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Imaging of the head and neck during the COVID19 pandemic

Joaquim Farinhas, MD, MBA[a], Jimmy S. Lee, MD, PhD, FRCPC[b]

From the [a]Department of Diagnostic Imaging and Interventional Radiology, Moffit Cancer and Research Center, Tampa, Florida
[b]Division of Neuroradiology, Department of Radiology, Montefiore Medical Center, New York, New York

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Imaging

There is a wide spectrum of clinical manifestation of COVID-19 in the head and neck, but often these do not have an imaging correlate. This review will highlight the most common imaging features of COVID-19 in the head and neck that can be seen on routine head and neck CT and MRI. In addition, situations where a more dedicated imaging protocol is required will be highlighted. Finally, as mass vaccination efforts are underway worldwide, post vaccination imaging can often complicate cancer surveillance imaging. Post vaccination imaging features and recommendations will be discussed.

Introduction

Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection results in a wide variety of clinical manifestations in the head and neck including: anosmia, cervical and axillary adenopathy, cranial neuropathies including optic neuritis, hearing loss, xerostomia and sialadenitis, Guillain-Barrè syndrome, brachial plexopathy, and venous thrombosis. With increasing vaccination efforts worldwide, awareness of post vaccination imaging features is also important to consider when assessing patients with head and neck symptoms, or in interpreting surveillance imaging for patients with known malignancy. While clinical manifestations of COVID-19 often will not have an imaging correlate, imaging remains a valuable tool to exclude other causes not due to SARS-CoV-2 infection. Conversely, a familiarity with the wide variety of imaging manifestations of COVID-19 in the head and neck also helps to include COVID-19 in the differential diagnosis when assessing patients who present atypically and have not yet been tested for SARS-CoV-2 infection. In this review, imaging manifestations will be predominantly presented in CT and MRI modalities, as these are the mainstay of head and neck imaging.

Neurologic

Routine CT and MRI scans remain the best templates in our approach to presenting the manifestations of COVID-19 as they relate to the head and neck. These studies often include the posterior fossa, and to variable degree, the supratentorial and suprasellar structures. A discussion of imaging manifestations of COVID-19 that involve the

Address reprint requests and correspondence: Joaquim Farinhas, MD MBA, Moffitt Cancer and research Center, Department of Radiology, Clinical Staff Building, 12902 USF Magnolia Drive, Tampa, FL 33612.
E-mail address: joaquim.farinhas@moffitt.org

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brain, will provide a greater understanding of common clinical presentations of this disease.

The most common clinical neurologic manifestations of COVID-19 include encephalopathy, stroke, and encephalitis.[1] The clinical presentation of encephalitis includes altered mental status >24 hours, with abnormal white blood cell count in the CSF or the presence of an acute, relevant brain lesion on MRI.[2] Encephalopathy presents as altered mental status >24 hours associated with seizure and/or focal neurologic deficits not fulfilling criteria for encephalitis with no evidence of toxic or metabolic factors.[2] Stroke presents clinically with sudden neurologic deficit secondary to acute vascular lesion seen on brain MRI or CT scan, in patients with transient focal neurologic deficit with normal MRI (transient ischemic attack) or in patients with cerebral venous thrombosis. In a multicenter study of 222 COVID-positive patients, the most common presenting clinical symptoms included: altered mental status, 52%; focal central neurologic deficit, 44%; peripheral limb weakness, 12%; and headache, 11%, with less common head and neck related symptoms including cranial neuropathy, 5%; anosmia, 3%; ageusia, 1.8%.[1] The main imaging finding in brain tissue for these entities will include focal, well-defined areas of brain edema manifesting as CT hypodensity on CT or T2-weighted hyperintensity on MRI.

There are rare case reports of COVID-associated rhombencephalitis. The majority of brain-associated COVID manifestations in the literature involve the cerebrum.[2] Acute necrotizing rhombencephalitis without cerebral lesions has been reported in a SARS-CoV-2 infected patient who presented with pulmonary artery and iliac vein thrombosis.[3] In addition, a rare case of rhombencephalitis temporally associated with Comirnaty vaccination have also been reported.[4] CT findings of rhombencephalitis include brainstem and cerebellar patchy hypodensity. MRI is superior to CT scan due to inherent streak artifact from the dense bone of the posterior fossa and skull base. MRI findings include T1-weighted hypointensity, T2-weighted and T2 FLAIR hyperintensity with associated diffusion-weighted restriction (Figure 1). The enhancement pattern may be linear, heterogeneous and in some cases, include ring enhancement if abscesses are present. Necrotizing rhombencephalitis is evident when these above findings are accompanied by blood products. This may manifest as hyperdensity on CT scan or blood products on the MRI scan which may yield susceptibility-weighted hypointensity on follow up imaging if acute/subacute blood products are not identified on acute presentation.

Stroke affects up to 3% of patients presenting with SARS-CoV-2 infection and up to 6% of patients admitted to the ICU.[2] Proposed pathogenesis in the creation of thrombi includes hypercoagulable state, cytokine storm, endotheliopathy, arterial dissection and vasculitis-like mechanism. In addition, cardiac emboli due to arrhythmias are reported in 10% of hospitalized patients and up to 40% of ICU patients.[5] Of all patients diagnosed with COVID-19 presenting with stroke, 35% will involve the posterior fossa and vertebrobasilar circulation (Figure 2).[5] Hemorrhagic stroke is reported between 22% and 26% of SARS-CoV-2 infected patients presenting with stroke.[5]

The earliest sign of ischemic infarct on CT scan may be the so called "dense vessel" sign, which in the case of CT neck, includes vertebral or basilar artery supply to the posterior fossa. Within a few hours of stroke onset, there may be loss of differentiation between the grey matter and white matter with effacement of sulci and/or cerebellar folia. As the area of ischemia becomes more hypodense, demarcation of the infarct margin becomes more conspicuous. As the stroke progresses from the acute to subacute stage, further delineation of the stroke makes it even easier to appreciate. Ischemic stroke may become hemorrhagic over time. The MRI scan offers the advantage of evaluating the movement of water molecules in the brain tissue with diffusion weighted imaging (DWI). This will detect the earliest ischemia, prior to detection on CT Images, as the movement of water molecules in and out of brain cells is disrupted, defining the infarct core. Within hours, T2-weighted signal increases. Within 2-3 weeks, the T2-weighted signal decreases. As swelling decreases, volume loss and encephalomalacia take form, yielding a chronic infarct. Susceptibility-weighted imaging (SWI) is superior to CT in the evaluation of hemorrhagic transformation of the infarct.

