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Short Communication

Changes in ganglioside antibody positivity rates during the COVID-19 pandemic

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ABSTRACT

Reports suggested an association between SARS-CoV-2 infection and GBS, but subsequent studies produced conflicting results regarding the incidence of GBS during the pandemic. This study assessed positivity rates for GQ1b, GM-1, GD1a, and GD1b for tests performed January 2016, through March 2021, at a national laboratory. Relative to pre-pandemic levels, positivity rates during the pandemic declined by 61% for GQ1b and 24% for GM-1, while unchanged for GD1a and GD1b. These findings suggest heterogeneity with positivity rates of GBS-associated ganglioside antibodies during the COVID-19 pandemic. Mitigation strategies during the pandemic may have reduced the frequency of certain forms of GBS.

1. Introduction

Many neuroimmunologic disorders are thought to result after a prior viral or bacterial infection. Various mechanisms have been proposed to trigger these presumed autoimmune disorders from molecular mimicry to bystander activation (Wanleenuwat et al., 2019). With the occurrence of the COVID-19 pandemic, case reports have suggested a temporal association between COVID-19 and some neuroimmunologic disorders, including disorders such as inflammatory antiganglioside neuropathies.

One such neuroimmunologic disorder is Guillain-Barré syndrome (GBS), which includes acute inflammatory demyelinating polyradiculoneuropathy (AIDP) and acute motor axonal neuropathy (AMAN). There are clear infectious triggers for GBS, including viral infections such as Epstein Barr virus, Zika virus, *Haemophilus influenzae* and bacterial infections such as *Campylobacter jejuni* (Koike and Katsuno, 2021). Several case reports have suggested a temporal association with SARS-CoV-2 infection and the subsequent occurrence of GBS (Table 1). This led to several epidemiologic studies to examine if the incidence of GBS increased during the COVID-19 pandemic. While several studies suggested an increase in GBS during the pandemic (Fragiel et al., 2021; Abu-Rumeileh et al., 2021; Filosto et al., 2021), a study from the United Kingdom found a reduction in the number of cases despite using a number of techniques to determine whether there was an association between COVID-19 and GBS (Keddie et al., 2021). In particular, cases of COVID-19 and GBS in various regions did not appear to correlate and the authors concluded there was no epidemiologic evidence that SARS-CoV-2 was causative of GBS (Keddie et al., 2021). However, in these studies GBS was viewed as a homogenous disorder. Interestingly, over 50% of GBS cases have identifiable antibodies present to various gangliosides (Cutillo et al., 2020), making it possible to examine whether the COVID-19 pandemic affected the different forms of GBS in a heterogeneous fashion.

In this study, we examined the occurrence of four ganglioside antibodies associated with GBS based on testing at a US reference clinical laboratory. Our goal was to assess changes in the incidence of these GBS-associated antibodies during the COVID-19 pandemic compared to pre-pandemic testing.

2. Materials and methods

All testing was performed by Quest Diagnostics. Detection of anti-GQ1B, anti-GM1, anti-GD1a, and anti-GD1b antibodies was performed using covalent ELISA technology tests developed and validated by Quest Diagnostics.

2.1. Study population

All GQ1b, GM1, GD1a, and GD1b, results from tests performed

Abbreviations: GBS, Guillain-Barré Syndrome.

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January 1, 2016 through March 31, 2021 that included a company-wide unique identifier were selected for potential inclusion in this study. The study population for each test result was limited to one result per patient; if any test detected antibodies, that patient was classified as positive. If antibodies were detected multiple times for the same patient, the earliest detection date was used to assess cohort trends in positivity over time. If the same patient was negative multiple times, the first negative date was used to assess cohort trends in positivity over time. Patients with indeterminate results were excluded as their status could not be classified.

2.2. Definitions

Patients were assessed geographically by United States Health and Human Services (HHS) Region. When patient state data were not available, the ordering clinician's account state was used. The "pandemic" period was defined as the period from April 1, 2020, through March 31, 2021 to align with quarterly analysis.

