BOOK 1

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NEUROLOGY
IN CLINICAL CASES

Dr. Kamen Genadiev Kamenov

NEUROLOGY IN CLINICAL CASES

BOOK ONE

This book combines in itself real clinical experience with practice-oriented theoretical knowledge in neurology. Its 70 documents present a total of 85 clinical cases that are illustrated with altogether 704 images and 9 classifications tables. Besides, there are also 10 short biographies that present 10 physicians who have contributed to the development of the neurology as a science worldwide.
We wish a pleasant and fruitful work to the readers of the book.

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Author's photography on the title page:
Vilyuchinsky volcano, Kamchatka, Russia

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008. In case of vertigo the cerebral arterial circulation should always be checked

This 68-years-old man has just been sent for in-hospital treatment by his GP because of recurring episodes of vertigo. His past medical history is notable of treated arterial hypertension and of active tobacco consumption of 30 pack-years that has been stopped approximately 15 years ago. In a very characteristic manner these episodes of vertigo appear mostly when he is standing and very rarely when he is lying. The worst for him is when he has to stand up: in this situation he always has vertigo and if he does not pay attention and stands up too quickly the vertigo is so strong that he could fall down if he does not hold himself to something. Because of this problem he learned first to remain seated for 1 to 2 minutes and to stand up only after this lapse of time. But lately even this stratagem does not help…

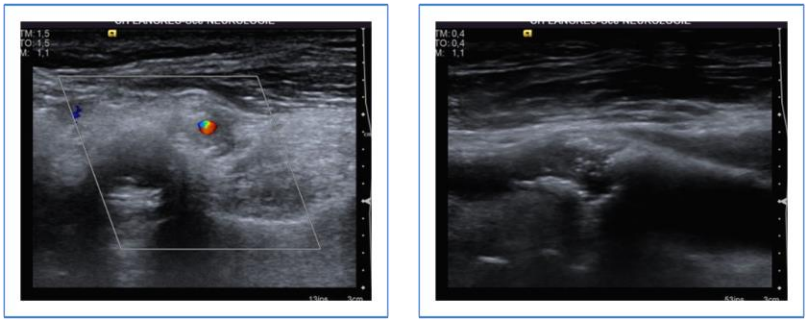
He has never had tinnitus and he has a good hearing with his both ears. In fact, he has never had any problems with his ears and his work is not noisy at all.

His vital parameters, his somatic examination and his neurologic examination are normal.

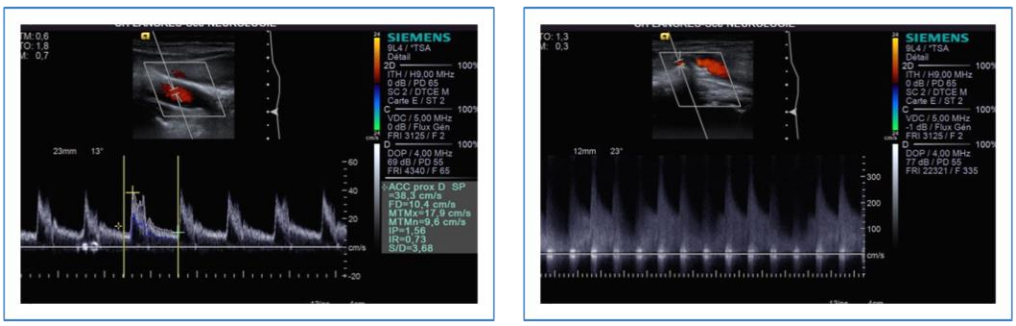
His blood analysis – full blood count, electrolytes, renal function, erythrocyte sedimentation rate, electrophoresis of the serum proteins – is without any particularities.

His brain scanner showed as the only particularity a minimal cortical atrophy.

The ultrasound examination of his cerebral arteries showed a clearly pathological result:



Already the transversal cuts in B-mode show an extremely important stenosing macroangiopathy that is maximal at the classical location, i.e. the carotid bulb. This becomes even more visible when the colour Doppler is added to the examination (1st image). The localisation at the level of the right carotid bulb is confirmed by the study in longitudinal plains in B-mode and the length of the stenosis is calculated to be between 1 cm and 1.5 cm (the total length of the image is 3 cm).



The ultrasound examination of the system of the right carotid arteries in longitudinal plains allows the localization of the stenosis and its quantification. The maximal systolic velocity on the level of the right CCA is 38 cm/s and in the stenosis it is 310 cm/s. The minimal diastolic velocity in the stenosis is 160 cm/s (note the important oscillation of the Doppler spectral signal that is due to the fact that this patient had a very short

neck with a cranially located carotid bifurcation and a very deep respiration, so it was difficult to fix the probe; manual calculation of the velocities).
These velocities in the stenosis correspond to a stenosis of at least 80 %.
This degree of the stenosis is confirmed by the rule of the index of the systolic velocities in the common carotid artery (proximal to the stenosis) and in the internal carotid artery (at the maximal stenosis), so by the rule of ACC/ACI index. This rule postulates that the percentage of the stenosis can be calculated according to the following mathematical formula:

Rule of the ACC/ACI index

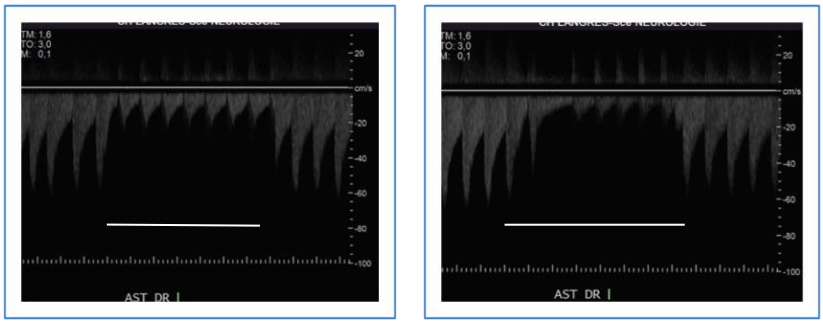
Percentage of the stenosis = (1 – $V_{maximal}$ systolic in the CCA / $V_{maximal}$ systolic in the ICA) x 100 %.