Orbit: *optic neuritis*

Orbital manifestations of COVID-19 such as optic neuritis have been reported during the acute infection and post recovery phase.[6,7] The exact pathophysiology is unclear, and various mechanisms proposed include sequelae of proinflammatory state with hypercoagulability and cytokine storm, result of systemic abnormalities including hypoxia and severe hypertension, or direct viral invasion.[8,9] Patients may present with sudden decline in visual acuity, eye or periorbital pain, or headaches. On MR imaging, there is T2 hyperintensity in the optic nerves, which may be unilateral or bilateral. Contrast enhancement may be present as well. Optimal technique to assess this is MRI orbits with and without contrast.

Sinonasal: *anosmia, olfactory bulb, mucormycosis*

Anosmia and dysgeusia have been reported as common early symptoms in patients with COVID-19, occurring in greater than 80% of patients in 1 series.[10] Imaging findings, however, are not commonly observed. MR imaging findings may show increased T2 signal in the olfactory bulbs and tracts with or without contrast enhancement, but this has only been demonstrated in 19% of patients in 1 series.[11] Thin section T2-weighted and post-contrast T1-weighted coronal images through the anterior cranial

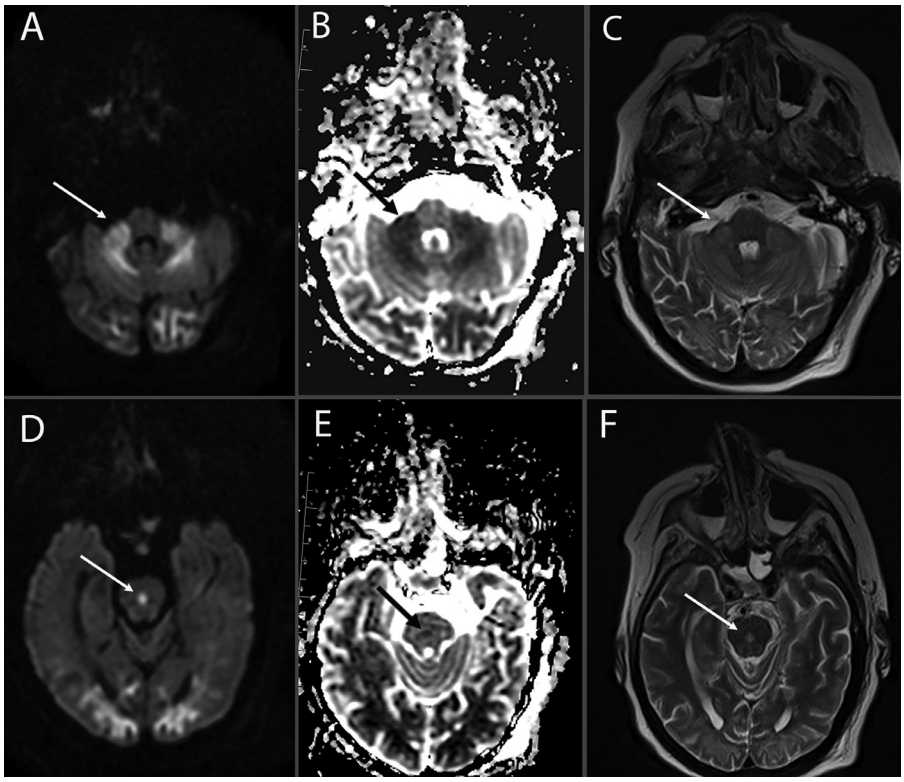

Figure 1 Encephalitis: Acutely ill 60-year-old female with known COVID-19, presents with worsening headache, progressing weakness, seizure and mental status changes. Axial MRI images include (A) Diffusion weighted image (DWI); (B) Apparent diffusion coefficient (ADC); and (C) T2-weighted image through the level of the middle cerebellar peduncles (arrows) showing bilateral, symmetric edema and diffusion restriction consistent with acute inflammation expected with viral encephalitis. (D) DWI; (E) ADC and (F) T2-weighted MRI images through the level of the pons demonstrate both pontine (arrows) and bilateral occipital lobe signal abnormality as described in A-C above. This patient did not demonstrate hemorrhage, or enhancement, which can be present to varying degrees in viral encephalitides. Work up for other infectious agents was negative. Given known COVID diagnosis and eventual precipitous demise, the presumptive diagnosis of COVID-associated encephalitis was ascribed. *Images courtesy of Dr. David D. Pasquale, MD.

fossa are optimal to demonstrate the olfactory apparatus. Targeted imaging protocols are required if this entity is suspected clinically as these sequences are not typically included on routine MRI of the brain. Other imaging findings include olfactory bulb atrophy after COVID-19 induced anosmia in patients with prolonged postinfectious anosmia. This may reflect persistent, chronic olfactory bulb injury in patients with persistent symptoms.[12]

