spinal fluid. Tr. 64-65. In support of his opinions, Dr. Small cited the petitioner's treating neurologist who stated that petitioner had meningoencephalitis of "unknown cause." See Pet. Ex. 4(a) at 130-31; Tr. 56. Dr. Small also cited a reference in petitioner's medical record stating that petitioner had aseptic meningitis, which Dr. Small explained indicated an inflammatory reaction in the spinal fluid without the presence of virus or bacteria. Tr. 64-65.

Dr. Small further opined that while test results were pending, petitioner was treated empirically "for the most common causes of infectious encephalitis" with antivirals and antibiotics. Tr. 99. Petitioner had respiratory depression, required a bronchoscopy, but then improved. Tr. 100. After discharge, he had depression and cognitive issues. Tr. 100. Dr. Small opined that the only "clear and inciting event" was "the proximate relationship of the Tdap vaccine." Id. He opined that "[c]onsidering Mr. Dunn's prior medical history and subsequent medical workup, there is no other suggestive or explainable cause for his meningoencephalitis but-for his Tdap vaccination." Pet. Ex. 9 at 3.

On cross-examination, Dr. Small conceded that petitioner here did not have evidence of demyelination or a pertussis infection, both of which distinguish petitioner's case from the case presented in Hiraiwa-Sofue, described above. Tr. 115; Pet. Ex. 27. Dr. Small agreed that having a pertussis infection is very different than receiving the acellular pertussis vaccine that petitioner received. Tr. 115. Dr. Small also conceded that the patient in the Aydin case report had hemorrhagic necrosis of the deep brain, which petitioner here did not have. Id.; Pet. Ex. 28.

With regard to Althen Prong Three, Dr. Small opined that the temporal association between vaccination and onset of petitioner's illness was appropriate. He testified that "[i]t takes time for the inflammatory reaction to develop" and it is not unusual for an immune response like the one he proposes here to take a week or two to occur. Tr. 100-01. The two cases reported in Aydin and Hiraiwa-Sofue had onset of six and eighteen days. Therefore, Dr. Small believes that petitioner's onset of fifteen to sixteen days post-vaccination is within the range of expected onset given his proposed theory. Tr. 103-04.

[18] Dr. Small explained that generally there are no commercial tests available to test for cytokines in cerebrospinal fluid. Tr. 91.

Dr. Small was asked to provide medical literature regarding reports of encephalitis and meningitis following the Tdap vaccination. See Pet. Ex. 13 at 1-2. In response, he cited an article by Dalmau,[19] in which the authors discuss encephalitis associated with antibodies against the N-methyl-D aspartate receptor ("NMDAR"). Pet. Ex. 11. The pathogenic mechanism of the illness is thought to be immune mediated. The article makes a reference to a patient who developed anti-NMDAR encephalitis following receipt of the TDaP-IPV (Polio) booster vaccination. Pet. Ex. 11 at 5. Dr. Small conceded that petitioner here did not have anti-NMDAR encephalitis. Tr. 111.

Dr. Small also referenced the Baxter study.[20] Pet. Ex. 13 at 2; Pet. Ex. 18. That article describes acute demyelinating events following vaccines, including acute disseminated encephalomyelitis ("ADEM") after the Tdap vaccine. Dr. Small conceded that petitioner did not have ADEM, and that petitioner's MRI did not show any evidence of demyelination. Tr. 78, 97, 111-12, 114.

Dr. Small conceded that there was no epidemiology support for an association between the Tdap vaccine and meningoencephalitis. Tr. 114. In an article cited by Dr. Small, authored by Singh,[21] the most common cause of encephalitis in 139 cases was viral infection (48%), followed by autoimmune (22%), and unknown/other (30%). Pet. Ex. 37 at 2. The most common viral infection was herpes simplex virus (HSV) at 38.9%, followed by varicella zoster virus (VZV) at 23.2%. Id. at 3. Vaccines were not listed in the Singh article as a cause of encephalitis.

As for respondent's position that petitioner's illness was caused by reactivation of a prior varicella infection, Dr. Small testified that in order to attribute petitioner's illness to varicella reactivation, he would need evidence that petitioner was "infected with varicella-zoster at the time of his neurological presentation." Tr. 107-08. Also, Dr. Small does not believe that varicella was the cause of petitioner's meningoencephalitis because his CSF tested negative for the virus. Tr. 107. Additionally, the note by Dr. Hong, stating that the vaccine was the cause of petitioner's illness, influenced Dr. Small's opinion. Tr. 108.

