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Case Report



# Cerebral Vasculitis and Hearing Loss After COVID-19 mRNA Vaccination

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## Abstract

A previously healthy adult man presented with headaches 2 weeks after receiving the first dose of BNT162b2 mRNA vaccine. A few days later, he developed hearing loss in both ears and blurry vision. Magnetic resonance imaging & vessel wall angiography of the brain revealed acute infarctions in the posterior circulation and new irregularity of the left posterior cerebral artery with associated concentric vessel wall enhancement. Cerebrospinal fluid testing revealed lymphocytic pleocytosis suggesting a vasculitic process. Extensive etiological workup was unrevealing for infectious & systemic inflammatory causes. He was treated with steroids and cyclophosphamide for 5 months with favorable clinical and radiographic response. Although a direct causal relationship is difficult to establish, given the onset of symptoms within 2 weeks of receiving SARS-CoV-2 mRNA vaccine, an immune mediated cerebral vasculitis that was triggered but not necessarily caused by the SARS-CoV-2 mRNA vaccine seems plausible. Treatment with immunosuppression rendered a favorable prognosis. This case highlights that immune mediated manifestations in the central nervous system (CNS) e.g. cerebral vasculitis, may occur after receiving SARS-CoV-2 mRNA vaccine. However, if treated early with immunosuppression, a favorable clinical and radiographic response can be achieved with treatment.

**Keywords:** MeSH terms: C10 –nervous system diseases, C20- immune system diseases other keywords: vasculitis, vaccination, stroke, neuro-otology, COVID

## Background

Cases of inflammatory conditions including myocarditis and cutaneous vasculitis after receiving coronavirus disease 2019 (COVID-19) messenger RNA (mRNA) vaccination have been reported previously. [1,2] Anecdotal reports of sudden sensorineural hearing loss (SNHL) after COVID-19 vaccination have also been recently emerging. [3] However, cerebral vasculitis with hearing loss has not been reported previously. Here, we present a case of cerebral vasculitis and SNHL 2 weeks after receiving the COVID-19 mRNA vaccine.

## Case Presentation

A previously healthy 60 years old man presented with debilitating bifrontal headaches 2 weeks after receiving the first dose of BNT162b2 mRNA vaccine. A few days later, he developed sudden complete hearing loss in the left ear. Another few days later, he lost hearing in his right ear also. There was no history of barotrauma or ototoxic drug exposure. Initial physical and neurological exam was normal except for bilateral sensorineuronal hearing loss. One week later, he became confused and reported blurry vision in both eyes prompting further work up.

## Investigations

Initial blood testing revealed elevation in his white blood cell count (15,000/mm3), erythrocyte sedimentation rate (64 mm/hour) and C-reactive protein (9.0 mg/L). Initial computed tomography (CT) of the brain, CT angiography (CTA) of the head and neck and magnetic resonance imaging (MRI) of the brain with gadolinium showed no acute abnormalities. One week later when he presented with confusion and bilateral blurry vision, MRI of the brain

with contrast was repeated and revealed acute infarctions in the bilateral occipital lobes and cerebellar hemispheres (Fig 1-A, B). MR angiography (MRA) of the brain and carotids with gadolinium showed a new stenosis of the left posterior cerebral artery (PCA) and mid-basilar artery (Fig 1-C). High resolution vessel wall magnetic resonance imaging (VWMRI) showed smooth concentric vessel wall enhancement of the left PCA (Fig 1-D) suggesting inflammation of the vessel walls. Cerebrospinal fluid (CSF) analysis revealed a predominantly lymphocytic pleocytosis (78 cells/mm3, normal <5 cells/mm3), elevated protein (198 mg/dL, normal 15-45 mg/dL) and IgG index (1.24, normal <0.65). CSF glucose, gram stain, bacterial and fungal cultures, and extensive viral PCRs were unremarkable (table 1). Audiometric evaluation revealed severe bilateral sensorineuronal hearing loss at all frequencies. An extensive etiological workup was unrevealing for embolic, hypercoagulability, infectious and systemic inflammatory causes (tables 1 &2).