COVID-19-associated mucormycosis (CAM) is an opportunistic fungal infection seen most often in severe and critical COVID-19 patients who underwent steroid treatment for COVID-19 during the inflammatory phase of disease. Several factors have been implicated in CAM, which include glucocorticoids use, poor blood glucose control and viral-induced lymphopenia.[13] A case series review revealed that most cases were reported from India (72%), affected male patients (78%), had diabetes mellitus (85%), had glucocorticoid use (85%), and had severe or critical COVID-19 (67%). The median time interval between COVID-19 diagnosis and the first evidence of mucormycosis infection was 15 days.[13] About 37% of patients had mucormycosis after an initial recovery from COVID-19. Rhino-orbital mucormycosis was most common (42%), followed by rhino-orbito-cerebral mucormycosis (24%) and nose and/or paranasal sinus (16%). The mortality rate was 34%, similar to non-COVID related mucormycosis.[13] Imaging modality of choice is MRI with contrast with complementary CT to assess bony involvement. Radiological appearances of CAM are similar to invasive fungal rhinosinusitis (Figure 3). The classic imaging feature is lack of contrast enhancement of invaded mucosa secondary to occlusion of small vessels, often referred as the "black turbinate" sign.[14] In disseminated and advanced craniofacial disease, non-enhancing devitalized and necrotic soft tissue at the orbits and central skull base can be seen. An important observation is that multicompartmental and extrasinonasal tissue infarction is possible without overt bone involvement, as fungal elements can disseminate from the nasal cavity via perivascular and perineural routes. Since surgery such as sinonasal debridement as an adjunct to antifungal therapy is associated with higher survival rates,[13] and the debridement surgery needs to be performed before the infection spreads to other adjoining anatomy such as the brain, prompt imaging is essential in the management.

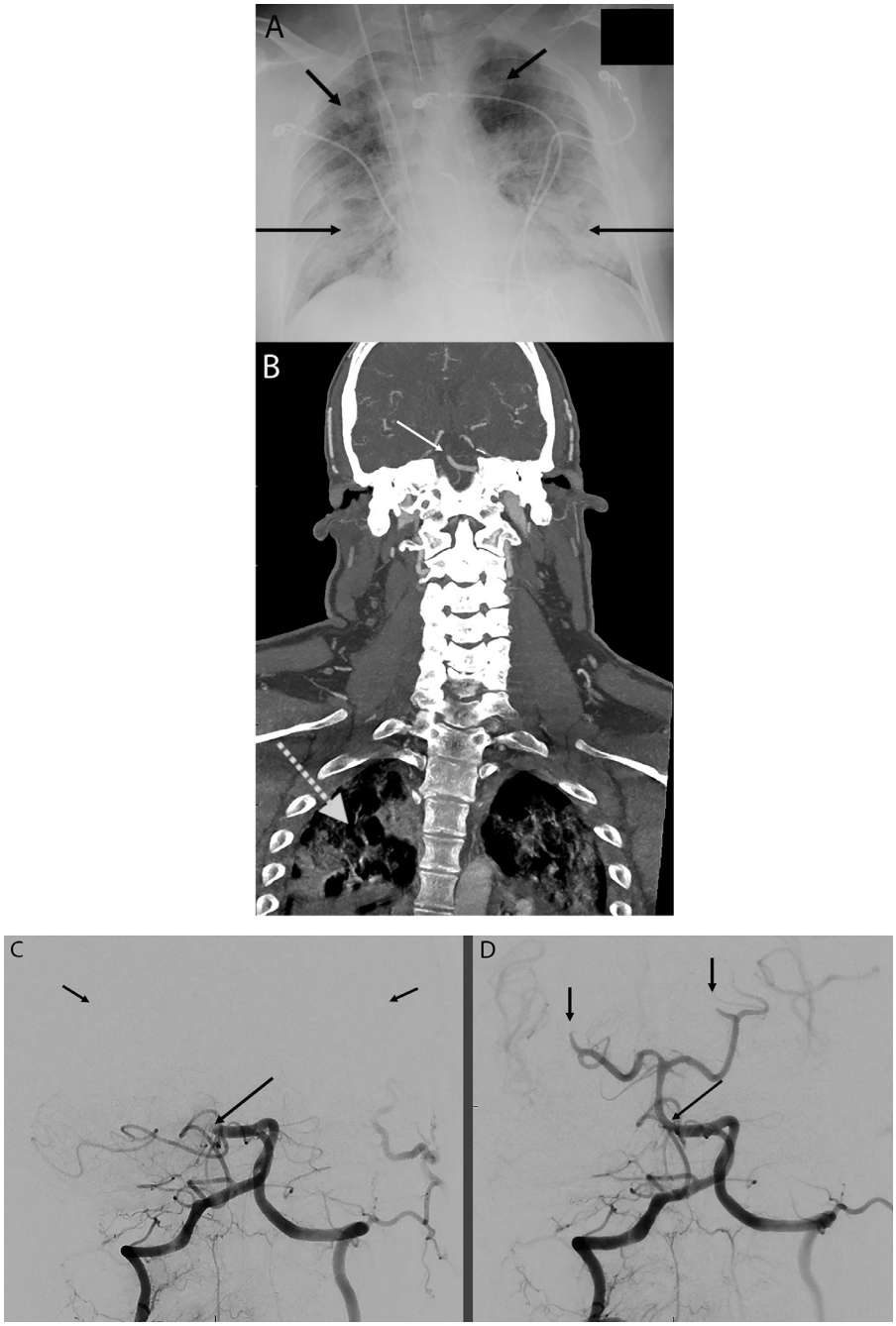

Figure 2 Thrombosis of the basilar artery: 49-year-old male with recent diagnosis of COVID-19 pneumonia, saturating at 50-60% oxygen carrying capacity, presents with headache, progressing weakness, seizure and AMS. Worsening respiratory failure and right body numbness and hemiparesis, progress to eventual coma. Final diagnosis was basilar artery occlusion syndrome. (A) Portable chest radiograph demonstrates bilateral lower lung air space opacities (long black arrows) with bilateral upper lung involvement (short black arrows). (B) CTA head and neck demonstrate the suspected basilar artery occlusion (solid white arrow). COVID-19 pneumonia is again seen in lung apices (dashed white arrow). (C) Thrombosis of the proximal basilar artery (long black arrow) is demonstrated on frontal radiographic projections of the posterior circulation during contrast injection in the right vertebral artery from mechanical thrombectomy procedure. The superior cerebellar and occipital circulation does not opacify (short black arrows) due to proximal occlusion. (D) After successful recanalization of the basilar occlusion, the basilar artery is now patent (black arrow), and the major vertebrobasilar vessel origins are seen. The distal posterior cerebellar and cerebral arteries do not fill, likely due to distal propagation of thrombus (short black arrows). *Images courtesy of Dr. David D. Pasquale, MD.

