

Imaging of Lemierre Syndrome in children and young adults

A case-series analysis

Lysiane Trachsel, 2nd year student in Master of Medicine at the University of Lausanne
Prof. François Gudinchet, Department of Medical Radiology
Dr. Med. Jacques Cherpillod, Department of Otorhinolaryngology

Centre Hospitalier Universitaire Vaudois
Rue du Bugnon 46
1011 Lausanne

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Abstract

Background / Purpose :

Lemierre Syndrome (LS) is defined by a recent oro-pharyngeal infection, the clinical presence or radiological demonstration of internal jugular vein (IJV) thrombosis and documented anaerobe germ, principally *Fusobacterium necrophorum* (Fn) leading to septicaemia and septic embolization. It is a rare infection described since 1900 and it nearly disappeared since the beginning of the antibiotic era. Even if it is seldom described in the literature, this infection is reappearing in the last 10 years, either because of the increase of antibiotic resistance or by modification of antibiotic prescription. The aim of this study is to describe the role of medical imaging in the diagnosis, staging and follow up of Lemierre syndrome, as well as to describe the ultrasound (US), computed tomography (CT) and magnetic resonance imaging (MRI) findings of this rare disease.

Patients and methods :

Radiological and medical files of patients diagnosed with Lemierre syndrome in the past 6 years at CHUV hospital were analysed retrospectively. The CT scan, US, colour Doppler US (CDUS) and MRI examinations that were performed have been examined so as to define their specific imaging findings.

Results

IJV thrombosis was demonstrated in 2 cases by US, by CT in 6 cases and MRI in one case. Septic pulmonary emboli were detected by CT in 5 patients. Complications of the LS were depicted by MR in one case and by CT in 1 case.

Conclusion :

In the appropriate clinical settings, US, CT or MR evidence of IJV thrombosis and chest CT suggestive of septic emboli, should lead the physician to consider the diagnosis of LS. As a consequence, imaging allows a faster diagnosis and a more efficient treatment of this infection, which in case of insufficient therapy can lead to death.

Introduction

LS, also called post-anginal septicaemia or necrobacillosis, is a rare but mortal complication of acute tonsillitis or tonsillar abscess in previously healthy children and young adults. The first descriptions were made by Courmont et Cade in 1900, then by Schottmuller in 1918 and finally by Teissier in 1929 and 1931. Lemierre defined it in 1936¹. A week after an oropharyngeal infection, the disease evolves into septic thrombophlebitis of the ipsilateral IJV. It then embolises and forms septic metastatic abscesses in other body parts but mostly in the lungs. The most common causative germ is *Fn*.

In Lemierre's time, the disease was "so characteristic that mistake was almost impossible" with a mortality rate of nearly 90%¹. With the discovery of antibiotics and better oral hygiene, the number of cases decreased^{3,12,13}. They were less severe, but mortality remained high²⁰. Later, its incidence as well as its gravity decreased to become a forgotten complication of acute tonsillitis². LS should be reminded because of its significant morbidity and potential lethality (4-12%¹⁵).

In the last 20 years, the number of case reports in literature increased. The reason why remains unclear¹⁹. Some say it could be a consequence of change in antibiotic prescription, increasing resistance of the germ or even the improvement of germ identification methods^{6,10,16,17,19}. Today only a couple of prospective studies have been made and showed an incidence of 0.6-2.3/mio and a mortality rate of 6.4% that certainly is underestimated^{2,13,15}. Unawareness among physicians, the unclear definition and the great variability and non-specificity of the symptoms creates a real difficulty to diagnose the syndrome and thus causes delay in treatment^{2,3,5,18}.

For better outcome and to avoid heavy surgery, early diagnosis is important. Microbiology may give the diagnosis of Lemierre syndrome in most of the cases, but it has some limits. The differential diagnosis is large, the microbiologist must be aware of its characteristics and the germ needs more time than other germs to grow^{8,15}. Sometimes patients have been under ineffective antibiotherapy before hospitalisation which can alter the results and finally, the germ is often found only in the late phases of the infection. As a consequence, a negative haemoculture does not infirm a LS^{9,13}. The improvement of radiological techniques in the last years may give a better alternative. CT, US, CDUS and in some cases MRI may diagnose the IJV thrombosis and septic emboli even when clinical signs are unspecific or absent. As these patients generally are hospitalised, these radiological tools are easily available. In addition, it is not only a diagnostic method but it also shows the extent of the lesions in the various cervical compartments, the size of the thrombus in the IJV and its collaterals, and

finally it shows septic emboli in the lungs or in other body parts. They also are useful follow-up tools.