2.3. Statistical analyses

Differences in proportions between groups were analyzed using the chi-square test. Trends in positivity rates among age groups were analyzed using the Cochran-Armitage test for trend. HHS region 9 (California, Arizona, Hawaii, Nevada) was used as the reference group in statistical analysis because it had the most patients. Multivariable logistic regression models were performed to assess the impact on positivity of potential changes in the demographic factors of patients tested for each antibody during the pandemic. The model used a stepwise entry criterion of $p < 0.05$ and excluded patients with missing values for any included factor. Data analyses were performed using SAS® Studio 3.6 on SAS® 9.4 (SAS Institute Inc., Cary, NC, USA). This Quest Diagnostics Health Trends® study was deemed exempt by the WCG Institutional Review Board (Puyallup, Washington).

3. Results

The potential cohort included 25,010 patients with GQ1b results,

Table 1
Antibody positive cases of Guillain-Barre syndrome or Miller-Fisher syndrome.

Author	N	Time course	Neurological symptoms	GBS specific antibody	Comments
Guilmot et al., 2021	3	Neurological symptoms developed 5–21 days after COVID-19 symptoms	One patient each developed cranial neuropathy and meningo-polyradiculitis, brainstem encephalitis, and delirium with associated involuntary movements and ataxia	Anti-GD1b IgG	Questionable pathogenicity of anti-GD1b due to highly variable clinical presentations. Also identified a case of anti-Caspr2 encephalitis.
Masuccio et al., 2021	1	Neurological symptoms developed 15 days after COVID-19 symptoms	Quadriparesis, decreased tactile and pain sensations in lower extremities, urinary retention, perineal areflexia, DTR increased in all limbs. Electrophysiology was indicative of acute motor axonal neuropathy and MRI showed hyperintense lesions in the spinal cord at T2	Anti-GD1b IgM	Rare case of both myelitis and GBS with antibody positivity. COVID-19 nasal swab was negative, but COVID-19 antibodies were found in the blood.
Gutiérrez-Ortiz et al., 2020	2	Neurological symptoms developed 3–5 days after COVID symptoms	Patient one had anosmia, ageusia, right internuclear ophthalmoparesis, right fascicular oculomotor palsy, ataxia. Patient two had areflexia, ageusia, bilateral abducens palsy.	Patient one was positive for Anti-GD1b IgG, patient two negative	Miller-Fisher syndrome was probable in one patient and polyneuritis cranialis was likely in the other.
Dufour et al., 2021	1	Neurological symptoms developed 21 days after positive COVID test	Ascending areflexic paralysis of lower extremities, absent DTR, ageusia, anosmia, MRI negative for demyelination.	Positive for Anti-GM1, anti-GD1a, anti-GD1b, anti-GQ1b	Resolution of symptoms was achieved with IVIG, but no neurophysiological studies were performed.
Kopscik et al., 2020	1	Neurological symptoms started 2 months before positive COVID test	Progressive weakness, numbness, difficulty walking, cranial nerve abnormalities, dysmetria, ataxia, and absent lower extremity reflexes.	Anti-GQ1b IgG positive	Patient did not have typical COVID-19 symptoms such as fever or respiratory involvement. Neurological symptoms developed before positive test for COVID-19
Lantos et al., 2020	1	Neurological symptoms started 2 days after COVID-19 symptoms developed	Reduced sensation and paresthesia in lower limbs, left eye drooping, blurry vision, enlargement of left cranial nerve 3 on MRI	Anti-GM1 IgG was in the equivocal range, all others negative	MFS was diagnosed, despite negative autoantibodies, due to consistent symptomatology with MFS.
Gigli et al., 2021	8	Unclear time course	Paresthesias, tetraparesis in multiple patients	1 patient positive for anti-GD1a IgG and anti-GT1b IgG, 5 negative, 2 not tested	While these patients may have GBS, the association seems questionable based on the unclear time course and lack of positive COVID-19 tests
Chan et al., 2021	2	Neurological symptoms developed 18–23 days after onset of COVID-19 symptoms	Patient one had paresthesias, gait disturbance, facial weakness, dysarthria, dysphagia, CSF results consistent with GBS. Patient two had paresthesias, gait disturbance, absent reflexes in the legs, facial weakness, autonomic dysfunction, respiratory failure.	Patient one was not tested, patient two was positive for anti-GM2 IgG/IgM	Electromyography was deferred in both patients due to infection control measures.
Lowery et al., 2020	1	Neurological symptoms developed 14 days after onset of COVID-19 symptoms	Gait ataxia, left facial and bilateral lower extremity weakness, dysphagia, quadriparesis, global areflexia, cranial nerve 3, 4, and 6 weakness.	Positive for Anti-GQ1b IgG	MFS with GBS overlap was diagnosed.
Petrelli et al., 2020	1	Neurological symptoms developed 17 days after onset of COVID-19 symptoms	Hypoesthenia, loss of mobility, upper limb flaccid paralysis, DTR absent on right side, electroneurography had absence of a demyelinating pattern, but showed axonal-only motor neuropathy	Positive for anti-GM1 IgG and anti-GD1a IgG	GBS diagnosed based on presence of autoantibodies and symptomatology.
Civardi et al., 2020	1	Neurological symptoms developed 10 days after onset of COVID-19 symptoms	Lower limb weakness, paresthesias, generalized areflexia, nerve conduction studies showed demyelinating pattern. Eventually developed quadriplegia and neuromuscular respiratory failure	Positive for anti-GM1 IgG, anti-GD1b IgG, anti-GD1a IgG	GBS diagnosed based on presence of autoantibodies and symptomatology.