On cross-examination, Dr. Small conceded that varicella infection can involve the central nervous system. Tr. 112. He also agreed that a patient can first have central nervous system involvement with a varicella infection without having any skin lesions. Id. When asked whether a patient can have central nervous system involvement with a varicella infection followed by

[19] Josep Dalmau et al., Clinical Experience and Laboratory Investigations in Patients with Anti-NMDAR Encephalitis, 10 Lancet Neurology 63 (2011) (Petitioner filed this article as Exs. 11 and 16).

[20] Roger Baxter et al., Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis, 63 Clinical Infectious Diseases 1456 (2016).

[21] Tarun D. Singh et al., The Spectrum of Acute Encephalitis: Causes, Management, and Predictors of Outcomes, 84 Neurology 359 (2015).

skin lesions, Dr. Small answered that he did not know. He deferred to an infectious disease physician to answer the question. Id.

Dr. Small confirmed that the petitioner's vesicular skin lesions were swabbed on December 23, 2014, and that the PCR analysis was positive for the varicella zoster virus. Tr. 130. He did not know how long it took to obtain the results of the PCR test. Id.

Importantly, Dr. Small testified that petitioner's clinical course was "completely consistent" with meningoencephalitis caused by varicella. Tr. 123. He also agreed that respondent's theory of causation, varicella infection reactivation, is a known cause of encephalitis. Tr. 124. Dr. Small described the mechanism of reactivation very similarly to the explanation given by Dr. Sriram. Compare Pet. Ex. 13 at 1-2, with Resp. Ex. E at 1.

Further, Dr. Small agreed that petitioner's cerebrospinal fluid showed an elevated white blood cell count and elevated protein, and that patients with varicella infection can have these findings. Tr. 120. Dr. Small also agreed that petitioner's varicella zoster IgG was elevated. Tr. 118-19. Initially, he testified that petitioner's clinical course was milder than patients he has seen with varicella infection and that his patients with the illness had higher white blood cell counts, more elevated protein cerebrospinal fluid counts, shingles along the course of nerves in the head, and significant brain swelling. Tr. 123-27. However, he later admitted that he also had patients with a milder clinical course, like that of petitioner. Tr. 128.

Lastly, Dr. Small discussed whether AGH and the VA have the ability to share a patient's electronic medical records. He testified that as recent as three months ago it was impossible for the two hospitals to share records electronically and this has made treating patients "very difficult." Tr. 119-20.

b. Respondent's Expert, Dr. Sriram[22]

Dr. Sriram agrees with Dr. Small as to petitioner's diagnosis of meningoencephalitis but does not believe that petitioner's Tdap vaccine caused his illness. Tr. 147. Instead, Dr. Sriram opines that reactivation of petitioner's VZV infection caused his meningoencephalitis. Tr. 146; Resp. Ex. A at 3.

Dr. Sriram defined meningoencephalitis as "a clinical diagnosis based to some extent on [] laboratory data." Tr. 146. A patient will have altered mental status which may be associated with focal deficits such as hand or leg weakness, speech problems, or seizures. Tr. 147. The cerebrospinal fluid may show elevated lymphocytes and protein which indicate inflammation of the meninges. Id.

With regard to petitioner's theory of causation, Dr. Sriram disagreed with Dr. Small that the Tdap vaccine can cause a cytokine reaction which increases the permeability of the blood brain barrier. Dr. Sriram testified that there is no evidence to support this hypothesis. See Tr. 152. Further, Dr. Sriram did not agree that the medical articles cited by Dr. Small support

[22] Respondent filed six expert reports authored by Dr. Sriram. Resp. Exs. A, E, M, N, P, S.

petitioner's causal theory. Dr. Sriram explained that the Kashiwagi study was done to examine the effects of the DPT, Hib, and PCV7 vaccines on lymphocytes in peripheral blood. Tr. 148. Since the article has nothing to do with the brain, Dr. Sriram does not believe that it supports Dr. Small's theory as it relates to the effect of cytokines on the blood brain barrier. Id. Moreover, Dr. Sriram testified that in Kashiwagi, the DTP vaccine decreased cytokine levels, except for a marginal increase of IL-6. Id.; Pet. Ex. 25 at fig.1. On cross-examination, however, Dr. Sriram agreed that the DTP vaccine caused an elevation of cytokines TNF-α and IL-6, and that he was previously mistaken when he testified that there was a decrease in cytokines. Tr. 210.