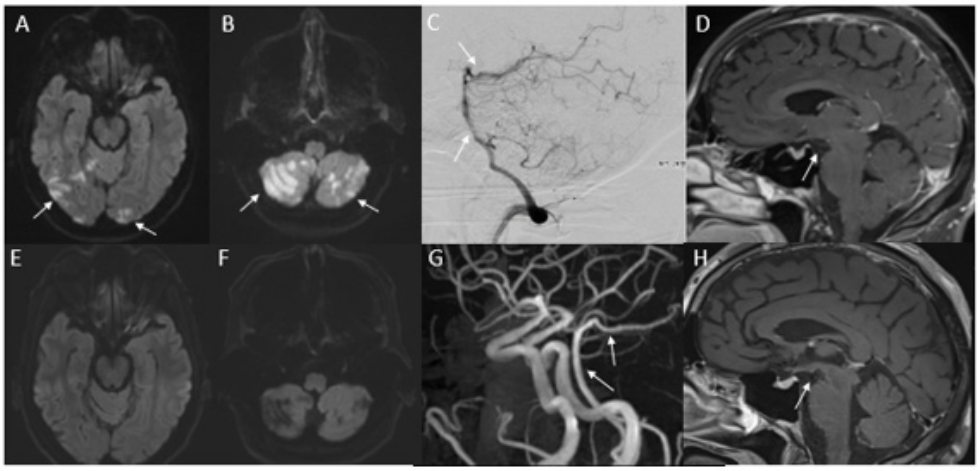


**Figure 1:**

**Table 1:** Vital Signs and Diagnostic Serological Work up

| | Patient Values | Normal Range |
|---|---|---|
| **Admission Vital Signs** | | |
| Heart rate (beats per minute) | 67 | |
| Respiratory rate (per minute) | 18 | |
| Blood pressure (mm/Hg) | 128/78 | |
| Temperature (°C) | 36.4 | |
| Pulse oximeter saturation (%) | 99% | |
| BMI | 25.77 kg/m$^2$ | |
| **Admission Laboratories** | | |
| WBC | 15.7 | 4.0- 10 per 10$^{*3}$/uL |
| %neutrophils | 79 | |
| %lymphocytes | 13 | |
| %monocytes | 8 | |
| %eosinophils | 0 | |
| %basophils | 0 | |
| Hemoglobin (g/dL) | 13.9 | 13.5 – 18 g.dL |
| Hematocrit (%) | 43 | 41 - 53% |
| Platelets (per cubic mm) | 393 | 150 – 400 per 10$^{*3}$/uL |
| Sodium | 137 | 136 - 145 mmol/L |
| Potassium | 4.7 | 3.4 - 5.1 mmol/L |
| Chloride | 98 | 98 - 107 mmol/L |
| Bicarbonate | 27 | 22 - 29 mmol/L |
| BUN | 14 | 8 - 23 mg/dL |