In our case the maximal systolic velocity in the right CCA is 38 cm/s and the maximal systolic velocity in the ICA in the stenosis is 310 cm/s, so the formula is

Percentage of the stenosis = (1 – 38/310) x 100 % = (1 – 0.11875) x 100 % = 0.88 x 100 % = 88 %.

In this manner a second ultrasound method gives the same result for the stenosis of the right carotid bulb. So, this is a haemodynamically significant stenosis of the right internal carotid artery.

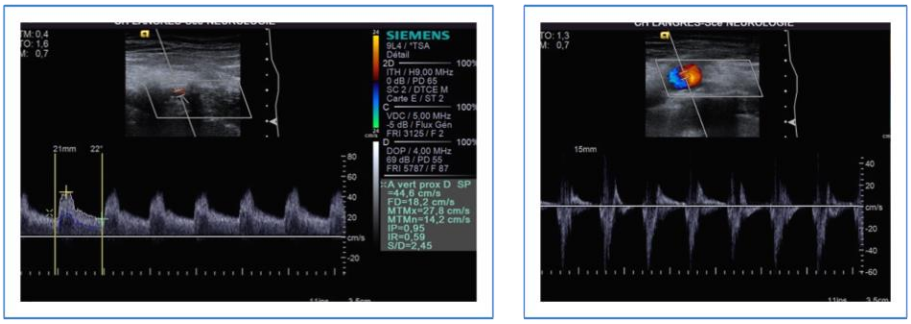
In order to be even more confident that this is a haemodynamically significant stenosis we studied the behaviour of the blood flow in the right supratrochlear artery in its natural state and also when there is a mechanic obstacle to its flow that is created by compression of its branches. Here is this result:



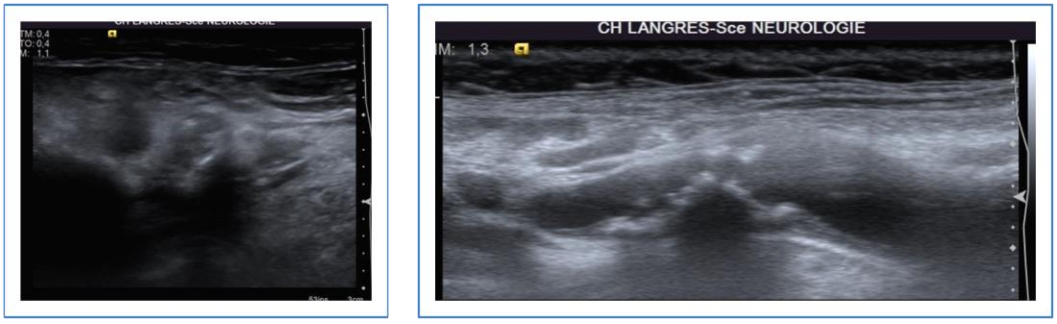
Registration of the flow in the right supratrochlear artery (AST) in its natural state and in case of compression of its branches. Note that in natural state the direction of the flow in the AST regarding the probe does not have any importance for our reasoning as the AST is a naturally tortuous artery.
The compression of the right facial artery and of right superficial temporal artery (indicated by the white bar) leads to a clear reduction of the flow in the right supratrochlear artery (1st image) with even an inversion of this flow (well visible on the 2nd image).
So, presence of one more ultrasound method that is in favour of a haemodynamically significant stenosis in the right ICA.

This patient has also had CT-angiography of the cerebral arteries that calculated the stenosis in the right carotid bulb to be 95 %.

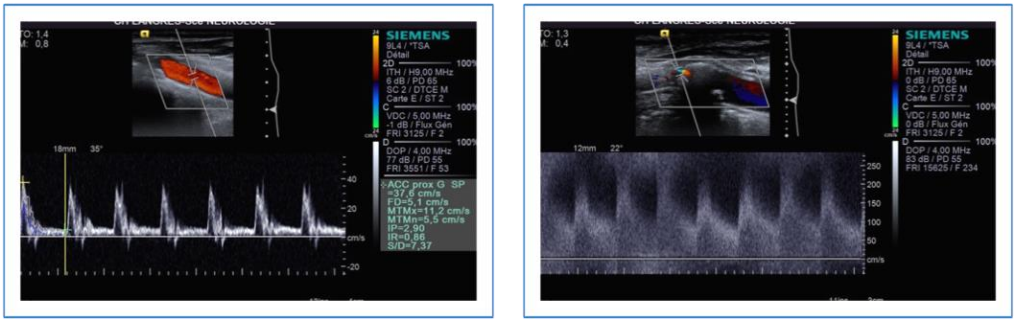
As far as the right extracranial vertebral artery and the right subclavian artery are concerned, their ultrasound examinations turned out to be normal:



Right extracranial vertebral artery (V_2) and right subclavian artery: normal results.
And what is the situation on the left? On the left the situation is practically the same:



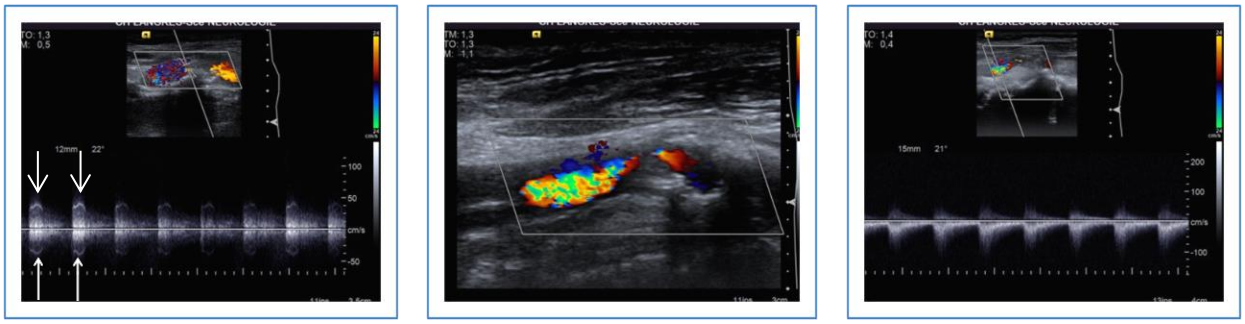
The B-mode shows the presence also on this side of an atheromatous narrowing on the carotid bulb. The longitudinal image has a total length of 25 mm, so the length of the plaque does not exceed one centimetre but this does not make it less dangerous at haemodynamic level as it can be seen by the rest of the examination:



The ultrasound examination of the system of the left carotid arteries in the longitudinal plains shows a maximal systolic velocity of 37.8 cm/s in the CCA, a maximal systolic velocity in the stenosis (i.e. in the initial part of the ICA) of 240 cm/s and a minimal diastolic velocity in the same location of 110 cm/s.
These velocities in the stenosis correspond to a stenosis between 70 % and 80 %.
The ACC/ACI index rule allows the calculation of the following degree of stenosis:

(1 – 38/240) x 100 % = (1 – 0,1583) x 100 % = 0,84 x 100 % = 84 %.

In addition, here it is possible to observe two more interesting phenomena that speak in favour of the significant degree of the stenosis, in particular we observe musical murmurs and also a superb poststenotic jet:

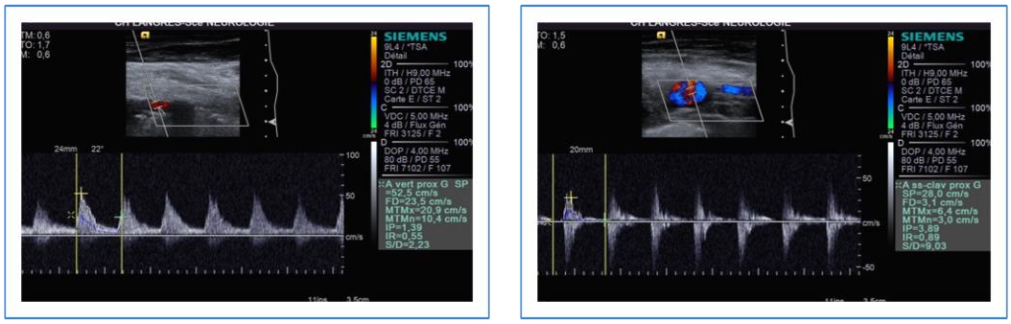


The 1st image shows the presence of musical murmurs in the form of mirror images compared to the baseline (arrows). The musical murmurs are due to turbulent arterial flow leading to vibrations in the adjacent structures to the artery in question. The presence of musical murmurs (called so because of the characteristic sounds that they produce) indicates the presence of a severe arterial pathology and usually this is an obliterating pathology.

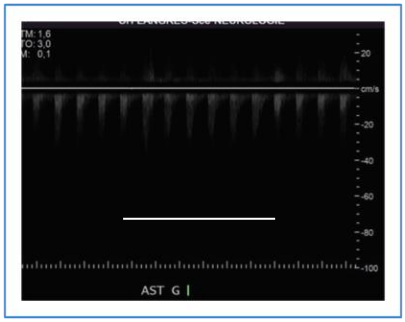
The 2nd image shows the poststenotic jet at the level of the left ICA that speaks in favour of a haemodynamically significant stenosis. The calculation of the maximal poststenotic systolic velocity in the jet refines the degree of the stenosis: if > 60 cm/s: stenosis up to 80 %, if it is < 60 cm/s the stenosis would be between 80 % and 90 %; this parameter is very low if the stenosis is > 90 %.

The 3rd image shows the maximal systolic velocity in the jet in our patient's case: it is 80 cm/s.

The ultrasound examination of the left extracranial artery and of the left subclavian artery shows normal results:



1st image: the left V_2 segment; 2nd image: the left subclavian artery in supraclavicular approach.



The flow in the left supratrochlear artery with simultaneous compression of the facial artery and of the superficial temporal artery on the same side (the white bar): absence of notable modifications of the signal, so on this case we cannot make any conclusions about the circulation in the left internal carotid artery.

The CT-angiography of the cerebral arteries showed the presence of a very high-degree stenosis of the left carotid bulb. It has been calculated at 96 %.

The discordance between, on the one hand, the degree of stenosis calculated on the basis of the maximal systolic and diastolic velocities in the stenosis and, on the other hand, the degree of stenosis calculated by the CT-angiography could be at least partially explained by the presence of calcifications in the stenosis what represents a difficulty for the exploration of the artery. There is also this particularity that in case of subocclusive stenoses of the extracranial internal carotid arteries the maximal systolic and diastolic velocities begin to decrease.

Finally it can be said that this patient presented subocclusive stenoses in the extracranial segments of his both internal carotid arteries and that was the only explanation for his vertigo (the ENT consultation showed no particularities and the search for an orthostatic hypotension gave a negative result).

This patient had a vascular surgery consultation and got the indication of a bilateral carotid endarterectomy. The stenosis of his left internal carotid artery has been operated first and two weeks later his right internal carotid artery stenosis has been operated, too. These surgical interventions have been carried out without any particularity and after they have been done this patient's vertigo disappeared.