As for the Rochfort article cited by petitioner, Dr. Sriram noted that it was an in vitro experiment, and thus, it does not speak to what may happen in humans. Tr. 150-52. Rochfort was an in vitro experiment where human microvascular endothelial cells were treated with cytokines IL-6 and TNF-α, and then studied. Pet. Ex. 26. According to Dr. Sriram, very large amounts of cytokines were used. Tr. 150-52. Dr. Sriram testified that researchers have not studied whether the blood brain barrier is permeable if a person is injected with high doses of cytokines. See Tr. 152.

Next, Dr. Sriram explained why the Hiraiwa-Sofue and Aydin articles do not support petitioner's theory. He opined that Hiraiwa-Sofue is not relevant because petitioner did not have evidence of demyelination, and petitioner is not pursuing a theory based on demyelination. Tr. 153. The Aydin study involved whole cell pertussis, and here petitioner did not receive a vaccine that contained whole cell pertussis, only acellular pertussis. Tr. 154. Moreover, petitioner here did not have hemorrhage necrotizing encephalitis, like the patient in Aydin. Id.

Dr. Sriram emphasized that none of the literature cited by petitioner states that the Tdap vaccine can cause a syndrome even close to what petitioner had—Mr. Dunn did not have ADEM or NMDAR encephalitis. Tr. 155. Further, Dr. Sriram did not agree that cytokines can cause encephalitis. Tr. 214. He explained that cytokines are associated with encephalitis but "are not causally connected to it." Id. Dr. Sriram testified that he has not seen any support in the literature to prove that cytokines cause a breakdown of the blood brain barrier in humans. Tr. 218.

Next, Dr. Sriram turned his focus to testifying in support of respondent's position that petitioner's meningoencephalitis was caused by an alternative factor unrelated to his Tdap vaccine—petitioner's VZV infection. General information about VZV infections is set forth in the Nagel and Gilden article[23] filed by respondent. Resp. Ex. H. VZV is a virus in the herpes family. Primary VZV infection generally occurs in childhood among non-vaccinated children and is known as chickenpox. Id. at 1. After primary infection, the virus becomes latent in nerve ganglia. Id. VZV infection reactivation may cause herpes zoster, also known as shingles. Id. VZV reactivation may also cause vasculopathy, meningoencephalitis, cerebellitis, and other neurological illnesses. Id. "VZV can reactivate and infect the meninges, brain parenchyma and nerve roots to produce a VZV meningoencephalitis." Id. at 4.

[23] Maria A. Nagel & Don Gilden, Neurological Complications of Varicella Zoster Virus Reactivation, 27 Current Opinion Neurology 356 (2014).

With regard to Althen Prong One, a medical theory of causation, Dr. Sriram's opinions were consistent with the information about VZV set forth in Nagel and Gilden. He explained that the herpes zoster virus is present in the ganglion cells of the body. Tr. 156. The virus resides in the neurons of the ganglion cells, and for reasons that are not clearly understood, the virus becomes activated. Id. One trigger for activation is immunosuppression, which is more common in persons as they become older, and also is "classic" in those between ages forty-five to fifty. Id. The virus that resides in the ganglion cells can either "come forward and cause infection of the skin or they go backwards and infect the meninges and the brain." Tr. 157. Persons who had chickenpox have the virus in their nerve cells, and it remains present throughout life. The virus can become activated and travel "proximally into the spinal cord or [] brain or distally out into the skin." Tr. 157-58. It is not uncommon for viral reactivation to cause both encephalitis and meningitis. Id. Dr. Sriram further explained that with herpes zoster reactivation, the virus can affect both the skin and nervous system. Id. VZV is a known cause, and the second leading cause of, viral encephalitis.[24] Resp. Ex. E at 2.

In support of his theory of causation, Dr. Sriram cited several medical articles. A number of which support his opinion that VZV can cause central nervous system illnesses, including encephalitis and meningitis, through the mechanism of reactivation. In Grahn and Studahl,[25] the authors state that VZV is the "second most common infectious etiology of encephalitis after the herpes simplex virus." Resp. Ex. F at 2. The authors describe the mechanism as follows: VZV is a herpes virus that may become latent in neurons in the dorsal root, autonomic, and cranial ganglia. Id. Reactivation of the virus may infect the skin, and central nervous system, causing encephalitis and meningitis. Id. And in Nagel and Gilden, the authors state that VZV can travel to the central nervous system to produce meningoencephalitis, as described by Dr. Sriram. Resp. Ex. H at 1; Tr. 160.