Few radiologic descriptions of this entity have been published in the literature⁵. The aim of this study is to describe the CT, US, CDUS and MRI findings of LS, and to evaluate the role of medical imaging in the diagnosis, staging and follow-up of LS.

Patients and Methods

A retrospective analysis of patients referred or diagnosed for LS to the University Hospital CHUV in Lausanne from 2005 to 2011 was performed using our institution's database (Radoffice).

The following keywords were used: LS, septic jugular thrombophlebitis, from 1 to 80 years old; 16 files matched. The criteria for case selection were the following: the disease had to start with an oro-pharyngeal infection, an IJV thrombosis or compression had to be identified as well as septic emboli. As 9 cases did not match this case description they were not taken into consideration : in four patients the initial infection was not oro-pharyngeal (otitis media or sinusitis) and in one of them the blood vessels were permeable. For two others the radiological patterns for LS were missing. In the last two cases, the infection was secondary to chemotherapy, radiotherapy or was paraneoplastic. Our series consisted in six women and one man, with a median age of 19 years (range: 3 - 24 years).

Imaging findings were studied on US, CDUS, CT scan and MRI if done, for each patient. The images were viewed and re-evaluated on a workstation (Advantage Windows software 4.3 GE Medical System) using 2D and 3D reconstructions. The diagnosis was then set by consensus. The following imaging protocols were used.

- US-doppler:

The ultrasound examinations (IU22 Philips Healthcare) were performed using Curved array C5-1 (MHz) C8-5 (MHz) and Linear array L12-5 (MHz) transducers. Gray scale study (US) of the neck were obtained to define the thrombosis in the internal jugular vein and to examine the surrounding soft tissue. CDUS images evaluated the continuous blood flow through the IJV.

- CT protocols:

Neck and chest images were acquired on a 64-detector VCT system (General Electric Medical System, Milwaukee, USA). Acquisitions were obtained after an antecubital intravenous injection of 1.5-2 ml /kg non-ionic contrast medium (Accupaque 300 or 350, GE

Healthcare, Giles, UK), with 10-20ml saline solution as bolus chaser. Helical CT acquisitions were started 30 seconds after injection, breath hold was asked when possible. 2D reconstructions were always used for measurements and 3D reconstructions (GE, Advantage Windows workstation, 4.3 software) were made when necessary.

Table 1 summarizes the 64-detector-row CT protocols that were used with subsequent CT dose index (CTDI) and dose-length product (DLP) values.

- MR Imaging Examinations:

The MR examination of the neck and of metastatic emboli was performed with a 1.5-T MR imaging unit (Symphony Magnetom, Siemens, Erlangen Germany) using a standard head coil combined with a two-channel dedicated surface neck coil. A transverse T2-weighted TSE sequence was performed with 3000/80 repetition time msec/echo time msec, 44 sections, a 4mm section thickness, a 0.4mm intersection gap, field of view of 20x25 cm, a matrix of 298x320, two acquired signals, an echo train length of 19. A transverse T1-weighted TSE sequence was performed with 800/8, 44 sections, a 4mm section thickness, 0.4mm intersection gap, a field of view of 20x25cm, a matrix of 250x512, three acquired signals, an echo train length of 9. Gadolinium based contrast material was injected (Gadoterate meglumin, Dotarem ®, Guerbet AG France) with a mechanical injector pump in an antecubital intravenous line, followed by 10-20ml of saline solution. The images were obtained without anaesthesia.

For each patient, the items examined were: Cervical neck compartments – Presence of thrombosis or compression of the IJV - Surrounding soft tissue, neck collections or abscesses - Septic emboli – Presence of lung infiltrates and nodules

Results

Results are summarized in tables 2 and 3

Clinical results :

All the patients were in good general health and it always initially started with an oropharyngeal infection. A sore throat, with high fever, neck pain or tenderness and adenopathy were generally present. Only one patient (case 7) presented with additional testicular pain.

In all patients, laboratory results confirmed an inflammatory syndrome with neutrophilia or leucocytosis and raised CRP.

Five of seven bacterial cultures were positive for *Fn*. One came out negative because the patient was under former antibiotherapy. In case 1, culture was not done because at the admission, no thrombus could be identified on US examination.

All received intra venous antibiotics, anticoagulation and NSAID. Two received corticosteroids. One patient needed a pleural drainage. Transfusion was necessary in two cases. In one case ventilation, bronchoscopic extraction of pus and a drainage were needed.