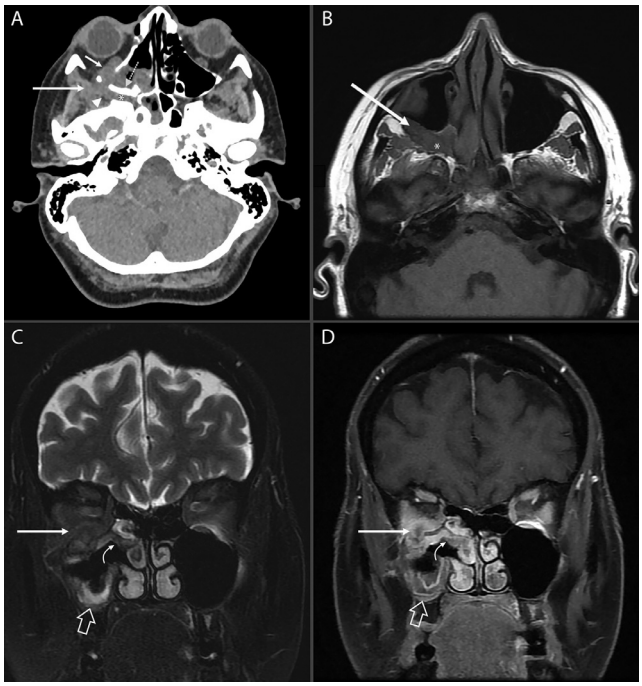

Figure 3 Rhino-orbital mucormycosis: 70-year-old man with history of follicular lymphoma status post chemotherapy, in remission, immunocompromised. Presents urgently with right orbital pain, proptosis and swelling, COVID positive and oxygen dependent. Failed first-line anti-fungal therapy. Subsequently underwent multiple surgeries to include: nasal endoscopy, right maxillectomy, sphenoidectomy, orbital decompression, resection of pterygopalatine fossa and pterygoid plates (postoperative images not shown). Lymphoma recurrence suspected on presentation; later pathology proven invasive fungal disease. (A) Axial, unenhanced CT of the head through the level of the orbits shows a mass centered in the right maxillary sinus invading the orbit (small white arrow), inducing mucoperiostial thickening of the adjacent maxillary sinus bone (dashed white arrow), with extension of soft issue into the right retroantral fat and masticator space soft tissues (long white arrow), and the pterygopalatine fossa (white asterisk). (B) T1-weighted non contrast axial image at the level of the pterygopalatine fossa shows similar soft tissue mass extending from right maxillary sinus into the retroantral fat and right masticator space (white arrow) in addition to the pterygopalatine fossa (white asterisk). (C) and (D) T2-weighted and T1-weighted post-contrast coronal images, respectively, through the orbits and middle meatus demonstrate the hypointense fungal elements invading the orbit from the maxillary sinus (white arrow). Non-enhancing mucosal soft tissue extending through the middle meatus toward the middle turbinate (curved white arrow) consistent with fungal invasion of the mucosa. Similar non-enhancing mucosal soft tissue in the inferior right maxillary sinus (white open arrow).

Audio-vestibular disorders

Audio-vestibular disorders in patients with COVID-19 have been reported. In 1 systematic review,[15] sensorineural hearing loss was found to be the most frequent audio-vestibular symptom described, occurring alone or in association with tinnitus and vertigo. IAC protocol MRI with and without contrast is the imaging modality of choice in these settings. While imaging is often negative, imaging features include CNVIII or cochlear enhancement (vestibulocochlear neuritis), intralabyrinthine enhancement (labyrinthitis), or intrinsic T1 hyperintensity of the membranous labyrinth (intralabyrinthine hemorrhage) (Figure 4).

Parotitis and xerostomia

The most common reported symptoms of COVID-19 include fever, dry cough, fatigue, muscle soreness, and headache.[16] As clinicians become more aware of atypical presentations, they contribute to the early detection and prevention of disease transmission. A case report describes a patient presenting 1 week prior to the ED with cough, dyspnea, and fever, with the diagnosis of COVID-19. Despite improvement of upper respiratory symptoms, she presented to another ED with symptoms of progressing parotitis and imaging findings including diffuse asymmetric swelling of unilateral parotid gland with adjacent periglandular fat infiltration absent sialolith, mass, infection, or abscess.[17] This was accompanied by progressive unilateral facial and neck swelling causing subjective malocclusion and trismus, resulting in decreased oral intake. Pertinent differential considerations include infectious parotitis, sialolithiasis, salivary gland abscess and neoplasm. Obstructing stone, mass, or abscess were excluded on the CT scan. Trismus resulted from inflammation surrounding the muscles of mastication. Free fluid may extend into surrounding anatomical neck spaces (Figure 5).

A variety of respiratory viral diseases can result in non-mumps parotitis including enteroviruses, influenza, parainfluenza, coxsackievirus, and Epstein-Barr viruses.[18] Viral parotitis can present with viral prodrome including flulike symptoms followed by 2-4 days of gradual swelling of both parotid glands, though unilateral involvement can be present in up to 25% of cases.[18] Acute suppurative parotitis can present with sudden onset of unilateral pain, swelling of the parotid gland and purulent discharge from Stenson's duct orifice upon massage of the gland.[18]

Although not seen on imaging, a brief mention of clinical xerostomia is warranted following the description of COVID-associated parotitis. Although medications and radiotherapy are the leading causes of dry mouth—due to insufficient saliva production by salivary glands—xerostomia has been reported in the literature in association with patients infected with SARS-CoV-2. Although the mechanism of alterations in saliva production in patients with COVID-19 is unclear, it is proposed that glandular infection with SARS-CoV-2 contributes to these presentations.[19] Angiotensin converting enzyme 2 (ACE2) receptor mediates entry of SARS-CoV-2 into the cell and ACE2 receptors were found in ductal elements of salivary glands. This provides a possible mechanism of linkage between COVID-19 patients and xerostomia.[19,20]