N = number of patients in study.

45,051 patients with GM1 results, 19,711 patients with GD1a results, and 18,962 patients with GD1b results. A small number of patients were excluded due to having only indeterminate/inconclusive results (4 for GQ1b, 3 for GM1, 1 for GD1a, and 3 for GD1b), leaving a final analytic cohort representing over 99.9% of potential patients for each outcome (Table 2).

Patient demographics and their respective associations with positive outcomes are shown in Table 2. Notable findings included a significantly higher proportion of males compared to females testing positive for GQ1b ($p < 0.001$), GD1a ($p < 0.001$), and GD1b ($p = 0.010$). GQ1b positivity rate decreased with increasing age groups ($p < 0.001$ for trend). Conversely, there was a statistically significant increase in the GM1 and GD1a positivity rates with increasing age groups ($p < 0.001$ for trend). The GQ1b positivity rate was highest in hospital inpatients (4.7%, 95% CI 4.1–5.2%) and lowest in neurology outpatients (1.1%, 95% CI 0.9–1.3%). We also examined the top ICD-10 diagnosis codes included on the requisition for both tests ordered and positive results (see Supplementary Table 1). The top diagnosis code associated with all of the antibodies ordered was polyneuropathy, followed by hereditary and idiopathic neuropathy and paresthesia of the skin. Because ICD10 diagnosis codes for Miller Fisher syndrome and acute ataxic neuropathy do not exist, more general diagnoses were associated with the anti-ganglioside antibodies associated with those disorders. Vitamin D deficiency was also a frequently listed ICD-10 code for all of the anti-ganglioside tests ordered.

Quarterly trends in patients tested and positivity are shown in Figs. 1 and 2. In general, testing volume was substantially reduced during the second quarter of 2020 (approximately 20% for most tests), consistent with the shutdown that occurred as a result of the pandemic. In subsequent quarters, testing generally increased, suggesting that testing for antibodies associated with neuroimmunologic disorders had returned to pre-pandemic levels or greater. The GQ1b positivity rate demonstrated a significant 61% decline during the pandemic period compared to the preceding period studied (1.2%, 95% CI 0.9–1.4%; versus 3.1%, 95% CI 2.9–3.4%, $p < 0.001$; Fig. 1A). In a logistic regression model adjusting for all demographic factors presented in Table 2, the GQ1b positivity rate was significantly lower during the pandemic period (AOR 0.33, 95% CI 0.26–0.43).

The GM1 positivity rate demonstrated a significant 19% decline during the pandemic period compared to the prior year (13.8%, 95% CI 13.2–14.5%; versus 17.0%, 95% CI 16.3–17.8%, $p < 0.01$; Fig. 1B). A logistic model adjusting for demographic factors confirmed this association (AOR 0.78, 95% CI 0.72–0.85) GM1 positivity rates also declined significantly in each of the prior two years; however, it is notable that the positivity rate in Q1 2021 (11.4%, 95% CI 10.3–12.6%) was the lowest rate during the study period.

Although it did drop substantially in the most recent quarter where data was available, the GD1a positivity rate was not significantly lower during the pandemic compared to the entire pre-pandemic period (6.5%, 95% CI 5.8–7.2%; versus 7.2%, 95% CI 6.8–7.6%, $p = 0.109$; Fig. 2A). The GD1b positivity rate was not lower during the pandemic compared to the prior year (1.8%, 95% CI 1.4–2.2%; versus 1.9%, 95% CI 1.5–2.4%, $p = 0.559$; Fig. 2B). Logistic regression models adjusting for demographic factors confirmed the lack of association.