Dr. Small also recognizes the theory of herpes zoster reactivation, and his explanation of the mechanism did not differ from that provided by Dr. Sriram. See Pet. Ex. 13 at 1-2; Tr. 171.

With regard to Althen Prong Two, a logical sequence of cause and effect, Dr. Sriram testified that petitioner had herpes zoster which infected his central nervous system, causing encephalitis and meningitis (meningoencephalitis), via the mechanism of viral reactivation. Tr. 156. Dr. Sriram provided several reasons why the evidence supported his opinion that petitioner's VZV infection was the more likely cause of his meningoencephalitis. First, petitioner had acute encephalopathy and his cerebrospinal fluid revealed elevated lymphocytes and protein consistent with VZV meningoencephalitis. Tr. 163-64. Dr. Sriram cited the

[24] See Don Gilden et al., Varicella Zoster Virus Vasculopathies: Diverse Clinical Manifestations, Laboratory Features, Pathogenesis, and Treatment, 8 Lancet Neurology 731 (2009); T. De Broucker et al., Acute Varicella Zoster Encephalitis Without Evidence of Primary Vasculopathy in a Case-Series of 20 Patients, 18 Clinical Microbiology Infection 808 (2011).

[25] Anna Grahn & Marie Studahl, Varicella-Zoster Virus Infections of the Central Nervous System – Prognosis, Diagnostics and Treatment, 71 J. Infection 281 (2015).

Gregoire study[26] to establish that there can be a wide range in the number of lymphocytes present in the cerebrospinal fluid of patients with meningoencephalitis. Resp. Ex. C at 2 tlb.1. In Gregoire, one patient had 13 and another patient had 358 lymphocytes in their CSF. Id.; Tr. 162-63. Dr. Sriram explained that the presence of and number of lymphocytes (indicating inflammation) in the CSF depends at what point in the illness the spinal fluid is drawn. Tr. 163.

The second basis for Dr. Sriram's Althen Prong Two opinion is that petitioner had a rash on his chest noted December 23, and a swab of the rash tested by PCR was positive for VZV.[27] Dr. Sriram cited medical articles which supported his position that a patient can have central nervous system symptoms with no rash, or the neurological symptoms can be followed by the rash. In Nagel,[28] the authors addressed the issue of whether the herpes zoster rash must be present for the virus to cause illness. Resp. Ex. O. The authors reviewed 30 cases of VZV vasculopathy, which caused stroke due to viral infection of the cerebral arteries. Id. at 1. A herpes zoster rash occurred in 19 out of the 30 cases. The authors concluded that the herpes zoster rash was "not required to diagnose varicella zoster virus vasculopathy." Id.

A positive CSF PCR is not required to find that a patient has VZV encephalitis. The Gilden article states, "[a]lthough a positive PCR for VZV DNA in CSF is helpful, a negative PCR does not exclude the diagnosis . . ." Resp. Ex. T at 3. Dr. Sriram explained that "[w]hile the specificity of [a] PCR assay for VZV is 100% [there are no false positive PCR values], the sensitivity varies." Resp. Ex. E at 2. A PCR done either very early or very late during the clinical course of encephalitis may be negative. Id. A negative PCR study does not exclude the diagnosis of VZV encephalitis. Resp. Ex. N at 2; see also Resp. Ex. T at 3.

Dr. Sriram cited a paper by De Broucker. Resp. Ex. G. There, four out of twenty patients had a negative cerebrospinal fluid PCR for VZV but had a herpes zoster rash before or after onset of acute VZV encephalitis. Id. at 1. As it relates to the timing of the herpes zoster rash, the authors state that "neurological presentations can also appear with, precede, or follow herpes zoster." Id. Dr. Sriram also cited the Grahn and Studahl article, which states that "the central nervous system manifestations might occur before the skin eruptions." Resp. Ex. F at 2.

Dr. Sriram agreed that based on the criteria set forth in De Broucker, petitioner would not have qualified as a "confirmed" case of VZV encephalitis because he did not have a positive cerebrospinal fluid PCR for VZV. Tr. 199. Dr. Sriram affirmed that in De Broucker, the authors

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[26] S. S. Gregoire et al., Polymerase Chain Reaction Analysis and Oligoclonal Antibody in the Cerebrospinal Fluid from 34 Patients with Varicella-Zoster Virus Infection of the Nervous System, 77 J. Neural Neurosurgery Psychiatry 938 (2006).