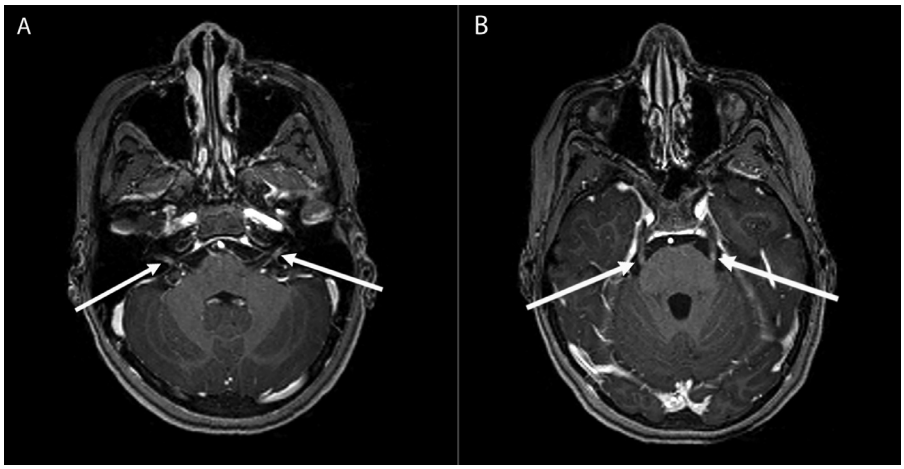

Figure 4 A 11-year-old girl COVID-19 positive 3 days prior presents with tinnitus and cranial neuropathy. This was followed by 4 days of fever, nausea, vomiting, ear pain, and diffuse abdominal pain with initial concern for multisystem inflammatory syndrome in children (MIS-C) –also known as pediatric inflammatory multisystem syndrome (PIMS)—in a patient with known COVID, elevated D-dimer of 936, lipase of 130, and whose course was complicated by ongoing fevers, worsening acute mental status changes. Lumbar puncture and clinical data suggesting possible encephalitis (infectious vs autoimmune). (A) Axial post contrast T1-weighted image shows bilateral IAC nerve root enhancement likely related to cochlear and/or vestibular cochlear nerves (white arrows) consistent with neuritis, likely contributing to tinnitus. (B) Axial post contrast T1-weighted image show bilateral trigeminal nerve, cisternal segment enhancement, consistent with trigeminal neuritis. *Images courtesy of Dr. David D. Pasquale, MD.

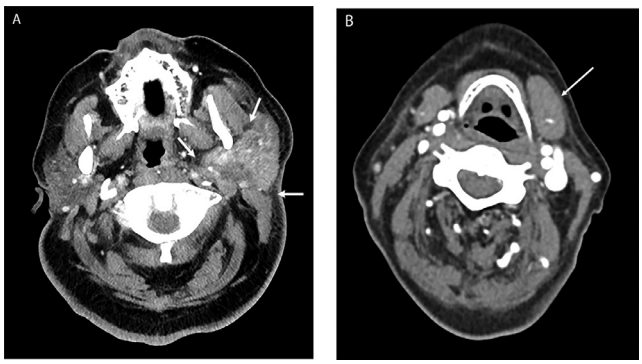

Figure 5 COVID-19 associated sialadenitis: Previous diagnosis of COVID-19, mild URI and headache. Admitted 5 days later with left sialoadenitis, bulging parotid and submandibular gland, facial pain, swelling and fever. (A) Enlarged left parotid gland with inflammation of the surrounding fatty soft tissue planes (white arrows). No salivary duct stones seen. (B) Ipsilateral, acalculous, left submandibular gland swelling (white arrow). *Images courtesy of Dr. David D. Pasquale, MD.

Lymphadenopathy

Post vaccine adenopathy after deltoid injection may present as ipsilateral supraclavicular or axillary adenopathy on imaging, or as a hypermetabolic focus on PET-CT mimicking nodal metastasis.[21] Vaccination-associated adenopathy is an imaging finding after administration of many vaccines and is not unique to COVID-19 vaccines. However, during mass vaccination effort due to a pandemic, frequency of post vaccination adenopathy may increase [22] and lead to more diagnostic conundrum in patients, particularly in those with history of or suspected suspicion of cancer (Figure 6).

Recommendation guidelines for imaging regarding post-vaccine adenopathy have been developed by a multidisciplinary panel from tertiary care cancer centers in the United States.[23] According to these recommendations, routine imaging examinations such as those for screening, should be scheduled before or at least 6 weeks after the final vaccination dose to allow for any reactive adenopathy to resolve. However, urgent clinically indicated imaging (eg, for acute symptoms, short-interval treatment monitoring, urgent treatment planning or complications) should not be delayed due to prior vaccination. The vaccine should be administered on the side contralateral to the primary or suspected cancer, and both doses should be administered in the same arm. Detailed vaccination information (eg, dates administered, injection site, laterality, and type of vaccine) should be included in patient imaging questionnaire, and this information should be made readily available to interpreting radiologists. Currently, there are no standardized system and different institutions will ask for this information depending on what type of examination is being performed, in particular those that cover the axilla, neck and chest regions. For example, at our institution, mammography, shoulder musculoskeletal or brachial plexus imaging will require detailed vaccination history. Therefore, an effective management will depend on good coordination of the referring physician, patient vaccination record, and radiologists, when managing adenopathy in the setting of COVID-19 vaccination.

Clinicians should be aware that incidental post vaccination adenopathy is more common during vaccination efforts. Vaccination should not be delayed due to upcoming imaging, and imaging should be scheduled before the first, or at least 6 weeks after, the final vaccination dose whenever possible.