4. Discussion

In this study, we examined the positivity rates for several ganglioside antibodies that had been shown to be temporally associated with SARS-CoV-2 infection in case reports (see Table 1). Specifically, we examined whether positivity for antibodies associated with GBS changed during the COVID-19 pandemic. This area is controversial in the literature. Some studies suggest an association between COVID-19 infection and GBS, while a larger study published by Keddie et al. utilizing a number of methodologies did not show an association and actually reported a decrease in cases of GBS in the United Kingdom. Studies on GBS are fraught with issues regarding case definition and ascertainment bias (Sevjar et al., 2011). We examined positivity rates for antibodies associated with various forms of GBS and found that GQ1b positivity rates declined dramatically after the onset of the pandemic. GM1 positivity rates also declined significantly during the pandemic, but this may reflect a continuation of a declining trend that was demonstrated prior

Table 2
Demographics of patient testing and positivity for GBS-associated antiganglioside antibodies.

	GQ1b		GM-1		GD1a		GD1b		
	Total	Positive	Total	Positive	Total	Positive	Total	Positive	
Total	25,006	660 (2.6)	45,048	7734 (17.2)	19,711	1390 (7.1)	18,959	556 (2.9)	
Sex									
Female	12,557	266 (2.1)*	21,191	3683 (17.4)	9921	586 (5.9)*	9748	251 (2.6)	**
Male (ref)	12,434	394 (3.2)	23,824	4050 (17.0)	9779	804 (8.2)	9197	305 (3.3)	
Age Group (years)									
<18 y	532	23 (4.3)*	418	60 (14.4)	218	8 (3.7)**	183	3 (1.6)	
18–29 y	1422	67 (4.7)*	1868	235 (12.6)*	910	48 (5.3)*	831	24 (2.9)	
30–49 y	5008	172 (3.4)*	7893	1337 (16.9)	3717	237 (6.4)*	3513	78 (2.2)	
50–69 y	10,498	271 (2.6)*	19,581	3401 (17.4)	8391	589 (7.0)	8205	276 (3.4)	
≥70 y (ref)	7538	127 (1.7)	15,273	2700 (17.7)	6469	508 (7.9)	6220	175 (2.8)	
Physician setting/specialty									
Neurology	9427	103 (1.1)*	19,214	3287 (17.1)	8673	609 (7.0)	8957	241 (2.7)	
Hospital	6565	305 (4.7)*	11,220	1925 (17.2)	4404	368 (8.4)*	3967	152 (3.8)	*
General Practice	2151	39 (1.8)*	3317	586 (17.7)	1611	104 (6.5)	1556	46 (3.0)	
Internal Medicine	952	12 (1.3)*	2958	495 (16.7)	1092	71 (6.5)	950	29 (3.1)	
All Others (ref)	5864	200 (3.4)	8119	1414 (17.4)	3857	235 (6.1)	3497	87 (2.5)	
Health and Human Services Region									
1: CT, MA, ME, NH, RI, VT	1609	55 (3.4)*	3270	594 (18.2)	1601	115 (7.2)	1070	37 (3.5)	
2: NJ, NY	3060	60 (2.0)	6878	1117 (16.2)	2833	213 (7.5)	2206	54 (2.5)	
3: DE, DC, MD, PA, VA, WV	2247	90 (4.0)*	3879	606 (15.6)	1381	95 (6.9)	1415	40 (2.8)	
4: AL, FL, GA, KY, MS, NC, SC, TN	5857	128 (2.2)	9324	1576 (16.9)	3978	293 (7.4)	4615	132 (2.9)	
5: IL, IN, MI, MN, OH, WI	2597	88 (3.4)*	2917	585 (20.1)*	1306	92 (7.0)	1664	61 (3.7)	
6: AR, LA, NM, OK, TX	1785	55 (3.1)**	3789	630 (16.6)	1584	122 (7.7)	1144	36 (3.2)	
7: IA, KS, MO, NE	726	9 (1.2)	1042	269 (25.8)*	802	53 (6.6)	599	19 (3.2)	
8: CO, MT, ND, SD, UT, WY	261	14 (5.4)*	342	39 (11.4)*	100	4 (4.0)	84	1 (1.2)	
9: AZ, CA, HI, NV (ref)	6067	135 (2.2)	12,301	2059 (16.7)	5276	338 (6.4)	5761	163 (2.8)	
10: AK, OR, ID, WA	581	13 (2.2)	1215	249 (20.5)*	825	62 (7.5)	388	13 (3.4)	

Chi-square test $p < 0.05$**; $p < 0.01$.