| | | |
|---|---|---|
| Creatinine | 0.84 | 0.70 - 1.20 mg/dL |
| Glucose | 105 | 70 - 140 mg/dL |
| **Inflammatory Serological Labs** | | |
| Erythrocyte sedimentation rate | 64 | <20mm/hr |
| C reactive protein | 9.0 | <3.0 mg/L |
| Antinuclear antigen, qualitative | Negative | Negative |
| Rheumatoid Factor | <10 | <14 IU/ml |
| C3 complement levels | 133 | 90 - 180 mg/dL |
| C4 Complement | 30 | 10 - 40 mg/dL |
| ANCA antibody | Negative | Negative |
| Smith autoantibody | 5 | 0 – 99 [AU]/ml |
| RNP autoantibody | 18 | 0 – 99 U/ml |
| SSA autoantibody | 12 | 0 – 99 [AU]/ml |
| SSB autoantibody | 13 | 0 – 99 [AU]/ml |
| SCL-70 autoantibody | 10 | 0 – 99 [AU]/ml |
| Jo-1 autoantibody | 40 | 0 – 99 [AU]/ml |
| Double stranded DNA antibody | 5 | 0 – 99 [IU]/ml |
| Centromere antibody | 12 | 0 – 99 [AU]/ml |
| Histone antibody | 14 | 0 – 99 [AU]/ml |
| Cryoglobulin | Negative | Negative |
| **Infectious Serological Labs** | | |
| COVID-19 PCR | Not Detected | |
| HIV-1 p24 antigen | Not Detected | |
| HIV-1/HIV-2 antibodies | Not Detected | |
| Hepatitis B surface antigen | Not Detected | |
| Hepatitis C antibody | Not Detected | |
| Lyme Ab (IgM + IgG) | Not Detected | |
| Syphilis IgG/IgM w/reflex RPR | Not Detected | |
| **Respiratory Pathogen Panel** (Nasopharyngeal Swab for PCR) | | |
| Adenovirus | None Detected | |
| Coronavirus 229E | None Detected | |
| Coronavirus HKU1 | None Detected | |
| Coronavirus NL63 | None Detected | |
| Coronavirus OC43 | None Detected | |
| Human Metapneumovirus | None Detected | |
| Rhinovirus/Enterovirus | None Detected | |
| Influenza A | None Detected | |
| Influenza B | None Detected | |
| Parainfluenza 1 | None Detected | |
| Parainfluenza 2 | None Detected | |
| Parainfluenza 3 | None Detected | |
| Parainfluenza 4 | None Detected | |
| Respiratory Syncytial Virus | None Detected | |
| Bordetella pertussis | None Detected | |
| Chlamydia pneumoniae | None Detected | |
| Mycoplasma pneumoniae | None Detected | |
| Bordetella parapertussis | None Detected | |
| **Hypercoagulability Labs** | | |
| Lupus Anticoagulant | Negative | |
| Beta-2-Glycoprotein IgM/IgG abs | Negative | |

| | | |
|---|---|---|
| Cardiolipin IgM/IgG abs | Negative | |
| Antiphosphatidylserine abs | Negative | |
| Homocysteine, serum | 6.8 umol/L | <15 umol/L |
| **Cerebrospinal Fluid Analysis** | | |
| Color | Colorless | |
| Clarity | Clear | |
| Red blood cell count | <2 /uL | <2/uL |
| Total nucleated cells | 78/uL | <5/uL |
| Neutrophil % | 1 | |
| Monocyte/Macrophage % | 43 | |
| Lymphocyte % | 56 | |
| Protein | 198 mg/dl | 15-45 mg/dl |
| Glucose | 70 mg/dl | 40-70 mg/dl |
| Myelin basic protein | >167 ng/mL | 0.0 – 4.7 ng/mL |
| CSF to serum IgG index | 1.24 | < 0.65 |
| Oligoclonal Bands | None | None |
| CSF Lyme ab | Negative | Negative |
| CSF VDRL | Non-Reactive | Non-Reactive |
| Angiotensin converting enzyme | <1.5 U/L | 0.0 – 3.1 U/L |
| Gram Stain | No organisms seen | |
| Bacterial Culture | No Growth | |
| Fungal Culture | No Growth | |
| Cryptococcal antigen | None Detected | |
| **CSF Pathogen Panel, PCR** | | |
| Escherichia coli K1 | Not Detected | |
| Haemophilus influenza | Not Detected | |
| Listeria Monocytogenes | Not Detected | |
| Neisseria meningitidis | Not Detected | |
| Streptococcus agalactiae | Not Detected | |
| Streptococcus pneumoniae | Not Detected | |
| Cytomegalovirus | Not Detected | |
| Enterovirus | Not Detected | |
| Herpes simplex virus 1 | Not Detected | |
| Herpes simplex virus 2 | Not Detected | |
| Human herpesvirus 6 | Not Detected | |
| Human Parechovirus | Not Detected | |
| Varicella zoster virus | Not Detected | |
| C. neoformans/gattii | Not Detected | |
| Other serological work up | | |
| Hemoglobin A1c | 6.1% | 4.0 – 6.0 % |
| Total cholesterol | 108 mg/dL | <200 mg/dL |
| Triglycerides | 119 mg/dL | <150 mg/dL |
| HDL cholesterol | 45 mg/dL | >40 mg/dL |
| LDL cholesterol | 39 mg/dL | <100 mg/dL |
| VLDL cholesterol | 24 mg/dL | 16 – 42 mg/dL |
| Non-HDL cholesterol | 63 mg/dL | <130 mg/dL |
| TSH | 0.563 IU/ml | 0.270 – 4.2 IU/ml |
| Sickle Cell Screen | Negative | Negative |
| Angiotensin Converting Enzyme | 9 U/L | 14-82 U/L |
| Vitamin D 25 Hydroxy, total | 16 ng/ml | >30 ng/ml |