[27] Dr. Sriram believes it is likely that petitioner had the zoster rash before December 23, but it was not detected. Tr. 164, 225. However, even if the zoster rash was not present until it was seen on December 23, Dr. Sriram's opinion would be the same. Tr. 225.

[28] Maria A. Nagel et al., The Varicella Zoster Virus Vasculopathies: Clinical, CSF, Imaging, and Virologic Features, 70 Neurology 853 (2008).

stated that the only way to confirm VZV is by a positive CSF PCR or evidence of antibodies to VZV.[29] Tr. 201. In Arruti,[30] the authors distinguish between "confirmed" and "probable" or "suspected" VZV-caused illness. Resp. Ex. J at 2. While a confirmed case is defined as one with positive cerebrospinal fluid PCR testing, a patient probably has VZV infection if there is a simultaneous rash or a rash that occurs shortly after illness onset and there are inflammatory markers in the CSF. Tr. 203.

In Arruti, the authors studied the clinical characteristics of central nervous system infections caused by VZV in 280 older patients (over age 65). Resp. Ex. J at 1. The authors discussed the incidence of varicella zoster central nervous system infections, stating that "VZV is a common viral agent causing central nervous system infections in the adult population, using ranking behind HSV." Id. at 7. The authors also discuss the incidence of skin lesions in the context of central nervous system infections caused by VZV:

> Previous studies have shown that neurological complications of HZ [herpes zoster] can occur without skin lesions, such complications being observed in 38 to 69% of patients with no skin involvement (cites omitted). In our study, only 16.7% of patients had no signs of skin involvement. Hence, our data indicate that HZ [herpes zoster] occurring simultaneously or shortly after CNS [central nervous system] neurological symptoms suggests causality in a high percentage of cases in the elderly. . . . In the eight patients with probable VZV encephalitis and no viral confirmation [PCR negative for VZV], the clinical syndrome in combination with skin involvement [the presence of an HZ rash] together with imaging and/or cerebrospinal fluid findings supported the diagnosis and suggested a causative link.

Id. Thus, based on Arruti, Dr. Sriram opines that petitioner would fall into the probable category. Tr. 203.

Lastly, Dr. Sriram testified that petitioner's elevated VZV titer >4000, was a value considered to be a striking elevation and confirmed recent activation of the varicella virus.[31] Tr. 167-70.

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[29] Dr. Sriram filed the Gregoire article to support his testimony that if a PCR of cerebrospinal fluid is negative for VZV, an anti-VZV specific antibody test may be performed to confirm diagnosis, but this test was not ordered for petitioner. Tr. 161-62; Resp. Ex. C at 1.

[30] Maialen Arruti et al., Incidence of Varicella Zoster Virus Infections of the Central Nervous System in the Elderly: A Large Tertiary Hospital-Based Series, 23 J. Neurovirology 451 (2017).

[31] Dr. Sriram testified that although the VZV titer is high, because he does not know petitioner's baseline level, this fact alone does not form the basis of his causation opinion but is consistent with it. Tr. 224.

When asked how long it would take to perform swab PCR test, Dr. Sriram answered that if the test is sent to an outside laboratory, it would take three to five days to obtain the results. Tr. 170. Dr. Sriram testified that based on petitioner's medical records, it appeared that the PCR swab test was sent out LabCorp in North Carolina on December 23. See Pet. Ex. 4(d) at 758.

As for Althen Prong Three, Dr. Sriram opined that the timing between vaccination and illness onset was appropriate for the VZV to cause petitioner's meningoencephalitis. Tr. 170-71. Dr. Sriram emphasized that when a person with shingles and altered mental status also has cerebrospinal fluid that shows inflammation, "the preponderance of the clinical opinion would be this is a zoster meningoencephalitis." Tr. 171. Dr. Sriram cited the study by Science,[32] which studied eighty-four children with VZV neurological complications. Resp. Ex. U at 2. Children were included in the study if the onset of their neurological symptoms occurred four weeks before or after the VZV rash. Id. The "median interval between onset of rash and onset of neurological manifestations was 5 days (range 6 days before to 16 days after the appearance of the rash)." Id. at 3.