The clinical course was good in patients 1, 4 and 5. Two patients evolved to an ARDS, one other had a recurring pain and patient 2 died one week after admission. The duration of hospitalisation went from 3 to 52 days.

Imaging:

In one case (no 4) thoracic images were normal.

On CT images, the tonsils were often hypodense. Sometimes they were phlegmonous or even abscedated. Because of the inflammation, perivascular spaces disappeared and lymphadenitis could be seen in all images.

6 patients had a thrombus in the IJV. Its radiological characteristics were: a filling defect, hypodensity, thickened and contrast enhanced vein wall and enlargement of the vein. On US-Doppler findings, the vein wall was thickened and non-compressible. In addition, no blood flow could be identified. In case 4, the abscessed collection in the area of the sternocleidomastoid muscle caused IJV compression.

5 patients showed lung nodules with an associated feeding vessel. These lesions were at different cavitation stages: 2 were non-excavated, others were partially (2) or completely (1) excavated. 2 patients had normal thoracic images.

Parenchymal infiltrates, frosted glass appearance, pleural effusions and inferior lobe condensations were also seen in some patients

Some complications could also be observed. In case 1, a Grisel syndrome defined by torticollis with C1 and C2 rotation, has been identified. This condition is secondary to the neck inflammation. Patient 6 evolved into a pneumothorax. Osteomyelitis explained testicular pain in patient 7 due to septic emboli in the ilio-psoas muscle and the ischio-pubic ramus.

Box 1 and 2 summarize CT and US patterns of the LS :

Box 1 : CT patterns	
Tonsils	- Phlegmon or abscess - Adenopathy
Thrombus	- Distension of the IJV - Thickened vein wall - Filling defect - Collateral venous network - Surrounding soft tissue edema
Lungs	- Pleural effusion - Frosted glass
Septic emboli	- Wedge-shaped peripheral opacities - Feeding vessel - Variable cavitation stages

Box 2 : US patterns	
Thrombus	- Low intensity echoes - Non compressible, distended vein - Absence of blood flow
Soft tissue	- Edema - Lymph nodes

Discussion

For this case-series analysis we used the definition of the “classical” Lemierre syndrome¹¹. It begins with pharyngotonsillitis which is then followed by swelling and tenderness along the sternocleidomastoid muscle due to septic thrombophlebitis of the IJV. After a few days, postanginal septicaemia develops with high fever, rigors, adenopathy and metastatic lung abscesses within a week. Other primary foci in the head are possible and cause very similar symptoms, they are therefore considered as variants of the LS or Lemierre-like syndromes. The infection has to be documented by blood cultures positive for *Fn* and/or dissemination to other regions e.g. metastatic pulmonary infection.

It is mostly seen in previously healthy young patients. The first symptoms show approximately 5 days after the onset of the tonsillitis even if correctly treated, frequently at the time of the apparent clinical resolution of the sore throat. It then progresses rapidly in 7 to 15 days³. Septic metastatic emboli are mostly found in the lungs but can also develop in other body parts such as in bones, joints, the abdomen, the CNS, the cardiovascular system and soft tissues with associated complications^{1,18}. As a result, the differential diagnosis can be very large: viral pharyngitis, meningitis, infectious mononucleosis, leptospirosis, acute bacterial pneumonia, atypical or aspiration pneumonia, endocarditis, intra-abdominal sepsis, septic arthritis or neoplasia^{4,6,18}. For Lemierre, the clinical diagnosis was so obvious that it was impossible to miss. Nowadays, as a consequence of the discovery of antibiotics, this potentially lethal complication has become scarce and few physicians are aware of it. The great panel of possible localisations and the various associated symptoms creates diagnostic confusion^{2,4,7,18}. High clinical suspicion in various specialities (general medicine, otorhinolaryngology, orthopaedics, surgery and radiology) is necessary to diagnose LS as

soon as possible^{9,17}. Early diagnosis is important to prevent the evolution towards complications that need surgical treatment or that can lead to death.