**Table 2:** Medications and Diagnostic imaging at presentation

| | |
|---|---|
| Medications Received | Aspirin<br>Atorvastatin<br>Ciprofloxacin<br>Cyclophosphamide<br>Methylprednisolone<br>Pantoprazole<br>Prednisone<br>Vitamin D |
| Imaging | |
| MRI Brain with and without contrast at initial presentation | Mild small vessel disease. No acute infarction. |
| MRI Brain with and without contrast 1 week after presentation | There is interval development of scattered areas of T2 FLAIR hyperintensity and corresponding diffusion restriction in the bilateral occipital lobes, right side greater than left in a gyriform pattern. New areas of FLAIR hyperintensities are also noted in left temporal lobe, left thalamus, splenium and bilateral cerebellar hemispheres. These are new since previous scan one week prior. |
| CT brain with CTA Head and Neck 1 week after presentation | 1. New hypodensities in the bilateral cerebellar hemispheres, right worse than left. New cortical hypodensities in the left temporal lobe and right temporal lobe. These findings may represent multiple infarctions.<br>2. New from one week prior, there is mild stenosis of the P1 segment of the left posterior cerebral artery.<br>3. Bilateral common carotid, internal carotid, and vertebral arteries are patent without hemodynamically significant stenosis. Arteries of the circle of Willis are patent. |
| Digital Subtraction Angiography 1 week after presentation | Selective right vertebral artery angiography demonstrates minimal filling of the right posterior cerebral artery circulation. A mild degree of multifocal narrowing of the vertebrobasilar circulation, including the left posterior cerebral artery and right posterior inferior cerebellar artery is seen, more pronounced in the medium and smaller vessels of the circulation suggestive of vasculopathy. |
| CTA Chest & Abdomen | No evidence of systemic large or medium vessel vasculitis |
| Transthoracic Echocardiogram with bubble study | Normal study, no patent foramen ovale. No valvular disease or vegetation seen. |
| Transesophageal Echocardiogram | Normal |
| Fluorescein Angiography: Both Eyes (OU) | No evidence of vasculitis, BRAO, or leakage OU. Normal IVFA OU |
| Electrocardiogram | Normal Sinus Rhythm |

## Differential Diagnosis

Most cases of sudden hearing loss are sensorineural, and less than 10% have an identifiable cause. Common causes are barotrauma, exposure to ototoxic medications, CNS infections (e.g. tuberculosis, Lyme disease etc), CNS demyelinating disorder (e.g. multiple sclerosis), CNS vasculopathies such as Susac syndrome, granulomatosis with polyangiitis or Cogan syndrome, or immune-mediated inner ear diseases.[4] The patient did not report any recent history of barotrauma and had not been exposed to any ototoxic drugs. Brain MRI showed no evidence of meningeal disease or typical demyelinating lesions, nor snow-ball lesions typical of Susac syndrome; a vasculopathy characterized by hearing loss, vision loss and infarcts in the corpus callosum. Common infectious, inflammatory, and systemic causes of CNS vasculitis were ruled out (table 1).