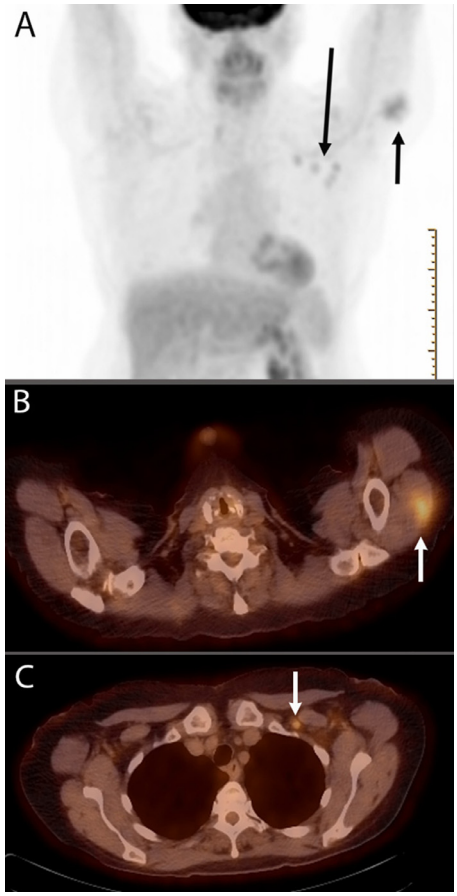

Figure 6 Post vaccination adenopathy: Mildly tender unilateral adenopathy incidentally found on routine PET/CT for bladder cancer staging 1 day post first COVID-19 vaccine. (A) PET/CT maximum intensity frontal projection demonstrates increased radiotracer uptake in the known left deltoid injection site (short black arrow) and left subpectoral lymphadenopathy (long black arrow). (B) Left deltoid injection site, and (C) Left subpectoral site confirmed on the fused, axial PET/CT images. *Images courtesy of Dr. David D. Pasquale, MD.

Vascular Thrombosis

The CT scan of the neck often includes the posterior fossa, the transverse and sigmoid sinuses. Oftentimes, the straight sinus, vein of Galen and cavernous sinus, and less often, portions of the high superior sagittal sinus are also included. The association between venous thrombosis in patients with COVID-19 is well documented in the literature.[24] Relevant to this communication, we will review mainly venous thrombosis with brief mention of arterial thrombosis.

Venous Thrombosis

In several reported cases, the COVID-19 symptoms preceded the clinical onset of venous sinus thrombosis by approximately 2 weeks.[25] This may imply that the hypercoagulability caused by SARS-CoV-2 infection may persist after the symptoms of COVID-19 resolve. There are a myriad of mechanisms by which SARS-CoV-2 is postulated to promote coagulation. First, D-dimer, fibrinogen, and fibrin degradation products are elevated relative to healthy controls.[26] Additionally, the SARS-CoV-2 interaction with the ACE2 receptor may result in endothelial injury, thereby promoting a hypercoagulable state.[26] The imaging manifestations of venous thrombosis on unenhanced CT of the neck may include a hyperdensity within the venous structure, for example a draining venous sinus intracranially.[27] This dense venous structure can persist for up to 1 to 2 weeks. Contrast administration is recommended when evaluating thrombosis of venous structures as it best delineates the thrombus as a filling defect within the lumen.

Catheter angiography, previously the gold standard in the evaluation of venous thrombosis, is now rarely necessary for diagnosis, supplanted by the introduction of CT and MR venography. The CT venogram of the neck and head is performed with injection of 100 mL of intravenous nonionic contrast medium (iodine, 300 mg/mL) at a rate of 3 mL/ sec with a 45-second delay between intravenous injection and the start of scanning.[28] Common, benign filling defects in the draining venous sinuses of the posterior fossa include arachnoid granulations. These often appear round, well-marginated and do not obstruct venous flow.

Cerebral Venous Thrombosis

Cerebral venous thrombosis is an uncommon subtype of stroke with an annual incidence of 2 to 5 cases per million.[24] This entity has a predilection for younger patients, particularly women.[29] The clinical presentation of headache is most common, followed by seizures and focal neurologic deficits. Cerebral venous thrombosis progresses to venous infarct in approximately 50% of cases.[28] Cerebral venous infarction results from thrombus propagation into draining cortical veins, causing significant extravasation of fluid into the brain resulting in vasogenic edema, focal cerebral edema and hemorrhage.[30] In 1 systematic review and meta-analysis of 57 patients with COVID-19, the most common locations of cerebral venous thrombosis included: transverse sinus, 65%; sigmoid sinus, 47%; superior sagittal sinus, 44%; deep and/or central vein, 37%; straight sinus, 21%; and cortical vein, 21% (sum of percentages >100% as patients may present with multiple occluded venous structures in 1 setting).[25] Approximately 40% of patients manifested local cerebral edema, 37% went on to venous infarction and 42% manifested parenchymal hemorrhage.[25] Hemorrhage secondary to cerebral venous thrombosis is related to the location of draining veins and suspected when bleeding does not correspond to arterial territories (Figure 7).

Cavernous Sinus Thrombosis

The cavernous sinus collects venous drainage intracranially and from orbits, midface, and neck. This may predispose to infection via thrombophlebitis or septic