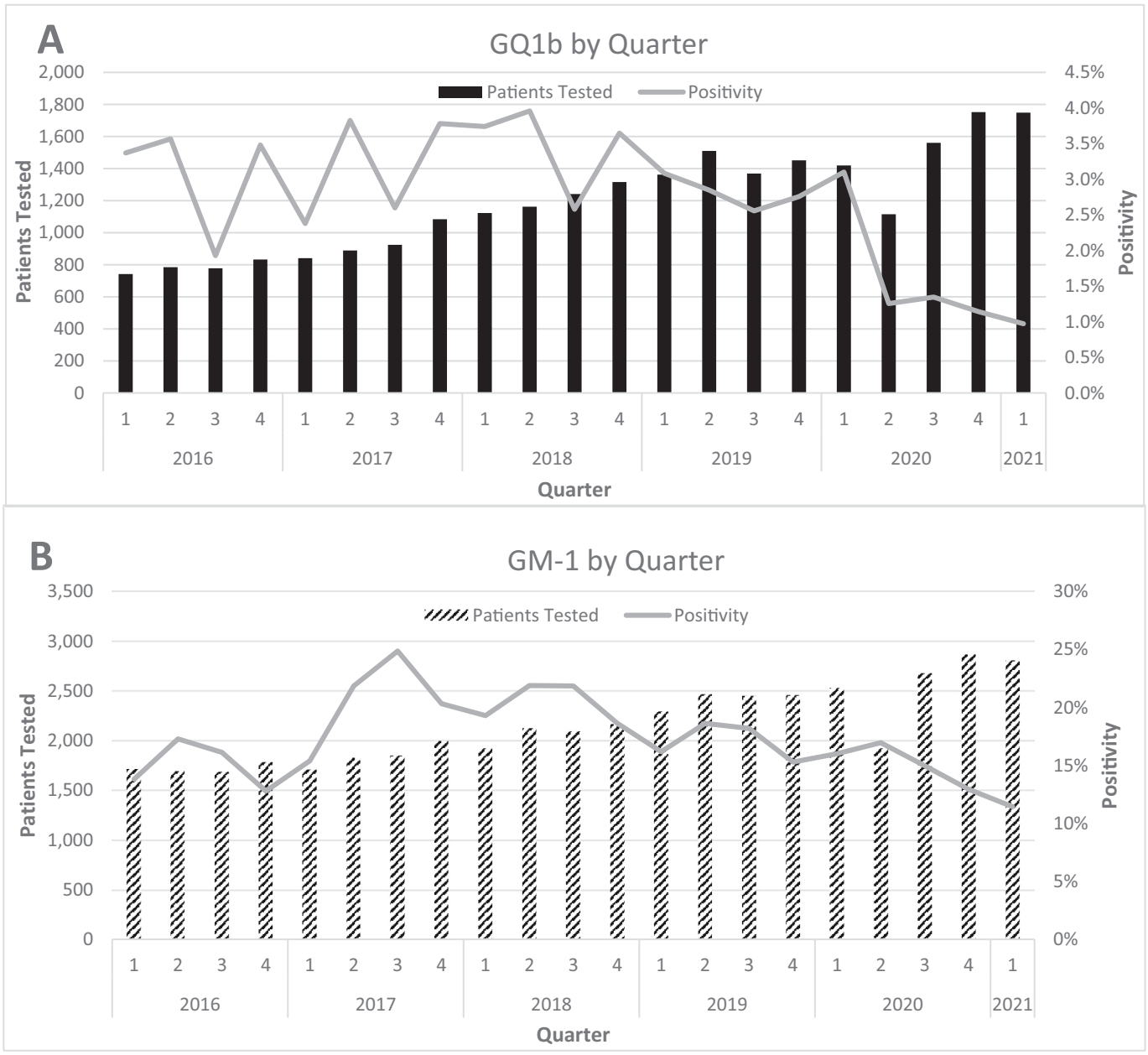

Fig. 1. Quarterly Number of Patients Tested and Positivity Rate for GQ1b (A) and GM1 (B).

to the pandemic. These findings may suggest, as others have concluded, that while COVID-19 may be able to trigger neuroimmunologic conditions such as GBS, that mitigation strategies such as social distancing, mask wearing, and hand hygiene could reduce exposure to infectious agents that might otherwise trigger some forms of GBS, particularly that associated with GQ1b.