Practical conclusions of this case

In case of vertigo of unknown aetiology (in a completely unknown patient or in a known patient in whom the vertigo is a new symptom) the exploration of the patient's cerebral arterial circulation is one of the compulsory paraclinical examinations.

025. A head trauma: two decades later (1)

The situation with this 38-years-old patient is serious: this evening, only 20 minutes earlier, he has been admitted in our hospital because of violent headaches that have appeared without any visible reason during the last night and that have little by little become stronger being now almost unbearable.

His clinical examination revealed the presence of a febrile confusion with minimal neck stiffness and no focal neurological deficits.

The blood analysis showed a hyperleukocytosis at 25600 / mm^3 (91 % polynuclears, 5 % lymphocytes and 4 % monocytes) and a CRP at 32 mg/l.

Hence, with no time to lose, a lumbar puncture has been done and it showed a cerebrospinal liquid with a slightly increased opening pressure and a turbid appearance, a xanthochromic supernatant, with countless leukocytes / mm^3 (85 % neutrophile polynuclears, 10 % lymphocytes and 5 % monocytes); 7400 red blood cells / mm^3, proteinorachia of 14.00 g/l and a glycorachia of 0.10 g/l for a glycaemia of 2.12 g/l. The microscopic examination showed the presence of rare Gram positive diplococci.

According to the results of the LCR study an antibiotic therapy has been started "at the tip of the needle with which the lumbar puncture has been done", that is, it has been started immediately.

The antibiotics that have been chosen were:

- Ceftriaxone (2 grams I.V. every 12 hours): this is one of the two C3G of first choice for the treatment of a Streptococcus pneumoniae meningitis and
- Vancomycin in continuous I.V. perfusion at a dose of 50 mg/kg b.w./d (it has been started in order to cover the patient in case the Streptococcus pneumoniae strain should be resistant to the Penicillin G (MIC, Minimal Inhibiting Concentration to the Penicillin G > 1 mg/l).

Two days later the results from the antibiotic susceptibility testing of the strain of Streptococcus pneumoniae isolated from the CSF and another such test of the same microorganism isolated from the blood cultures that have been done at patient's admission have shown that this strain is susceptible to the Penicillin (MIC to the Penicillin G at 0.012 mg/l). This result allowed to stop the Vancomycin and to continue the treatment by Ceftriaxone with the above mentioned dose for a total duration of 16 days.

Clinically, the confusional state has disappeared one day after the beginning of the antibiotic treatment, the headaches diminished without completely disappearing during the first week and their persistence was due to a diffuse cerebral oedema as it was shown by the two head CT that have been done. This cerebral oedema needed a temporary treatment with Mannitol and finally it disappeared with, as an epiphenomenon, the disappearance of the headaches.

The patient was quickly mobilized in order to prevent the eventual complications of a prolonged bedrest and he recovered completely.

The Pneumococcus is known as cause of meningitis in the human beings, especially if there is an osteomeningeal breach. So, in case of Pneumococcus meningitis we have to answer the question if there is one or more osteomeningeal breach(es).

As usual, everything has begun by the medical history once the patient could answer our questions starting from the second day of his in-hospital treatment. We could understand that:

- the headaches have been preceded by pains in his right ear with which he does not hear well;

- **18 years and one months earlier** our patient has had a car accident with a cerebellar haematoma on the right that had to be evacuated by a neurosurgical operation; and it is since that time that he has begun to not hear well with his right ear; so, this is a right posttraumatic hearing loss than was neither explored, nor assisted by a hearing aid;
- our patient has neither had other traumas, nor other operations in his life;
- he does neither take any medication regularly, nor does he smoke, nor has he any allergy.

The diagnostic workout has been continued by a head CT with thin sections in order to search for an osteomeningeal breach and / or other arguments in favour of an infectious focus that could have been the starting point of his meningitis.

The head CT showed the presence of a breach in the retro mastoid segment of the right occipital bone, loss of transparency of some posterior cells of the right mastoid process with another bone breach at that level and a (vascular) clip in the right half of the posterior fossa.

Once we could answer the question about the source of the infection we had to confront the next question, i.e. what could be done in order to minimize or even to eradicate the risk for a new meningitis.

First of all, we asked for an ENT consultation that has been done in our hospital but it could not make things progress.

At that moment we searched the neurosurgeons who, 18 years earlier, have operated this patient. Once they were informed about the situation they accepted to consult him.

In the meantime the treatment of this patient's acute meningitis has been successfully ended, he has returned to a normal health state and finally he could return home on the 11th of November, after 17 days of in-hospital treatment and – as noted above – after 16 days of correct antibiotic treatment.

……

Exactly 50 days later we were on duty during the New Year's Day.

In the morning of this day we have been summoned in the emergency ward where among the new patients was this same man who has been treated from his meningitis two months earlier. He has come just after midnight because of the reappearance since last night of headaches as 2 months earlier. In the night he has been examined and his somatic examination and his neurological examination had turned out to be normal, the blood tests have shown a hyperleukocytose at 13400 white blood cells / mm^3 and a normal CRP < 3 mg/l. As consequence, he had received one gram of Paracetamol by the mouth and he had fallen asleep. This morning he had been awoken by his headaches that have already been stronger (VASP at 8/10) and a low-grade fever had appeared. The new colleague in the emergency ward wants to make a lumbar puncture and asks for our opinion.

Having in mind the recent Pneumococcus meningitis, the presence of post traumatic osteomeningeal breaches (the patient has had his neurosurgical consultation and he was awaiting an ENT consultation and a new head CT with thin sections) and the patient's current situation, the decision for the lumbar puncture has been approved as the best diagnostic option in this setting.