The most frequently involved germ is *Fn*, a Gram negative anaerobic bacillus. It is considered as a commensal germ of the oro-pharynx by most of the authors. Its many synonyms (over 50) created great confusion, and may delay diagnosis^{2,18}. The invasion mechanisms are not yet understood but the weakening of the mucosae by infection or a concomitant disease and poor oral hygiene certainly are contributing elements^{6, 8,10,13,15,17}. A previous infection with the Epstein-Barr virus may be a predisposition as it can cause transient immunosuppression^{6,17}. As a second step, it invades the perivascular spaces, with possible symptoms depending on the affected area (e.g. cranial nerve IX-XII compression). It is followed by coagulation and haemagglutination in the ipsilateral IJV that form a favourable anaerobic environment. It causes thrombus formation and antibiotoxic resistance. Swelling along the sterno-cleido-mastoid muscle is the main clinical sign of thrombophlebitis but is not always present^{3,17}. Septic embolization in the whole body is due to haematologic or lymphatic dissemination, which occurs mostly in the lungs but can also be seen in other tissues such as joints, bones, kidneys and the central nervous system. For the treatment, at least two intra venous antibiotics with good anaerobic coverage are used : metronidazole and penicillin for 3 to 6 weeks. Clindamycin as monotherapy can be used in case of allergic reaction. The response to the treatment is quite slow due to sequestration, some areas are difficult to drain. Concerning the use of anticoagulation, there is some controversy⁹. The more time it takes to diagnose, the more severe the complications are. More invasive treatments, such as drainage or surgery will be needed in these cases.

According to Chirinos et al, in 81.7% of cases *Fn* is the only germ found. In 10.1%, the infection is in combination with other germs such as *Streptococcus sp* or *Staphylococcus sp*. Negative results (12.8%) do not exclude the diagnosis⁹. It could be a false negative, the sample may have been taken too early (haemocultures become positive only in the late phases). Haemocultures take 48 hours to 7 days to become positive¹⁸. In 69.7% of cases, culture was the diagnostical element instead of clinical elements or radiological patterns⁹.

With the development of new imaging techniques in the last ten years, radiology has become a good method to diagnose LS. It permits to show ORL and pulmonary lesions that may not be clinically evident : thrombus of the IJV and emboli are the important elements to find. Once documented they are sufficient to confirm the diagnosis^{5,15}.

On standard X-rays, poorly defined airspace shadowing or consolidations in the lower lungs zones can be seen but are quite unspecific.

On enhanced CT scan images, soft tissue oedema, venous distension and thicker vessel wall on the IJV path, define the thrombus and its localisation. Contrast products cannot pass through the occlusion which creates a typical filling defect sign. Spreading of the thrombus to its branches (e.g. : the facial vein or the peritonsillar veins) is possible. As a consequence of the obstruction, the formation of a collateral vein network can be seen. The tonsillar, peritonsillar, cervical and vascular spaces disappear due to edema, abscesses or phlegmon formed by the inflammatory process and lymph nodes are often present. This can cause the compression of the IJV and also induce septicaemia without thrombus formation. In the chest, multiple peripheral opacities, angiocentred and of variable cavitation stages have to be found as they represent the septic emboli. The cavitation stage evolves rapidly. The differential diagnosis of these cavitory nodules is : Wegener's granulomatosis, histiocytosis, septic emboli, tuberculosis, fungal pneumopathy or neoplasia. Nowadays, CT scan is the most accessible imaging method. The new machines reduce patient dose and give good quality images. Nearly 100% sensitivity can be obtained in finding the septic emboli and new reconstruction methods permits to see the whole IJV¹⁰.

US and US Doppler show low intensity echoes, a non-compressible distended vein and an absence of flux. As a consequence of inflammation, edema of the surrounding soft tissue can be seen. There are several limitations with this method. There are some inaccessible locations: the cranial basis, the mandibular angle and the clavicle. Gas bubbles can be present and a poor organised thrombus can be missed. It also is an operator dependent method. As a consequence, a negative CDUS doesn't exclude LS. On the other hand, it is a cheaper and more available method and is safer for the patient as it is non-ionising.

MRI is a very sensitive method for the soft tissue delineation and for the blood clot staging. It is an expensive method that can be used for more complicated cases.

Imaging is also useful for the follow up. It takes a long time before the thoracic images become normal again, and the thrombus of IJV often remains, with development of a collateral venous network to compensate the obstructed IJV.

This study has some limitations that have to be noticed : the small amount of patients doesn't allow us to generalize our conclusions. Some cases may not have been identified because of the retrospective perspective of the study. We also couldn't know the reasons behind the choice of the imaging methods.