One limitation of this study is that we could not determine the specific temporal association of COVID-19 exposure with positivity for ganglioside antibodies. This was partly because SARS-CoV-2 seropositivity is several fold higher than molecular testing positivity, mostly because of the large number of asymptomatic infections occurring in the general population (Rogawski et al., 2021; Stefanelli et al., 2021). Thus, we only compared the positivity rate of antiganglioside antibodies during the pandemic to pre-pandemic levels. In addition, some of the variation in test positivity may represent seasonal variation known to occur with GBS (Webb et al., 2015).

As the exact reason for testing was unknown, there is potential selection bias. However, we were able to view the top 30 ICD-10 codes associated with the ordering of anti-ganglioside testing and this information for the top 10 codes is provided in Supplementary Table 1. Because Quest Diagnostics does not perform all GBS-associated antibody testing in the country, these data should be interpreted as a large, but not exhaustive sample of national data. In fact, this study is one of the largest to date assessing neuroimmunological complications during the COVID-19 pandemic. However, we were not able to review patient charts for specific symptoms of Guillain-Barré to determine reliability of ICD-10 diagnosis codes. In addition, our estimates may be conservative; not every case of GBS demonstrates antibody positivity. Moreover, some clinicians do not order antibody testing even if they suspect GBS. However, the frequency of vitamin D deficiency may reflect the known association of low vitamin D levels observed in GBS and CIDP (Elf et al., 2014). Notably, as vaccines became available for SARS-CoV-2, reports began to surface of GBS in association with vaccination (Allen et al., 2021; Maramattom et al., 2021). One study of 702 patients known to have GBS then vaccinated for SARS-CoV-2 found only one patient required short-term medical care for recurring symptoms (Shapiro Ben