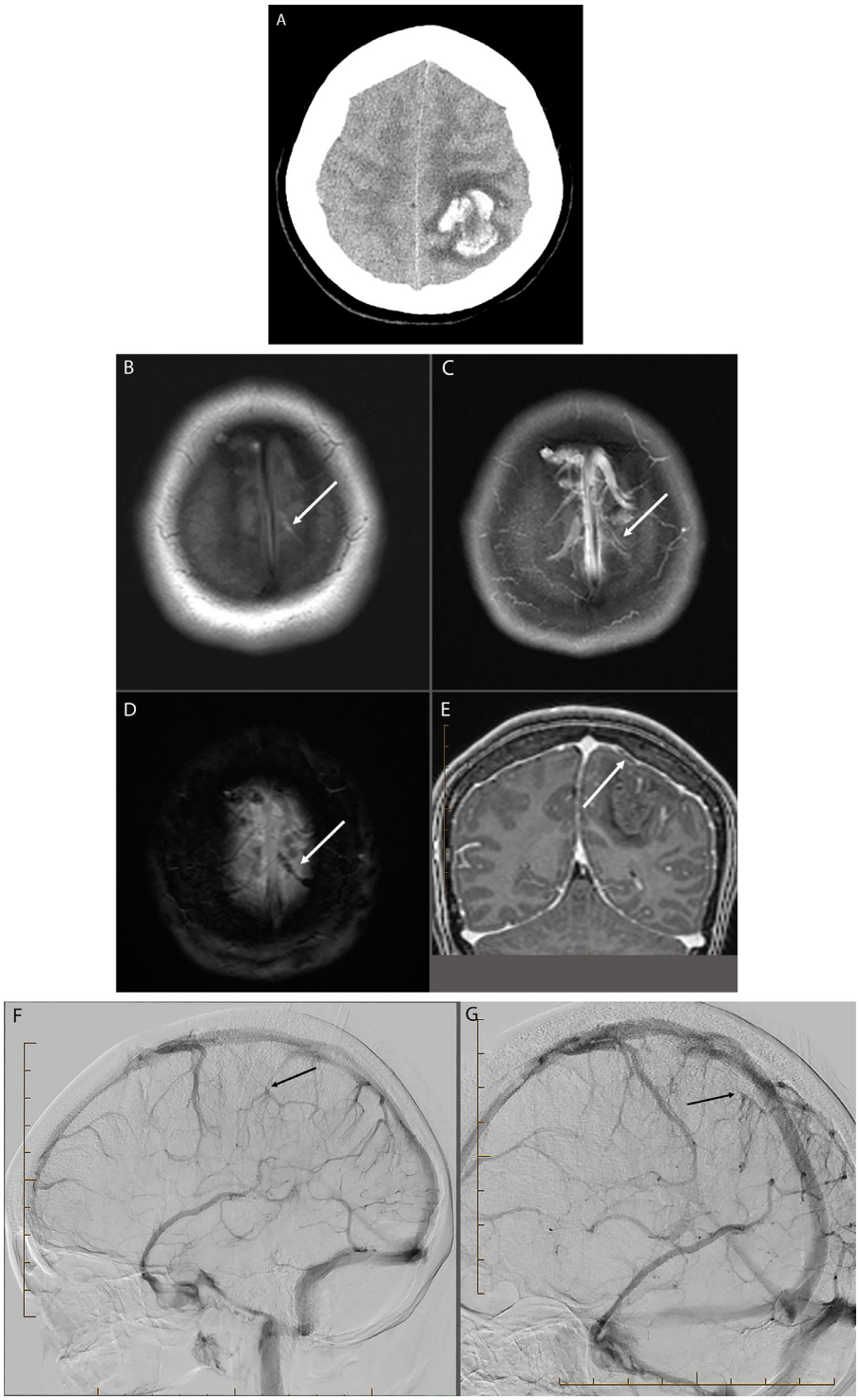

Figure 7 Cerebral venous thrombosis: 25-year-old female with headache, recently COVID positive. (A) Head CT showed left parietal lobe hemorrhage. (B) Unenhanced T1-weighted axial image at the vertex of the head shows a linear hyperintensity (white arrow) to the left of midline, corresponding to a superficial left anterior parietal cortical vein suggesting subacute thrombus. (C) Post-contrast T1-weighted axial image at the same level demonstrates the same vein containing a filling defect (white arrow) suspicious for presence of thrombus. (D) Axial susceptibility-weighted image (SWI) at the same level confirmed blood products in this cortical vein (white arrow). (E) Coronal T1-Weighted post-contrast image from reformatted volumetric acquisition demonstrates the filling defect (white arrow), supporting the diagnosis of CVT. Diagnostic catheter cerebral angiogram to evaluate the etiology of hemorrhage. (F) Lateral projection from the venous phase of catheter angiogram after injection of the left ICA shows an abrupt cut-off (black arrow) of the left parietal cortical draining vein. (G) Right anterior oblique projection of the same injection again shows the abrupt cut-off (black arrow) of the same left parietal cortical vein consistent with CVT. *Images courtesy of Dr. David D. Pasquale, MD.

emboli. Aseptic or noninfectious cavernous sinus thrombosis includes hypercoagulable disorders such as malignancy, pregnancy, arteriovenous malformations, and inflammatory conditions such as Tolosa–Hunt syndrome. The symptoms of cavernous sinus thrombosis can overlap with other common ophthalmic presentations, such as orbital cellulitis and orbital apex syndrome. Common symptoms include ptosis, proptosis, chemosis, ophthalmoplegia (cranial nerve III, IV, VI), and loss of sensation in the ophthalmic and maxillary divisions of the trigeminal nerve (V1 and V2). Rarely, visual loss can occur in this entity due to optic nerve ischemia, retinal ischemia, and anterior ischemic optic neuropathy.[29] An Italian study followed 85 COVID-19 positive patients in a single hospital, treated with thromboprophylaxis on admission. Forty-three patients developed venous thromboembolism. Seven of these involved the internal jugular vein.[31] The overall incidence of thrombotic events in patients infected with SARS-CoV-2 is as high as 79%.[32]

Arterial thrombosis

The incidence of arterial thrombosis in patients with COVID-associated hypercoagulability remains low, approximately 3.7%.[33] Internal carotid artery thromboembolism is reported in the literature mainly as isolated case reports.[34-37](Figure 2)

Guillain-Barrè Syndrome

Guillain-Barrè syndrome (GBS) is a rare disease representing the most frequent cause of acute flaccid symmetrical weakness of the limbs and areflexia usually reaching its peak within a month. The etiology and pathogenesis remain largely enigmatic and the syndrome results in death or severe disability in 9%-17% of cases despite immunotherapy.[38] In general terms, GBS encompasses a wide range of clinical syndromes with an acute inflammatory polyradiculoneuropathy, muscle weakness, and reduced reflexes.[38] Guillain-Barré syndrome is diagnosed by the combination of clinical presentation, CSF study, and electrophysiological criteria. CSF abnormalities are characterized by increased protein without pleocytosis, which is a non-specific finding, seen in many of the conditions which mimic GBS on imaging and clinically. Nerve conduction abnormalities include slow or blocked nerve conduction, prolongation of distal latency, and f-waves.[39]

The modality of choice for evaluating GBS is contrast-enhanced MRI. It is essential that contrast be administered if the diagnosis is suspected as non-contrast sequences are essentially normal.[40] Typical imaging findings in Guillain-Barré syndrome are surface thickening and contrast enhancement on the conus medullaris and the lumbar nerve roots of the cauda equina.[40] The most common site of enhancement in Guillain–Barré syndrome is the anterior nerve roots, although enhancement of the posterior nerve roots is also seen.[40] In the brain, the facial nerve (CN VII) is the most commonly affected cranial nerve.[39] T2-weighted STIR sequences may demonstrate asymmetrical thickening and hyperintensity of post-ganglionic roots supplying the brachial and lumbar plexuses on STIR sequences.[41] Brainstem and cervical spine meningeal enhancement can also be seen on post contrast T1-weighted sequences.