On cross-examination, Dr. Sriram was questioned about the note made by Dr. Hong, one of petitioner's treating physicians at the VA. Dr. Sriram did not agree that the following note by Dr. Hong was an opinion that the Tdap vaccine caused petitioner's illness. On February 4, 2015, the title of Dr. Hong's note is "Allergy & Immunology Adverse Event Note." Pet. Ex. 5(b) at 296. It states that petitioner "was admitted to AGH on 12/19/2014 with AMS. LP shows aseptic meningitis result. Had Tdap vaccine on 12/2/2014." Id. Dr. Sriram did agree that this note and other entries in the VA records referenced that petitioner had an allergy or adverse drug reaction to Tdap. Tr. 188.

Dr. Sriram explained that according to the AGH records, the PCR swab test of petitioner's vesicle was sent out for testing on December 23. December 25 was a holiday, and petitioner was discharged on December 27.[33] Dr. Sriram testified that it was "highly unlikely" that the PCR test results (positive for VZV) would have come back the day after Christmas in time for someone to review it and make an assessment or recommendation about the diagnosis prior to the patient's discharge.[34] Tr. 189.

[32] Michelle Science et al., Central Nervous System Complications of Varicella-Zoster Virus, 165 J. Pediatrics 779 (2014).

[33] During his testimony, Dr. Sriram stated petitioner was discharged on December 26. Tr. 189. Petitioner was actually discharged on December 27.

[34] After the hearing, the PCR swab test results from LabCorp were filed. Pet. Ex. 39. They showed the test report date was December 30, 2014. Thus, it appears Dr. Sriram was correct that the results were not available before the petitioner's hospital discharge on December 27.

V. LEGAL FRAMEWORK

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 300aa-10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Human Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 300aa-13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Human Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Human Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, petitioners must prove that that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Human Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); Pafford v. Sec'y of Health & Human Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner who satisfies this burden is entitled to compensation unless respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 300aa-13(a)(1)(B).

B. Causation

To receive compensation under the Program, petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 300aa-13(a)(1)(A) and 11(c)(1); Capizzano v. Sec'y of Health & Human Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because petitioner does not allege that he suffered a Table injury, he must prove that the vaccine he received caused his injury. To do so, he must establish, by preponderant evidence: (1) a medical theory causally connecting the vaccine and injury ("Althen Prong One"); (2) a logical sequence of cause and effect showing that the vaccine was the reason for the injury ("Althen Prong Two"); and (3) a showing of a proximate temporal relationship between the vaccine and injury ("Althen Prong Three"). § 300aa-13(a)(1); Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Thus, petitioner must provide a reputable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on assertions. Rather, a vaccine claim must

be supported either by medical records or by the opinion of a medical doctor. § 300aa-13(a)(1). In determining whether petitioner is entitled to compensation, the special master shall consider all material contained in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 300aa-13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' offered experts and rule in petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence"); see also Althen, 418 F.3d at 1280 (providing "close calls" are resolved in petitioner's favor).

Another important aspect of the causation-in-fact case law under the Vaccine Act concerns the factors that a special master should consider in evaluating the reliability of expert testimony and other scientific evidence relating to causation issues. In Daubert v. Merrell Dow Pharmacy, Inc., 509 U.S. 579 (1993), the United States Supreme Court listed certain factors that federal trial courts should utilize in evaluating proposed expert testimony concerning scientific issues. In Terran v. Secretary of Health & Human Services., 195 F.3d 1302, 1316 (Fed. Cir. 1999), the Federal Circuit ruled that it is appropriate for special masters to utilize Daubert's factors as a framework for evaluating the reliability of causation-in-fact theories actually presented in Program cases.

The Daubert factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community. Terran, 195 F.3d at 1316, n.2 (citing Daubert, 509 U.S. at 592-95). In addition, where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." Broekelschen v. Sec'y of Health & Human Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing Lampe v. Sec'y of Health & Human Servs., 219 F.3d 1357, 1362 (Fed. Cir. 2000)). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the ipse dixit of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." Snyder v. Sec'y of Health & Human Servs., 88 Fed. Cl. 706, 743 (2009) (quoting Gen. Elec. Co. v. Joiner, 522 U.S. 146 (1997)).

In deciding the issues in this case, the undersigned has considered the record as a whole. § 300aa-13(a)(1). The undersigned has reviewed and relied on statements in the medical records, as medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras v. Sec'y of Health & Human Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). In addition, the treating physicians' opinions are "quite probative," as treating physicians are in the "best position" to evaluate the vaccinee's condition. Capizzano, 440 F.3d at 1326. However, no treating physician's views bind the special master, per se; rather, their views should be carefully considered and evaluated. § 300aa-13(b)(1); Snyder, 88 Fed. Cl. at 745 n.67. Each opinion from a treating physician should be weighed against other, contrary evidence present in the record—including conflicting opinions

from other treating physicians. Hibbard v. Sec'y of Health & Human Servs., 100 Fed. Cl. 742, 749 (2011), aff'd, 698 F.3d 1355 (Fed. Cir. 2012); Caves v. Sec'y of Health & Human Servs., 100 Fed. Cl. 119, 136 (2011), aff'd, 463 F. App'x. 932 (Fed. Cir. 2012); Veryzer v. Sec'y of Health & Human Servs., No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), mot. for rev. denied, 100 Fed. Cl. 344 (2011).