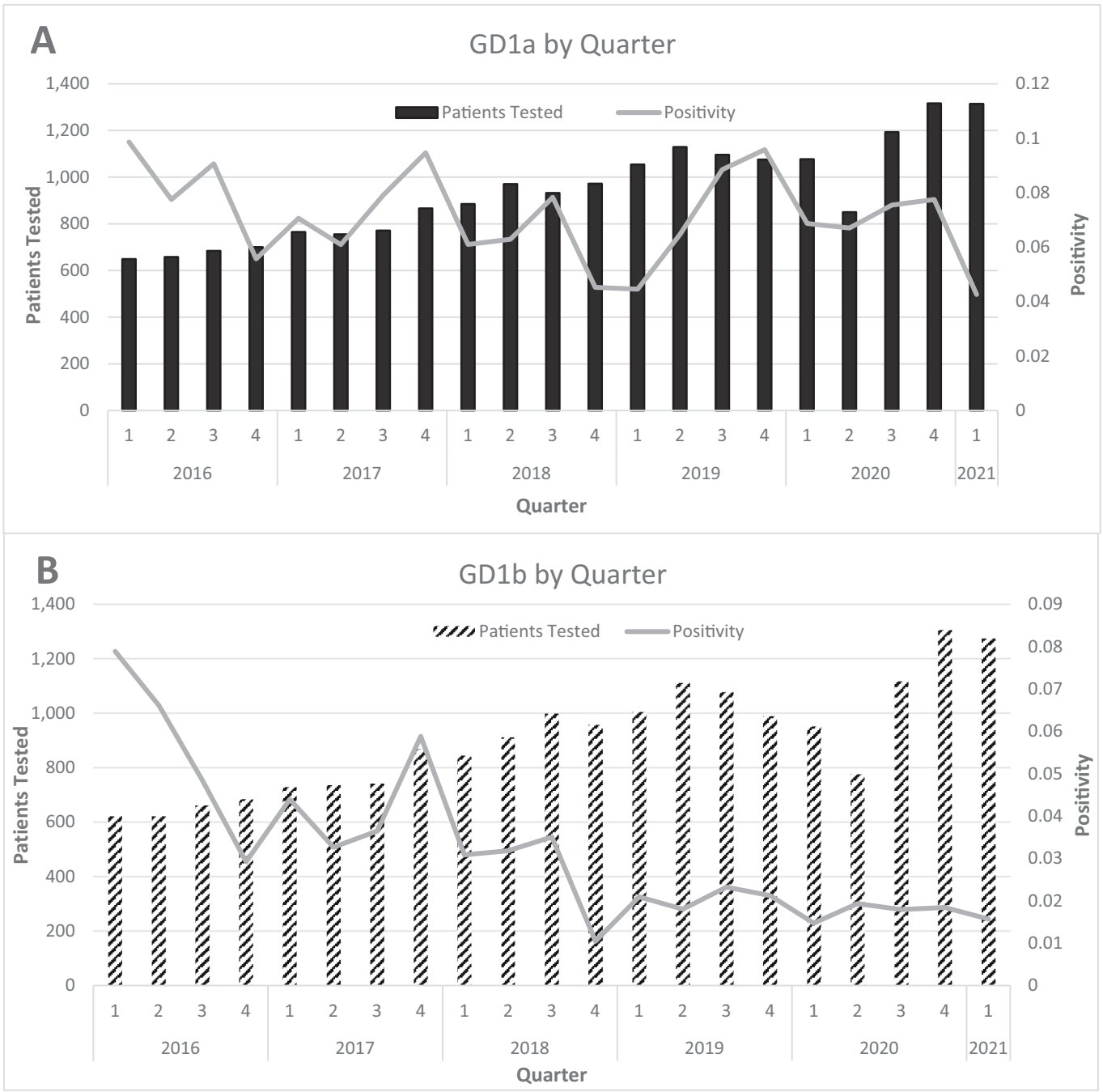

Fig. 2. Quarterly Number of Patients Tested and Positivity Rate for GD1a (A) and GD1b (B).

David et al., 2021). For this reason, we specifically examined the positivity rates for ganglioside antibodies prior to the time when vaccines became available to the general public.

5. Conclusions

These data suggest that heterogeneity in terms of the effect that the COVID-19 pandemic has had on rates of GBS associated with ganglioside antibodies. In particular, while positivity rates for GD1a and GD1b remained largely unchanged during the pandemic, rates of positivity for GQ1b and GM1 were significantly reduced during the pandemic. While these findings do not exclude the possibility that immune responses to SARS-CoV-2 may trigger autoimmune responses to gangliosides as suggested in several case reports (see Table 1), they do suggest that mitigation strategies taken during the pandemic could possibly have reduced the frequency of certain forms of GBS, such as those mediated by GQ1b and GM1.

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MKR, JKN, RAL, and HWK are employees of Quest Diagnostics and

MKR and HWK own stock in Quest Diagnostics.

References

Abu-Rumeileh, S., Abdelhak, A., Foschi, M., Tumani, H., Otto, M., 2021. Guillain-Barre syndrome spectrum associated with COVID-19: an up-to-date systematic review of 73 cases. J. Neurol. 268, 1133–1170.

Allen, C.M., Ransamy, S., Tarr, A.W., et al., 2021. Guillain-Barre variant occurring after SARS-CoV-2 vaccination. Ann. Neurol. 90, 315–318.

Chan, M., Han, S.C., Kelly, S., Tamimi, M., Giglio, B., Lewis, A., 2021. A case series of Guillain-Barré Syndrome After COVID-19 infection in New York. Neurol. Clin. Pract. 11, e576–e578.

Civardi, C., Collini, A., Geda, D.J., et al., 2020. Antiganglioside antibodies in Guillain-Barré syndrome associated with SARS-CoV-2 infection. J. Neurol. Neurosurg. Psychiatry 91, 1361–1362.

Cutillo, G., Saariaho, A.-H., Meri, S., 2020. Physiology of gangliosides and the role of antiganglioside antibodies in human diseases. Cell. Mol. Immunol. 17, 313–322.

Dufour, C., Co, T.K., Liu, A., 2021. GM1 ganglioside antibody and COVID-19 related Guillain Barre syndrome - a case report, systemic review and implication for vaccine development. Brain Behav. Immun. Health. 12, 100203.

Elf, K., Askmark, H., Nygren, I., Punga, A.R., 2014. Vitamin D deficiency in patients with primary immune-mediated peripheral neuropathies. J. Neurol. Sci. 345 (1–2), 184–188.