Conclusion

Clinical and paraclinical elements (laboratory, microbiology and radiology) have to be considered as a whole so as to narrow the differential diagnosis of LS. In previously healthy young patients presenting with septicaemia and pulmonary symptoms with history of oropharyngeal infection, it is advised to carry out an enhanced CT scan. If IJV thrombosis and/or septic emboli are documented and matches the clinical picture of LS, an aggressive treatment should be started as soon as possible (even before the results of haemoculture) to avoid complications or fatal issue.

Tables and illustrations

Table 1 - 64-detector CT acquisition protocols			
Age (years)/weight (kg)	1-6/10-25	6-12/25-40	>12/>40
Neck			
Scout view AP+lateral 80kVp, 10mA	+	+	+
Gantry rotation time (s)	0.6	0.6	0.6
Pitch	1.375	1.375	1.375
Slice thickness (mm)	1.25	1.25	1.25
kVp/mA	100/120	100/150	100/160
CTDIw (mGy)	3.35	4.19	4.47
DLP (mGycm)	23.46	35.61	37.98
Matrix size	512x512	512x512	512x512
FOV (mm)	240	240	240
Thorax			
Scout view AP+lateral 80kVp, 10mA	+	+	+
Gantry rotation time (s)	0.5	0.5	0.5
Pitch	1.375	1.375	1.375
Slice thickness (mm) nom/rec	0.625/2.5	0.625/5 (2.5)	0.625/5
kVp/mA	100/160	120/120-180	120/180
CTDIw (mGy)	4.62-7.39	4.49-6.74	6.74
DLP (mGycm)	92.36-184.73	112.36-202.25	202.25
Matrix size	512x512	512x512	512x512
FOV (mm)	240	240	240

Table 2 - Clinical features

No / Sex / Age (y)	Clinical history	Status	Laboratory results	Cultures	Treatment	Evolution	Hospitalisation
1) F/3	Sore throat, vomiting, left torticollitis	Temperature 37.4°C, tachycardia, hypotension. Apathy and dehydration. Painful left angulo-mandibular and cervical adenopathy. Tonsillar erythema. Right head deviation and painful neck mobilisation. Neutrophilia, raised CRP.	Neutrophilia, raised CRP	Not done	iv antibiotics, NSAID, anticoagulation	Good	3 days
2) F/16	Sore throat, persistent fever, dyspnea, odynodysphagia	Temperature 39.1°C, tachycardia, pulmonary hypoventilation	Neutrophilia, raised CRP, coagulation disorders.	<i>Fn</i> and <i>S. viridans</i>	iv antibiotics, anticoagulation	<i>P. aeruginosa</i> overinfection, right superior lobe atelectasia. Cardio-respiratory arrest	8 days
3) F/19	Sore throat, fever, right abdominal pain	Temperature 39.3°C, tachycardia, hypotension. Tonsillar erythema. Right painful mandibular adenopathy. Painful palpation of the right hypochondrium and epigastrium.	Neutrophilia with left deviation, raised CRP, anemia, lympho- and thrombopenia. Hepatic tests alterations.	<i>Fn</i>	iv antibiotics, anticoagulation, pleural effusion drainage	Sepsis, ARDS then improvement	30 days
4) F/20	Phlegmonous cervical adenopathy	Hypertrophic tonsils. Cervical adenopathy. Right laterocervical pain, swelling and erythema.	Leucocytosis and raised CRP	<i>Fn</i>	iv antibiotics, NSAID, anticoagulation	Good	5 days
5) F/24	Pharyngitis, fever, odynophagia, neck pain	Temperature 40°C. Hypertrophic tonsils, bilateral adenopathy. Painful neck palpation and mobilisation	Neutrophilia with left deviation, raised CRP. Anemia. Thrombopenia.	Negative	iv antibiotics, antalgia anticoagulation, corticosteroids	Good	20 days
6) F/16	Sore throat, fever, strong headache	T° 39.5°. Tachycardia, hypotension. Diffuse lung crackles. Trismus, tonsillar hyperemia and hypertrophy. Painful cervical adenopathy, painful palpation. Hepatosplenomegaly.	Neutrophilia with left deviation, raised CRP. Thrombopenia. Coagulation disorders	<i>Fn</i>	iv antibiotics, NSAID, anticoagulation, corticosteroids. Assisted ventilation. Platelet and erythrocyte transfusion. Bronchoscopy with extraction of pus. Thoracic drainage.	Septic shock, ARDS, left pneumothorax then slow improvement.	30 days
7) H/19	Odynodysphagia, récurrent fever, respiratory dependent thoracic pain, abdominal and testicular pain.	Bilateral tonsillar hypertrophy and erythema. Tense abdominal wall with positive McBurney, Murphy and psoas' sing. Bilateral painful inguinal adenopathy.	Neutrophilia with left deviation, raised CRP, lymphopenia, leucopenia	<i>Fn</i>	iv antibiotics, antalgia, transfusions, anticoagulation	Cervical and inguinal pain. Recurrent fever. Then slow improvement.	52 days