So, the lumbar puncture has been done 15 minutes after our consultation.

Here are its results:

Studied parameter	Tube 1	Tube 2	Tube 3
Appearance before centrifugation	Trouble	Trouble	Trouble
Erythrocytes	7 / mm^3	3 / mm^3	15 / mm^3
Leukocytes	2960 / mm^3	2600 / mm^3	2700 / mm^3
Leukocyte differential count	Polynuclear neutrophils: 70 %, lymphocytes: 20 %, monocytes: 10 %		
Proteinorachia	1.30 g / l		
Glycorachia	0.40 g / l (for capillary glycaemia of 1.31 g / l)		
Bacteriological study	No visible microorganisms		
Bacterial culture **(known 5 days later)**	**Positive: presence of *Haemophilus influenzae***		
Mycological culture	Negative (result available in 48 hours)		
Search for Koch's bacillus	Negative (result available in 7 days)		

So, once again an acute purulent meningitis.

At the admission it seemed that most probably this acute purulent meningitis was once again caused by Streptococcus pneumoniae, even if the search for the urinary antigen of this bacterium turned out to be negative. Hence, we have immediately started the same antibiotic treatment as two months earlier – Ceftriaxone at the dose of 2 grams every 12 hours and Vancomycin in continuous perfusion at the dose of 3 grams every 24 hours after the application of an initial charge dose.

The patient was once again transferred to the reanimation ward and once again he had a quick improvement of his clinical state and biologic parameters.

On the 5th day of his new in-hospital admission and treatment we received the results from the bacteriological culture of his lumbar puncture with the following conclusion: **presence of *Haemophilus influenzae***.

In this way the final diagnosis of this patient's acute medical problem was an acute purulent meningitis due to Haemophilus influenzae.

The antibiotic treatment by Ceftriaxone at the dose of 2 grams every 12 hours I.V. has been continued for 21 days; the symptomatic treatment was progressively diminished and finally stopped. The patient was once again successfully cured from his acute infectious problem but his basic medical problem was still here: the presence of posttraumatic osteomeningeal breaches.

And in order to solve it he needed specialized surgical care.

So, our neurosurgical colleagues have been informed about this patient's new acute meningitis and they have been asked to take him for this specialized surgical treatment earlier than initially planned.

In the meantime the patient had once again a cerebral scanner with thin sections through the pyramid of the right temporal bone. In comparison to the same examination that has been done two months earlier (v.s.) this new examination showed three new elements:

- several hydroaeric levels in the right mastoid cells,
- appearance of an air-like density at the level of the right jugular foramen and
- asymmetry of the jugular foramens with fragmentation of the medial wall of the right one.

In summary: two more elements in favour of the diagnostic hypothesis that our patient's basic problem was the presence of a chronic infection of the right mastoid cells with the presence of posttraumatic osteomeningeal breaches that favoured the appearance of acute purulent meningitides.

As the patient has been cured from his second acute purulent meningitis he had returned home, this time with a sick leave and a preventive antibiotic treatment by amoxicillin/clavulanic acid at the dose of 1000 mg/125 mg t.i.d., by the mouth, to take till the neurosurgical intervention.

……

Three more months have passed and our patient has been finally cared for by our neurosurgeons.

The specialized neurosurgical care consisted of an operation that was carried out simultaneously by neurosurgeons and ENT surgeons. Its goal was first of all to identify the osteomeningeal breaches and then to close them by clogging using a biological glue. This operation was successful and after a short in-hospital stay after the surgery our patient could return home.

Shortly thereafter we have seen him at our neurological consultation and he was in perfect health, without any treatment and with a normal familial and professional life.

Both he and we have considered that all bad things were over for him and that his medical problem has been resolved forever.

Alas, we all were wrong. The continuation of this problem manifested itself 3 years later.

In order to know more

Streptococcus pneumoniae (Pneumococcus)

Streptococcus pneumoniae is a bacterium that is highly pathogenic for the human being. It belongs to the genus Streptococcus. It is Gram-positive microorganism that has a typical form: it resembles an irregular sphere with a pointed part through which it enters in contact with another pneumococcus and this whole looks like the number 8.

Streptococcus pneumoniae has a well-defined capsule. It contains the capsular antigens that allow the serological identification of more than 90 strains of this microorganism. It could be proved that it is the capsule with its specific antigen that determines the degree of virulence of Pneumococcus. The virulent pneumococci are encapsulated and their capsule are smooth (S), the loss of capsule leads to loss of the virulence and the colonies become rough (R).

The pneumococcus also possesses somatic antigens. These somatic antigens are common to the other streptococci and they are from two types:

- protein antigens and
- a polysaccharide antigen of type “C” (**Note**: the C reactive protein provokes the precipitation of this polysaccharide antigen of type “C”).

Pneumococcus does not secrete toxins and its pathogenicity is based on its multiplication.

It is a facultative anaerobic bacterium, it does not possess catalase, its colonies are transparent, it transforms the haemoglobin into biliverdin.

It colonizes as commensal the respiratory tract of 5 to 10 % of the people in good health.

Streptococcus pneumoniae causes the lobar pneumonia, bronchopneumonia, purulent pleurisy, sinusitis, otitis, conjunctivitis and meningitis.

It is sensible to most antibiotics with the exception of the aminoglycosides and it has a relative resistance to the tetracyclines. A growing problem for the treatment of the pneumococcal infections is the increasing resistance of certain pneumococcus subtypes to the penicillin and, recently, also to the macrolides.

There are several vaccines against Streptococcus pneumoniae; the most common are the conjugated vaccine conferring protection against 13 subtypes and the polysaccharide vaccine conferring protection against 23 subtypes of streptococcus pneumoniae.