Filosto, M., Cotti Piccinelli, S., Gazzina, S., et al., 2021. Guillain-Barre syndrome and COVID-19: an observational multicentre study from two Italian hotspot regions. J. Neurol. Neurosurg. Psychiatry 92, 751–756.

Fragiel, M., Miro, O., Llorens, P., et al., 2021. Incidence, clinical, risk factors and outcomes of Guillain-Barre in COVID-19. Ann. Neurol. 89, 598–603.

Gigli, G.L., Bax, F., Marini, A., et al., 2021. Guillain-Barré syndrome in the COVID-19 era: just an occasional cluster? J. Neurol. 268, 1195–1197.

Guilmot, A., Slootjes, S.M., Sellimi, A., et al., 2021. Immune-mediated neurological syndromes in SARS-CoV-2-infected patients. J. Neurol. 2021 (268), 751–757.

Gutiérrez-Ortiz, C., Méndez-Guerrero, A., Rodrigo-Rey, S., et al., 2020. Miller fisher syndrome and polyneuritis cranialis in COVID-19. Neurology. 95, e601–e605.

Keddie, S., Pakpoor, J., Mousele, C., et al., 2021. Epidemiological and cohort study finds no association between COVID-19 and Guillain-Barre syndrome. Brain. 144, 682–693.

Koike, H., Katsuno, M., 2021. Emerging infectious diseases, vaccines and Guillain-Barré syndrome. Clin. Exp. Neuroimmunol.. https://doi.org/10.1111/cen3.12644.

Kopscik, M.R., Giourgas, B.K., Presley, B.C., 2020. A case report of acute motor and sensory polyneuropathy as the presenting symptom of SARS-CoV-2. Clin. Pract. Cases Emerg. Med. 4, 352–354.

Lantos, J.E., Strauss, S.B., Lin, E., 2020. COVID-19-associated miller fisher syndrome: MRI findings. Am. J. Neuroradiol. 41, 1184–1186.

Lowery, M.M., Taimur Malik, M., Seemiller, J., Tsai, C.S., 2020. Atypical variant of Guillain Barre syndrome in a patient with COVID-19. J. Crit. Care Med. 6, 231–236.

Maramattom, B.V., Krishnan, P., Paul, R., et al., 2021. Guillain-Barre syndrome following ChAdOx1-S/nCoV-19 vaccine. Ann. Neurol. 90, 312–314.

Masuccio, F.G., Barra, M., Claudio, G., Claudio, S., 2021. A rare case of acute motor axonal neuropathy and myelitis related to SARS-CoV-2 infection. J. Neurol. 268, 2327–2330.

Petrelli, C., Scendoni, R., Paglioriti, M., Logullo, F.O., 2020. Acute motor axonal neuropathy related to COVID-19 infection: a new diagnostic overview. J. Clin. Neuromuscul. Dis. 22, 120–121.

Rogawski, E.T., Guertin, K.A., Becker, L., et al., 2021. Assessment of seroprevalence of SARS-CoV-2 and risk factors associated with COVID-19 infection among outpatients in Virginia. JAMA Netw. Open 4, e2035234.

Sevjar, J.J., Baughman, A.L., Wise, M., Morgan, O.W., 2011. Population incidence of Guillain-Barre syndrome: a systematic review and meta-analysis. Neuroepidemiology. 36, 123–133.

Shapiro Ben David, S., Potasman, I., Rahamim-Cohen, D., 2021. Rate of recurrent Guillain-Barré syndrome after mRNA COVID-19 vaccine BNT162b2. JAMA Neurol. 78, 1409–1411. https://doi.org/10.1001/jamaneurol.2021.3287.

Stefanelli, P., Bella, A., Fedele, G., et al., 2021. Prevalence of SARS-CoV-2 IgG antibodies in an area of northeastern Italy with high incidence of COVID-19 cases: a population-based study. Clin. Microbiol. Infect. 27, 633.e1–633.e7.

Wanleenuwat, P., Iwanowski, P., Kozubski, W., 2019. Antiganglioside antibodies in neurological diseases. J. Neurol. Sci. 408, 116576.

Webb, A.J.S., Brain, S.A.E., Wood, R., Rinaldi, S., Turner, M.R., 2015. Seasonal variation in Guillain-Barre syndrome: a systematic review, meta-analysis, and Oxfordshire cohort study. J. Neurol. Neurosurg. Psychiatry 86 (11), 1196–1201.