Table 3 – Radiological findings

No	Standard radiography	CT scanner			US - Doppler	MR
		Cervical	Thoracic			
1)	Scoliotic attitude	Thickened left prevertebral, parapharyngeal and perivascular spaces. Adenopathy under the left sternocleidomastoid muscle. Enlarged left IJV with thrombosis (above the adenophlegmon, until the jugular gulf). Torticollis : C1 and C2 rotation (Grisel's syndrome).				
2)	Nodules	Sigmoid sinus thrombosis until the transverse sinus. Right IJV thrombosis from lower cervical part until facial veins and superior vena cava. Bilateral vertebral venous plexus thrombosis and enlarged epidural venous network. Pretracheal adenopathy.	Bilateral pleural effusions. Frosted glass. Angiocentrated septic emboli, some excavating. Inferior lobe condensations. Bilateral parenchymal infiltrations.			
3)	Infiltrations	Right tonsillar abscess. Right lingual and facial vein thrombosis, until the IJV.	Bilateral infiltrations. Septic emboli. Hilar infiltrations. Frosted glass.		Right retromandibular vein segmentar thrombosis. Touches IJV (but remains permeable). Thrombophlebitis	
4)	Normal	Right retro-sternocleidomastoid abscessed collection. Right jugulocarotid chain adenopathy. Compressed right IJV	Normal			
5)		Cervical adenopathy. Thrombosis under the skull base, 5mm diameter on the anterior face of the IJV, 12mm long.	Non excavated septic emboli.			
6)		Thrombosis on the whole length of the left IJV, left retromandibular vein and left external jugular vein thrombosis. Edema and disappearance of parapharyngeal fat. Cervical adenopathy.	Para-aortic, hilar adenopathy, also in the Barette space. Multiple nodules. Left pneumothorax, bilateral pleural effusion, frosted glass excavated septic emboli. Lobar atelectasis. Condensations in both inferior lobes.		Left IJV thrombosis from the subclavian vein until the angulo-mandibular region. No blood flow. thrombophlebitis, non-compressible. Adenopathy along the jugulocarotid chain.	
7)		Phlegmon in the tonsillar space. Thrombosis of the right IJV.	Peripheral septic emboli in the inferior lobe. Peribronchial thickening.		Right femoral vein thrombosis.	Right IJV thrombophlebitis with phlegmon. Inferior jugular chain adenopathy. Right tonsillar abscess.