## Treatment

Treatment with intravenous methylprednisolone 500mg/day for 3 days, followed by oral prednisone 80mg/day (tapered over 5 months) and oral cyclophosphamide (2mg/kg/day) were initiated for presumed CNS vasculitis with vestibulocochlear involvement possibly triggered by the SARS-CoV-2 mRNA vaccine. Given initial response to treatment, he was continued on treatment with oral steroids and cyclophosphamide for 5 months.

## Outcome and Follow-Up

After one month of treatment, the patient's mental status normalized, headaches resolved, and mild subjective improvement in his hearing was reported. His vision stopped deteriorating any further and he was able to maintain 20/30 vision in both eyes with a homonymous hemianopia. Serum inflammatory markers nor-

malized. After 3 months of treatment, audiogram showed mild improvement in his hearing at low frequencies (table 3), MRI brain showed no further infarcts (Figure 1-E, F) and MRA brain showed resolution of the previously seen arterial abnormalities and vessel wall enhancement (Fig 1-G, H & table 4). The patient remains clinically and radiographically stable at 1 year.

**Table 3:** Audiometry Testing Results

| | |
|---|---|
| Audiometry Testing | **INTERPRETATION OF HEARING STATUS**<br><br>**RIGHT EAR:** *Sensorineural hearing loss*<br><br>**LEFT EAR:** *Sensorineural hearing loss*<br><br>**COMPARISON TO PREVIOUS TESTING, if applicable (5 months later)**<br><br>Comparison of follow up testing 5 months after initial testing (after 3 months of treatment) suggests an **improvement in low frequency hearing in the left ear.**<br><br>**AUDITORY/FACIAL NERVE FUNCTION: via acoustic reflex testing**<br><br>**RIGHT EAR PROBE EAR:** (ipsilateral right stimulus ear; contralateral left stimulus ear):<br><br>**Acoustic Reflex Pattern** Ipsilateral and contralateral acoustic reflexes were absent at 500-2000 Hz.<br><br>**Acoustic Reflex Decay** *(left stimulus ear): Could not test.*<br><br>**LEFT EAR PROBE EAR: (**ipsilateral left stimulus ear; contralateral right stimulus ear):<br><br>**Acoustic Reflex Pattern** Ipsilateral and contralateral acoustic reflexes were absent at 500-2000 Hz.<br><br>**Acoustic Reflex Decay** *(right stimulus ear): Could not test.*<br><br>**HEARING ASSESSMENT: via pure tone and speech testing**<br><br>**RIGHT EAR:**<br><br>**Hearing Sensitivity:** *Moderately-severe to severe SNHL.*<br><br>**Word Recognition Score:** *Could not test due to degree of hearing loss.*<br><br>**LEFT EAR:**<br><br>**Hearing Sensitivity:** *Severe through 1000 Hz rising to moderately-severe SNHL.*<br><br>**Word Recognition Score:** *Could not test due to degree of hearing loss.* |

**Table 4:** MRI Brain and MRA Brain results 3 months after treatment.

| | |
|---|---|
| MRI and MRA Brain with and without contrast – 3 months after treatment | No evidence of an acute intracranial abnormality.<br><br>No evidence of additional intracranial infarction since the prior examination.<br><br>Interval resolution of the small focus of enhancement in the left terminal ICA segment on the high-resolution vessel wall sequences. There is no abnormal enhancement on the high-resolution vessel wall imaging sequences on today's scan.<br><br>Stable unremarkable intracranial MRA with resolution of previously seen vasculopathy in the left PCA and basilar arteries. |