This germ was isolated for the first time in 1881 and it was named Diplococcus pneumoniae in 1920; since 1974 it has been renamed Streptococcus pneumoniae. The name Pneumococcus has been given as for decades after its discovery it has been considered as pathogen mostly of pneumonias.

The C reactive protein (the CRP)

The C reactive protein is a pentameric protein (made of five subunits) that is being produced in the liver and the adipocytes in response to an acute inflammation, its role being the activation of the complement system. It has been discovered in 1930 and since 1977 it has been used in the clinical practice for the diagnosis and surveillance of the different inflammatory responses (N < 6 mg/l).

Actually we dispose of the ultra-sensible CRP (sensitivity level of 0.2 to 5.0 mg/l) that is useful as one of the prognostic factors for the risk for cardio-vascular diseases (CVD). Principle:

- low risk for CVD if us-CRP < 1.0 mg/l;
- medium risk for CVD if us-CRP between 1.0 and 3.0 mg/l;
- high risk for CVD if us-CRP > 3.0 mg/l (according to the AHA and to the USA-CDCP).

A level of us-CRP < 2 mg/l in people treated by statin diminishes the risk for myocardial infarction and for death by coronary accident.

Haemophilus influenzae

Haemophilus influenzae is a Gram-negative bacterium from the Pasteurellaceae family that has been discovered for the first time in 1892 by the German physician Richard Pfeiffer during a flu pandemic. As a consequence it has been considered as the causative agent of the flu till 1933 when it has been found that the causative agent of the flu is a virus.

Because of all this in the first decades after its discovery Haemophilus influenzae was commonly known under the names of Pfeiffer's bacillus and Bacillus influenzae.

Haemophilus influenzae has the form of a small rod or coccobacillus (i.e. an oval form that is intermediary between the round of the cocci and the rod of the bacilli). There is no specific arrangement of the bacteria among them. It is a facultative anaerobe, it produces catalase, oxydase and also beta-lactamases and it can modify the form of its penicillin-binding proteins.

The genome of Haemophilus influenzae has been completely deciphered in 1995.

In 1930 two main categories of Haemophilus influenzae have been discovered:

- *the encapsulated strains* which are in total 6: *a*, *b*, *c*, *d*, *e*, *f*; the possession of a capsule renders them very virulent because it protects them from the phagocytosis and from the lysis by the complement; the infections caused by the encapsulated forms are or can be severe – otitis media, epiglottitis, pneumonia, meningitis, arthritis, bacteraemia and sepsis;
- the strains without capsule, named also *nontypable forms (NTHi)* : they are less invasive, saprophytes of the ENT sphere where they can cause local infections – otitis media, sinusitis, pharyngitis, pneumonia, conjunctivitis.

It seems that in the nature Haemophilus influenzae causes diseases only in the human beings who are also its only natural reservoir.

It possesses a mechanism of adhesion to its host's cells that is called **T**rimeric **A**utotransporter **A**dhesins (TAA). The TAA are proteins localized on the external membrane of the Gram-negative bacteria and they help the bacteria to infect their target cells by adhesion to the surface of these cells. Every TAA is made of three identical proteins, hence "trimeric".

The diagnosis of an infection by Haemophilus influenzae is considered confirmed when it has been isolated from a sterile compartment of the human body (CSF, blood etc.).

The injectable C3G are the treatment of first choice of the infection by Haemophilus influenzae. The germ is sensible also to amoxicillin combined with an inhibitor of the penicillinase; in case of allergies to the beta-lactam antibiotics the pristinamycin or a fluoroquinolone are also a good choice.

The resistance to the beta-lactams by secretion of beta-lactamase is a major problem in the treatment of the Haemophilus influenzae infections. There are already strains resistant to the fluoroquinolones.

The chemoprophylaxis in case of meningitis with H. influenzae is recommended

- for the non-vaccinated contact persons, including the adults;
- in the communities with children below the age of 2 years and
- in the families with one child below the age of 4 years.

It has to be started in the seven days after the diagnosis is established in the index case and it is based on the administration of rifampicin by the mouth: 10 mg/kg b.i.d. for 4 days without exceeding 1200 mg per day. In the infants who are of the age below one year this dose has to be reduced by the half.

The prevention of the infections by H. influenzae is done by the vaccine against the encapsulated b form of this bacterium (*Hib*). It is efficient for the prevention of the Hib infections, but not for the prevention of the infections with the other encapsulated strains and with the strains without capsule.

Special note: H. influenzae is frequently associated with Streptococcus pneumoniae in case of respiratory tract infections. Their interaction is very complex and it is still not known if this is a synergetic interaction or an antagonistic one.

038. The cerebral metastases (1)

This 46-years-old woman who is in a good general state has just been admitted in our neurological ward for diagnostic care and appropriate treatment in the presence of weakness of her right hand. This deficit has appeared little by little three days earlier and it has installed itself within a time lapse of several hours without any obvious cause. Additionally she has felt some low-intensity vertigo and she says that since the last two or three weeks she has no appetite.

Her former medical problems are the following ones: a scoliosis since childhood, a migraine without aura, an active tobacco consumption of one pack of cigarettes per day for several years, a LAGB (laparoscopic adjustable gastric band) that has been put in place 16 years earlier and that has been taken away two years later (her weight before the LAGB was of 118 kg, actual weight of 59 kg).

The familial medical history reveals that a brother is deceased from a lung cancer, another brother is deceased from a cerebral cancer (still living 2 sisters and 1 brother, all three actually in good health).