Fig. 1 – Case 2



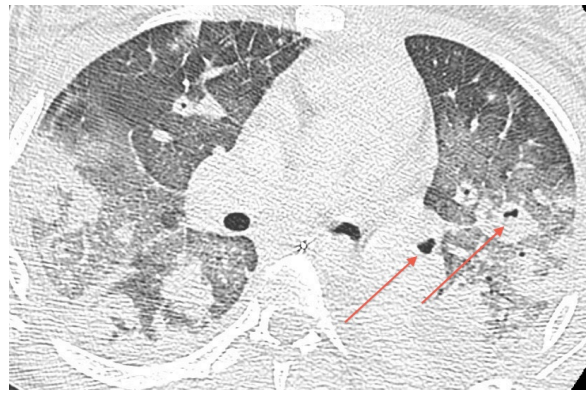
a



b



c



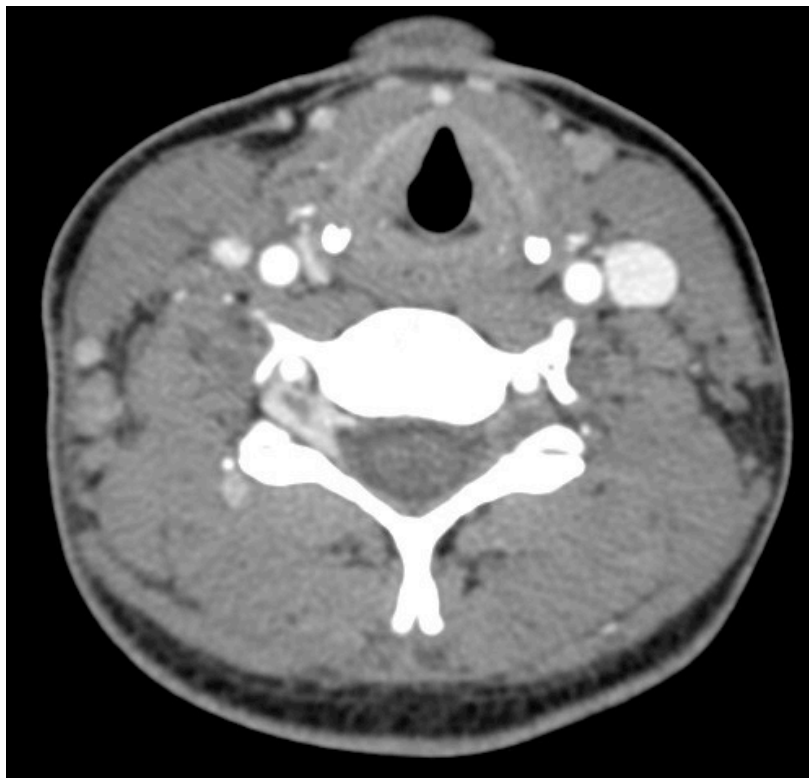
d

a. Contrast-enhanced transverse CT scan of the neck demonstrates a hypoattenuating non-occlusive intraluminal thrombus within the right internal jugular vein **b.** Same technique at the aortic arch level : the extension of the right jugular vein thrombus is seen in the Superior vena cava **c.** Contrast enhanced CT scan shows multiple peripheral lung nodules with cavitation in two large nodules (arrows) **d.** Axial high-resolution CT image shows multiple ground glass opacities with poorly defined, partially cavitating peripheral nodules

Fig. 2 – Case 4



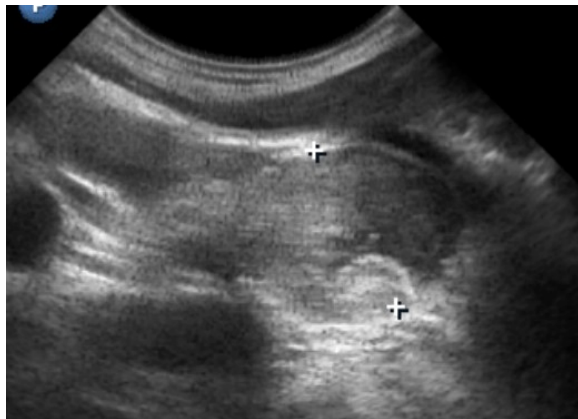
a.



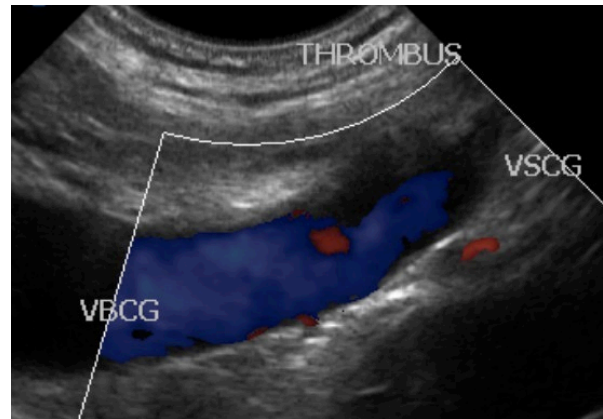
b.

a. Contrast enhanced CT scan at the level of the hyoid bone (a.) and cartilage (b.) shows asymmetry of the right IJV, collateral circulation and edema of the right neck soft tissues.

Fig. 3 – Case 6



a.



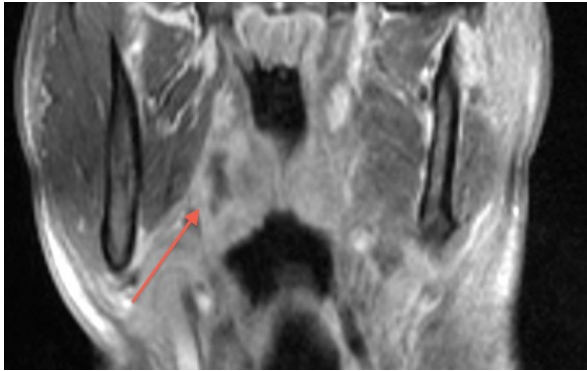
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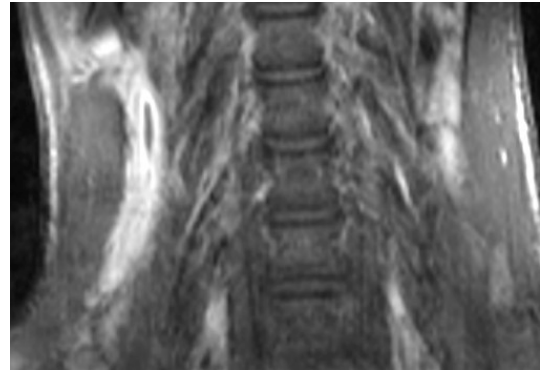
c.