C. Alternative Causation

A petitioner who satisfies his burden of proof under the standards set forth above is entitled to compensation unless respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 300aa-13(a)(1)(B). Therefore, in this case, even if petitioner met his burden of proof on causation, he is not entitled to compensation where the respondent has proven by preponderant evidence that a factor unrelated to the vaccination caused his meningoencephalitis. See Doe v. Sec'y of Health & Human Servs., 601 F.3d 1349, 1358 (Fed. Cir. 2010) ("[Petitioner] Doe never established a prima facie case, so the burden (and attendant restrictions on what 'factors unrelated' the government could argue) never shifted."). The Vaccine Act provides that "factors unrelated to the administration of the vaccine," are those "which are shown to have been the agent . . . principally responsible for causing the petitioner's illness, disability, injury, condition or death." § 300aa-13(a)(2)(B).

In Munn, the Federal Circuit affirmed the special masters' findings of alternate causation. The special master "concluded that '[petitioner] succumbed to infection' and thus that the pneumonia led to her death" not the encephalopathy alleged to be caused by the vaccination. Munn v. Sec'y of Health & Human Servs., 970 F.2d 863, 872 (Fed. Cir. 1992).

Here, respondent has identified an alternative cause of petitioner's illness: Reactivation of VZV infection. Thus, the undersigned undertakes an analysis based on the Althen prongs as to whether VZV reactivation—a factor unrelated to petitioner's vaccination—caused petitioner's meningoencephalitis. See Forrest v. Sec'y of Health & Human Servs., No. 14-1046V, 2019 WL 925495, at *1 (Fed. Cl. Spec. Mstr. Jan. 28, 2019) (finding respondent showed by preponderant evidence that a VZV infection was responsible for petitioner's injury).

VI. Alternative Factor Analysis

A. Althen Prong One: Respondent's Medical Theory

Under Althen Prong One, respondent must set forth a medical theory explaining how VZV infection reactivation can cause meningoencephalitis. Andreu v. Sec'y of Health & Human Servs., 569 F.3d 1367, 1375 (Fed. Cir. 2009); Pafford, 451 F.3d at 1355-56. The theory of causation must be informed by a "sound and reliable medical or scientific explanation." Knudsen, 35 F.3d at 548; see also Veryzer v. Sec'y of Health & Human Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 300aa-13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If respondent relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion.

See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Human Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) ("Expert opinion is no better than the soundness of the reasons supporting it." (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

Respondent's proposed mechanism of causation is varicella zoster viral reactivation. The mechanism is not disputed by petitioner's expert. Further, the experts do not disagree with petitioner's diagnosis, meningoencephalitis, and they both agree that VZV infection can cause this illness. Respondent's proposed mechanism is a sound and reliable explanation of pathogenesis as illustrated by the testimony of both experts and the medical literature filed by respondent. Therefore, the undersigned finds that respondent has established by preponderant evidence that VZV reactivation can cause meningoencephalitis.

B. Althen Prong Two: Logical Sequence of Cause and Effect

Under Althen Prong Two, respondent must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). Respondent must show "that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (citations omitted).

The central issue here is not whether VZV reactivation can cause meningoencephalitis, but whether it did in petitioner's case. The undersigned finds that respondent has proven by preponderant evidence that it did for the following reasons.

Petitioner had chickenpox as a child. His clinical course was consistent with meningoencephalitis caused by VZV. Petitioner had altered mental status, his CSF analysis was consistent with inflammation caused by a viral infection, he had an abnormal and elevated VZV titer, and he had skin vesicle which tested positive for the virus. Moreover, the medical literature shows that next to the herpes simplex virus, VZV is the second leading cause of viral meningoencephalitis. And further, Dr. Small agreed that petitioner's clinical course was "completely consistent" with VZV infection. Tr. 123.

