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Imaging of the head and neck during the COVID19 pandemic

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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.

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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-Barré 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
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. 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. 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. 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%. 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. 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. In addition, a rare case of rhombencephalitis temporally associated with Comirnaty vaccination have also been reported. 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. 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. Of all patients diagnosed with COVID-19 presenting with stroke, 35% will involve the posterior fossa and verteobasilar circulation (Figure 2). Hemorrhagic stroke is reported between 22% and 26% of SARS-CoV-2 infected patients presenting with stroke.

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. 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. 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.

**Sino nasal: 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. 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. Thin section T2-weighted and post-contrast T1-weighted coronal images through the anterior cranial
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 multicompartamental and extrasinosal 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 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.
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.
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. 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 perilobular fat infiltration absent sialolith, mass, infection, or abscess. 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-numps parotitis including enteroviruses, influenza, parainfluenza, coxsackievirus, and Epstein-Barr viruses. 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. Acute supplicative 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.

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. 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.

Audio-vestibular disorders

Audio-vestibular disorders in patients with COVID-19 have been reported. In 1 systematic review, sensorineural hearing loss was found to be the most frequent audiovestibular 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).
**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.

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**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.*
iad 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. Additionally, the SARS-CoV-2 interaction with the ACE2 receptor may result in endothelial injury, thereby promoting a hypercoagulable state. 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. This dense venous structure can persist for up to 1 to 2 weeks. Contrast administration is recommended when evaluating thrombus 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. Common, benign filling defects in the draining venous sinuses of the posterior fossa include arachnoid granulations. These often appear round, well-margined 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. This entity has a predilection for younger patients, particularly women. 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. 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. 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). Approximately 40% of patients manifested local cerebral edema, 37% went on to venous infarction and 42% manifested parenchymal hemorrhage. 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).

Venous Thrombosis

In several reported cases, the COVID-19 symptoms preceded the clinical onset of venous sinus thrombosis by approximately 2 weeks. This may imply that the hypercoagulability caused by SARS-CoV-2 infection may persist after the symptoms of COVID-19 resolve. There are a myr-
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 thrombo-
sis includes hypercoagulable disorders such as malignancy,
pregnancy, arteriovenous malformations, and inflammatory
conditions such as Tolosa–Hunt syndrome. The symptoms
cavernous sinus thrombosis can overlap with other com-
onal 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, vi-

sual loss can occur in this entity due to optic nerve isch-
emia, retinal ischemia, and anterior ischemic optic neu-
ropathy. An Italian study followed 85 COVID-19 positive
patients in a single hospital, treated with thromboprophyl-
laxis on admission. Forty-three patients developed venous
thromboembolism. Seven of these involved the internal
jugular vein. 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, ap-
proximately 3.7%. 33 Internal carotid artery thromboem-
bolism is reported in the literature mainly as isolated case
reports. (Figure 2)

Guillain–Barré Syndrome

Guillain–Barré syndrome (GBS) is a rare disease repres-
enting the most frequent cause of acute flaccid symmet-
rical weakness of the limbs and areflexia usually reach-
ing its peak within a month. The etiology and pathogen-
esis remain largely enigmatic and the syndrome results
in death or severe disability in 9%-17% of cases despite
immunotherapy. In general terms, GBS encompasses a
wide range of clinical syndromes with an acute inflam-
matory polyradiculoneuropathy, muscle weakness, and re-
duced reflexes. Guillain–Barré syndrome is diagnosed by
the combination of clinical presentation, CSF study, and
electrophysiological criteria. CSF abnormalities are char-
acterized 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. Typical imaging findings in Guillain–
Barré syndrome are surface thickening and contrast en-
hancement 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. In the brain, the facial nerve (CN

VII) is the most commonly affected cranial nerve. 39 T2-
weighted STIR sequences may demonstrate asymmetri-
cal thickening and hyperintensity of post-ganglionic roots
supplying the brachial and lumbar plexuses on STIR se-
quences. Brainstem and cervical spine meningeal en-
hancement can also be seen on post contrast T1-weighted
sequences.

A review of 45 patients with COVID-19 from 29 pub-
lished articles demonstrated clinical presentations includ-
ing paresthesia or pain, limb weakness, and cranial nerve
symptoms. 42 Although most MRI brain findings were nor-
mal, cranial nerve (especially facial nerve) enhancement
was the most common abnormal finding. (Figure 8) 42 Ab-
normal spine findings included lumbosacral root enhance-
ment, radiculitis and brachial and/or lumbosacral plexitis,
leptomeningeal enhancement and myelopathy. CT is not
the modality of choice to evaluate Guillain–Barré 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. 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) 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 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.
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. It is crucial to correlate lung CT findings with epidemiologic history, clinical presentation, and RT-PCR test results.

**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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