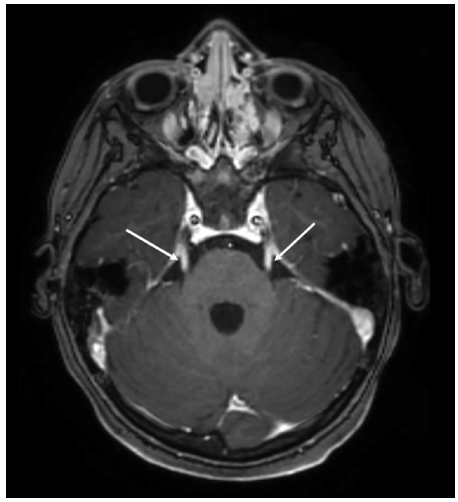

Figure 8 A 4-year-old female with COVID-19, suspected Guillain-Barré: Axial, post-contrast, T1-weighted image at the level of the cisternal trigeminal nerve as it enters the pons shows bilateral cranial nerve V enhancement (white arrows), confirms clinical suspicion of Guillain-Barré syndrome.

A review of 45 patients with COVID-19 from 29 published articles demonstrated clinical presentations including paresthesia or pain, limb weakness, and cranial nerve symptoms.[42] Although most MRI brain findings were normal, cranial nerve (especially facial nerve) enhancement was the most common abnormal finding.(Figure 8)[42] Abnormal spine findings included lumbosacral root enhancement, radiculitis and brachial and/or lumbosacral plexitis, leptomeningeal enhancement and myelopathy.[42] CT is not the modality of choice to evaluate Guillain-Barrè syndrome as the sensitivity for the evaluation and enhancement of nervous tissues is much greater with MRI.

Pulmonary

The lung apices are included in all CT scans of the neck, and as such a brief discussion of the lung findings in patients infected with SARS-CoV-2 presenting with COVID-19 is warranted. CT sensitivity seems to be high in patients with positive RT-PCR.[43] Typical CT findings in individuals with COVID-19 were ground-glass opacities, particularly on the peripheral and lower lobes, and bilateral multiple lobular and subsegmental areas of consolidation, especially in ICU patients.(Figure 9)[44] It is important to realize that CT is not the standard for the diagnosis of COVID-19, but its findings help suggest the diagnosis in the appropriate setting. A single-center study of 205 CT scans performed for reasons other than COVID-19

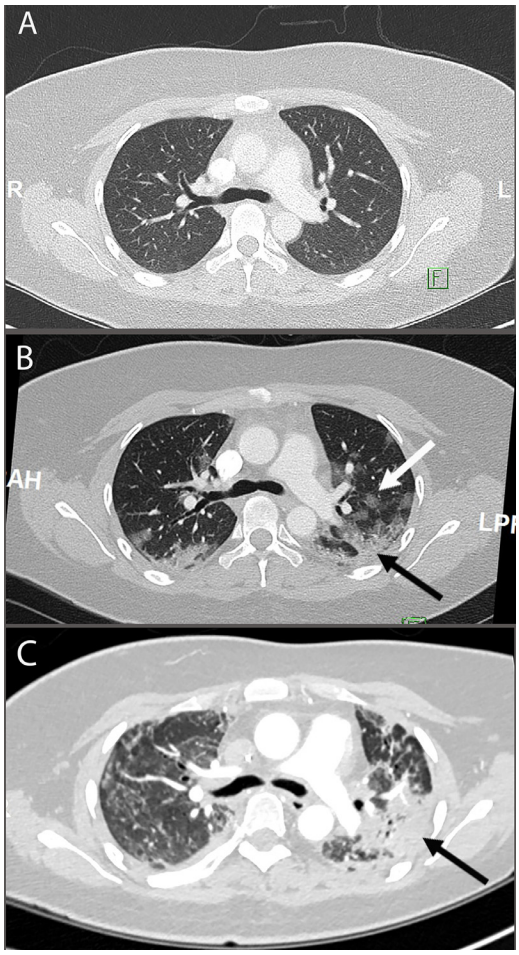

Figure 9 COVID-19 pneumonia: (A) March 2021. Normal upper lung in patient undergoing surveillance for head and neck cancer. (B) May 2021. One week of dizziness, fatigue, productive cough, fevers, and abdominal pain prior to diagnosis of COVID-19 infection. Develops areas of ground-glass opacities (white arrow) in the posterior lung with peripheral nodularity, consolidation and atelectasis (black arrow). (C) October 2021. Diffuse ground-glass opacities, bronchiectasis compatible with post-COVID ARDS and developing pulmonary fibrosis. Consolidation in the left upper lobe suspected superimposed secondary infection (black arrow).

yielded incidental lung findings leading to the suspicion of COVID-19. Six patients (3%) were confirmed COVID-19 positive by RT-PCR, with the majority of patients experiencing mild to moderate symptoms.[45] It is crucial to correlate lung CT findings with epidemiologic history, clinical presentation, and RT-PCR test results.[46]

Summary

The SARS-CoV-2 virus is a formidable infectious agent due to its ease of transmission and its potential for devastation of at-risk patient populations. Although head and neck imaging is often not performed for the purpose of COVID-19 diagnosis, abnormalities involving the anatomy described in this communication can sometimes raise the suspicion of COVID-19 if the appropriate clinical questions and testing is subsequently pursued. This combination of radiologic and clinical awareness will protect the community by identifying infected patients early and inserting them into the proper treatment regimen.

Declaration of conflict of interest

All authors declare no conflict of interests.

Disclosure

The authors reported no proprietary or commercial interest in any product mentioned or concept discussed in this article.

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