Dr. Small testified that in order to diagnose petitioner with VZV caused illness, he would need to see evidence that petitioner was infected with the virus at the time he presented with neurological symptoms. When asked whether a patient could first have central nervous system involvement, followed by a skin rash, Dr. Small stated that he did not know. Medical literature filed by respondent answered the question. Articles by De Broucher, Grahn and Studhal, and Arruti all state that neurological manifestations may occur before the skin vesicles of herpes zoster appear. The fact that several articles address this issue suggest that the factual circumstances here, where the rash occurs after the neurological symptoms, is not unique to this petitioner.

Petitioner's onset of altered mental status was December 19, and his herpes zoster rash was present on December 23, while petitioner was still encephalopathic. Thus, he had

neurological symptoms and a rash at the same time. The Arruti article speaks to this exact scenario. Their patient data indicated that the herpes zoster rash occurred either "simultaneously or shortly after CNS [central nervous system] neurological symptoms." Resp. Ex. J at 7.

In addition to the support from medical articles, the petitioner's medical records also offer support for Dr. Sriram's position that the rash can follow central nervous system symptoms. On December 23, an infectious disease physician charted that, "skin lesions in VZV encephalitis may develop after AMS even during [treatment]." Pet. Ex. 4(a) at 35. This contemporaneous note is persuasive evidence in support of respondent's position.

With regard to medical record entries by Dr. Hong and Dr. Pae, attributing petitioner's illness to the Tdap vaccine, the undersigned finds these references are not persuasive evidence of causation based on the totality of the facts and circumstances. Dr. Hong and Dr. Pae could not have been informed by the petitioner that he had a positive skin test for herpes zoster, because at the time the petitioner saw these doctors, he did not know that information. Neither of these physicians saw nor treated the petitioner while he was a patient at AGH. Dr. Hong and Dr. Pae treated the petitioner after his discharge from AGH, when he presented for follow-up care at the VA. Neither of these physicians reference the positive PCR skin test results in their records. Based on Dr. Small's testimony, it would have been difficult, if not impossible, for them to review the petitioner's AGH record. Tr. 119. If Dr. Hong or Dr. Pae had reviewed the petitioner's positive VZV skin test results, which was very important and relevant data that could inform their opinions as to causation, it is likely that at least one of them would have noted the results. Lastly, the LabCorp report date is December 30, three days after petitioner's discharge from AGH. Based on the available record, it does not appear the report was seen by either Dr. Hong or Dr. Pae.

The undersigned generally finds opinions of a treating physician to be persuasive evidence of causation, but for all of the reasons stated above, Dr. Hong's statement about Tdap causation is not persuasive here. The far more sound and reliable evidence as to causation specific to petitioner's case is that offered by respondent and Dr. Sriram.

The undersigned does find the contemporaneous records created by the physicians at AGH to be persuasive. On approximately twelve occasions, the treating physicians suggest that the etiology of petitioner's illness is "likely viral" or "infectious." See, e.g. Pet Ex. 4(a) at 9-62. This information, along with all of the reasons discussed above, combine to provide preponderant evidence that petitioner's illness was caused by VZV infection reactivation.

C. Althen Prong Three: Proximate Temporal Relationship

Under Althen Prong Three, respondent must provide "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." De Bazan v. Sec'y of Health & Human Servs., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The acceptable temporal association will vary according to the particular medical theory advanced in the case. See Pafford, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does not constitute preponderant evidence of vaccine causation. See, e.g., Veryzer, 100 Fed. Cl. at 356

(explaining that "a temporal relationship alone will not demonstrate the requisite causal link" and there must be "a medical theory causally connecting the vaccine and injury").

The onset of petitioner's altered mental status occurred on December 19, and his herpes zoster skin rash was present on December 23. Based on testimony of Dr. Sriram, and the journal articles by Arruti, De Broucker, Grahn and Stadahl, and Science, the timeline of illness and rash is consistent with the clinical course of VZV meningoencephalitis occurring with viral reactivation. Thus, the undersigned finds the respondent has proven by preponderant evidence that the temporal association between the mechanism of reactivation on the onset of illness is appropriate.

VII. CONCLUSION

It is clear from the medical records that Mr. Dunn suffered as a result of his illness, and the undersigned extends her sympathy to him. However, this case cannot be decided based upon sympathy but rather by an analysis of the evidence.

For all of the reasons discussed above, the undersigned finds that respondent has established by preponderant evidence a factor unrelated to vaccination caused petitioner's illness. Therefore, petitioner is not entitled to compensation and his petition must be dismissed. In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master