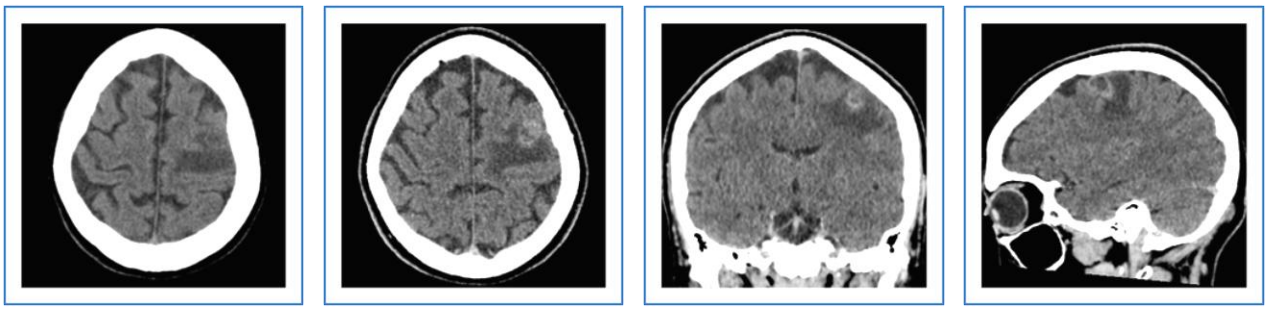
After the medical history we proceed to the somatic clinical examination that shows normal vital parameters and a mild oedema of her right hand.

Our patient's neurological examination shows:

- a right-sided cheiro-oral deficit (weakness of the right hand and right hands at 2/5, of the right forearm and of the right arm of 4/5 and a mild central right facial palsy) and
- a minimal hypo- and dysesthesia with the same distribution as the motor deficit.

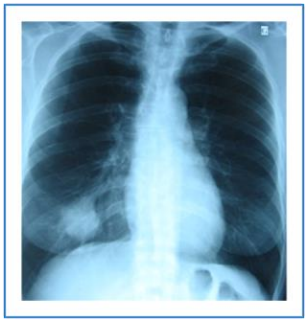
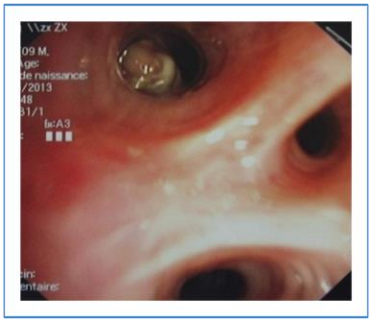
The standard blood tests proved to be without particularities.

At this moment it is quite logical to ask for a brain imaging study in order to look for the cause of our patient's neurological deficits. A CT is easily available what is not the case with the MRI, so a head CT has been done first without and then with contrast and its result is not reassuring:



Presence of a localized cerebral oedema without mass effect that surrounds an annular lesion of the right parietal lobe; the lesion takes the contrast in its periphery like a ring, so the first evoked hypothesis is the presence of a secondary lesion (the 1st image is a native one and the three other images have been obtained already after the injection of a contrast medium).

The head CT result is even more disturbing in view of the thorax X-ray that has been done practically in the same time as the head CT. A bronchoscopy has been done on the next day:

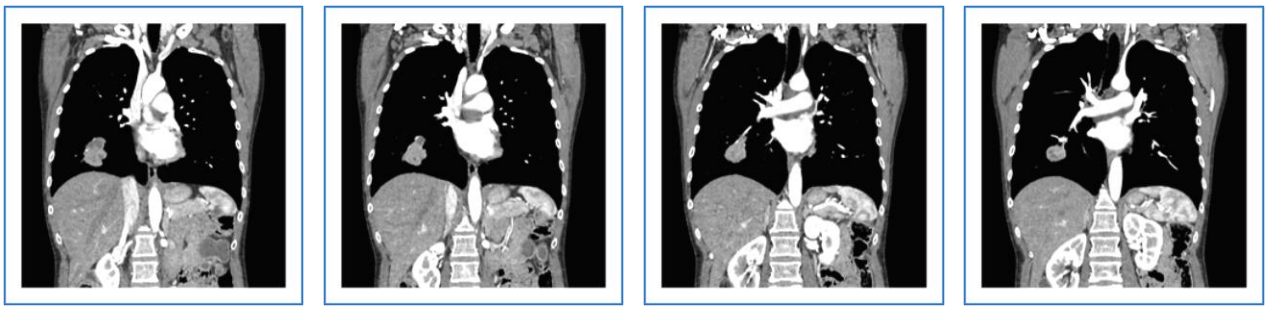
Chest X-ray: normal appearance of the heart. Hyperinspiratory chest with a right-sided antero-basal opacity translating a primary lung tumour.

Bronchoscopy: Presence of a mass in the bronchi of the right inferior lobe. A biopsy has been done.

At that moment the initial clinical diagnosis is the following one: right-sided sensory and motor deficit with cheiro-oral distribution in the presence of a single cerebral lesion at the junction between the cortex and the subcortical white matter in the left frontal region just in front of Ronaldo's fissure; this lesion appears to be a single cerebral metastasis from a primary lung cancer.

The diagnostic work-up has been continued as it follows:

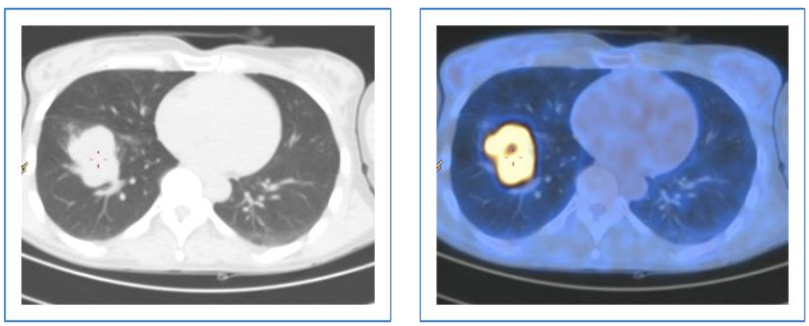
A CT of the thorax, of the abdomen and the pelvis with contrast that confirmed the presence of:



A large multilobulated lesion at the level of the inferior right lobe with several hypodense zones: **central necrosis**. This lesion is in contact with the oblique fissure anteriorly and with the diaphragm inferiorly. Presence of an anteriorly localized **component of a retro-obstructive condensation**. No other evident focal lesion has been observed. Heterogeneous appearance of the lung parenchyma: **smoker's lung**.