a. Transverse US image of the left internal jugular vein depicts hyperechogenic thrombus in the vein (callipers) **b.** Longitudinal CDUS of the left internal jugular vein shows the tip of the thrombus **c.** Contrast enhanced CT scan shows left external jugular vein collateral vessels, occluded left internal jugular vein and edema of the surrounding soft tissues.

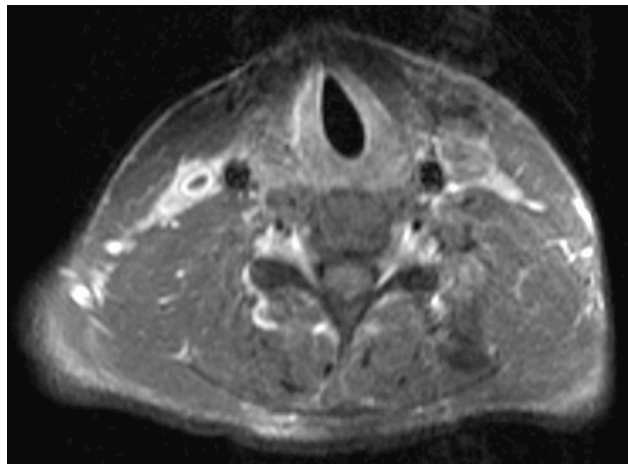
Fig. 4 – Case 7



a.



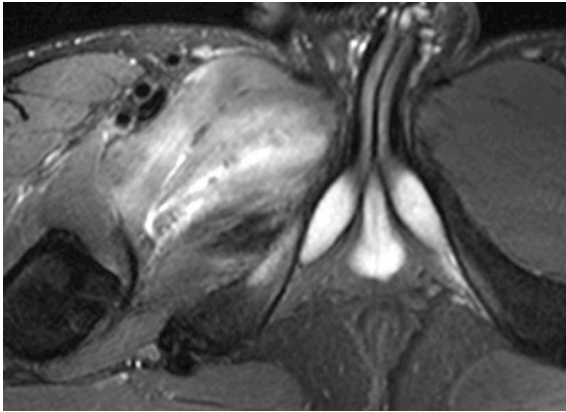
b.



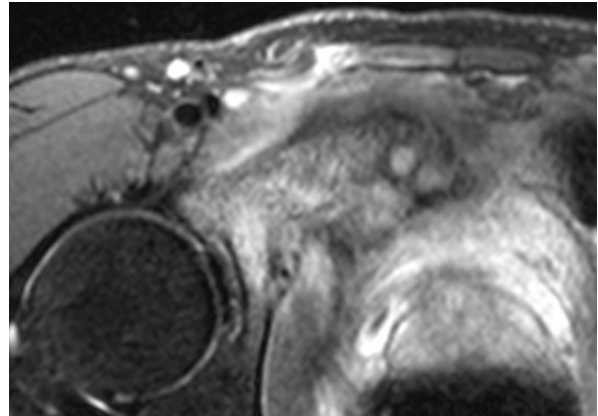
c.

a. Coronal T1 Weighted contrast-enhanced FatSat MR depicts right lymphadenitis and tonsillitis (arrowhead) **b.** Same technique at internal jugular vein level shows a thrombus with focus of low signal intensity surrounded by contrast enhancement **c.** The axial image shows a ring enhancing jugular vein wall.

Fig. 5 – Case 7



a.



b.

a. Axial T2 Weighted FatSat MR image showing an abscess of the right iliopsoas muscle and hypersignal of the right ilio-pubic ramus (b.) consistent with secondary osteomyelitis.

Fig. 6 – Case 1



a.



b.

Grisel syndrome : a. Contrast enhanced axial CT of the neck depicts IJVT, left lymphadenitis and right rotation of the head. 3D CT Surface shaded display shows secondary C1-C2 subluxation (b.)

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