## Discussion

Otolaryngologic manifestations, including SNHL, have been frequently described following vaccination, particularly after influenza, meningococcal, rabies, tetanus and diphtheria vaccines.5 Furthermore, multiple case reports and case series noted the occurrence of SNHL in the first few days following the COVID-19 vaccine administration.[5,6] This association between SNHL and vaccination has been previously refuted in a large-scale study by Baxter et al.5 Similarly, Formeister et al compared the incidence of SNHL in individuals receiving the COVID-19 vaccine to that expected in the wider population and reported lower rates in vaccinated individuals.3 This finding should be cautiously interpreted given the susceptibility of extrapolated epidemiological analysis to bias and underreporting, as well as the questionable reliability of SNHL diagnosis in this study.[7]

SNHL occurring within 2 weeks after COVID-19 vaccination in our patient raises the possibility of the vaccine being the most likely trigger, especially after ruling out infectious, drug-induced, meningeal and demyelinating causes. The pathophysiology remains unclear, but autoimmune responses have been reported after vaccines, the plausible mechanisms being molecular mimicry, the production of autoantibodies, as well as the role of certain vaccine adjuvants in causing end organ damage.[8] In cases of SNHL in particular, multiple hypotheses have been described including immunocomplex-meditated autoimmune antibodies directed to the cochlea, and inflammatory and immune-mediated microvascular ischemic cochlear injuries, which is likely what occurred in our patient's case.5

While histologic confirmation of vasculitis is considered the gold standard for diagnosing cerebral vasculitis, the combination of cerebral vessel wall imaging findings and the inflammatory CSF profile, combined with the resolution of vessel wall enhancement and clinical improvement after treatment with steroids, served as strong evidence for a diagnosis of probable cerebral vasculitis, sparing the patient an invasive intervention. In our patient's case, an extensive evaluation for secondary causes of cerebral vasculitis was unrevealing for an alternative etiology, including systemic autoimmune disorders, an infectious process, or malignancy.

Cases of systemic and cerebral vasculitis triggered by vaccines have been previously reported. However, most cases were that of IgA mediated cutaneous vasculitis triggered by the influenza vaccine, in addition to Behcet's disease and cerebral angiitis following the human papilloma virus vaccine .[9] More recently, a few case reports described de novo systemic vasculitides (one case of ANCA-associated vasculitis and one case of IgA vasculitis with renal involvement) after mRNA SARS-CoV-2 vaccine.[10,11] The mechanism of this association remains unclear but could likely be due to an aberrant immune response to the spike protein or mRNA of SARS-CoV-2 in predisposed individuals10. One case of intracerebral hemorrhage after mRNA SARS-CoV2 vaccine was recently described, with vasculitis being the postulated underlying cause [12].

To our knowledge, this is the first report of hearing loss and cerebral vasculitis occurring within 2 weeks after the administration of the mRNA COVID-19 vaccine. Although a direct causal relationship is difficult to establish, however, given the temporal proximity of symptoms to the vaccine administration and the unremarkable extensive workup for other infectious or inflammatory causes, an immune mediated vasculitis affecting the CNS and the cochlear vessels that was likely triggered but not necessarily directly caused by the SARS-CoV-2 mRNA vaccine seems plausible.

In conclusion, although rare, immune-mediated manifestations involving the central nervous system and cochlear vessels can occur after receiving SARS-CoV-2 mRNA vaccine. Early recognition and treatment with immunosuppression can render a favorable clinical and radiographic response if treated early in the disease course. Despite these very rare immune mediated adverse manifestations, the benefits of vaccination still outweigh potential risks. Further studies geared towards identifying potential host factors that may portray a higher adverse risk profile against mRNA vaccines would be of value.

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None

## Conflict of interest statements

No conflict of interest

**Statement of ethics and consent:** This article does not contain any studies with human or animal subjects performed by any of the authors. The patient discussed in this manuscript has given written consent to publish this work.

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