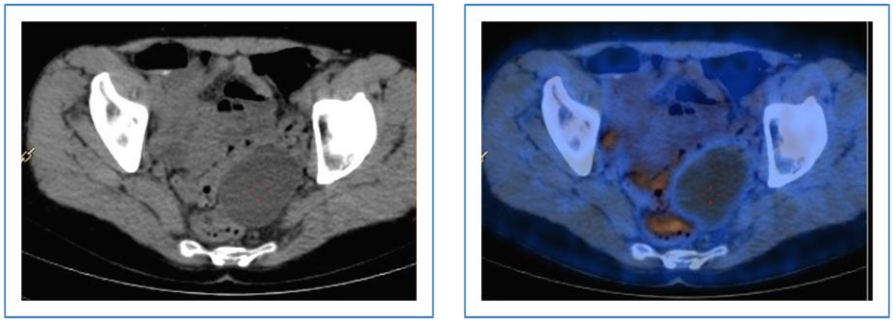
At abdominal and at pelvic level (the images are not represented here): **A large cystic image at the level of the left uterine appendages estimated at 66 x 54 x 59 mm.**

A whole-body PET scan using ^{18}F-FDG that showed the following result:

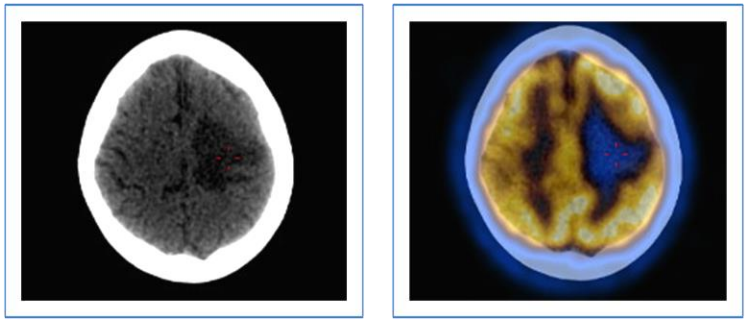


At cervical and thoracic levels: the voluminous multilobulated lesion in the parenchyma of the right inferior lobe (antero-basal segment) has a volume of 50 cm^3 and an intensive hypermetabolism (maximal SUV = 11.2),

with a central hypometabolism speaking in favour of a necrosis; there are no other fixation abnormalities neither on the level of the lung parenchyma nor on the level of the main lymph node areas.
*(**SUV**: see the note at the foot of the document).*



At abdominal and pelvic levels: no significant fixation abnormality is being noted, including for what concerns the liver parenchyma and the adrenal glands; the voluminous mass of the left uterine appendages is hypometabolic and its density approaches the density of a liquid.



At cerebral level: a large band of a relative hypometabolism in left frontal and parietal regions due to the presence of the oedema around the lesion.

At the level of the bones and of the bone marrow: no notable anomaly in the fixation.

Conclusion: The TEP scan using ^{18}F-FDG allows detecting an intense hypermetabolism concerning the known pulmonary lesion confirming its very suspicious character and the absence of arguments in favour of another regional or distal localisation with the exception of the known cerebral metastasis. Particularly, the left uterine appendages mass is hypometabolic.

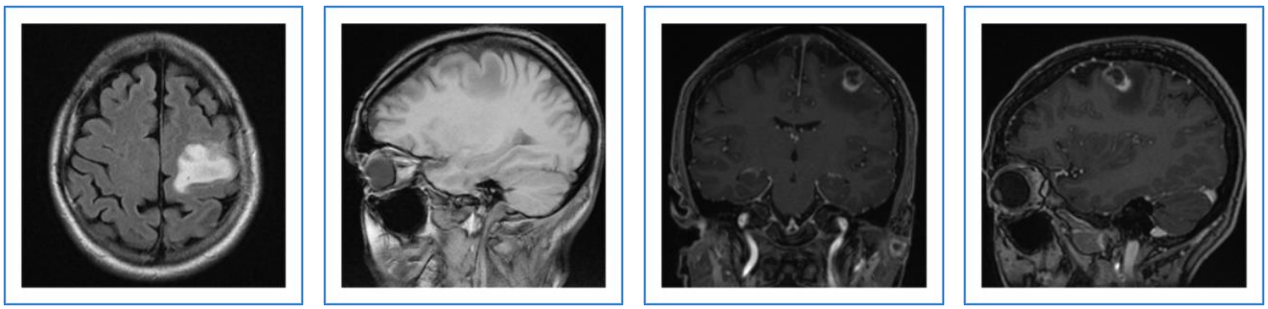
Specialized blood tests: Tumoral antigen NSE at 20.4 µg/l (N < 16.3 µg/l), beta-2 microglobulin at 1.48 mg/l (N < 1.31 mg/l); in the same time the other tumoral markers – Cyfra 21-1, ACE, AFP, Ca 125, Ca 15.3 and Ca 19.9 have normal levels.

Ultrasound examination of the cerebral arteries: This examination proved to be absolutely normal both in the extracranial and in the intracranial segments of the cerebral arteries.

Blood pressure Holter: The mean blood pressure over the 24 hours is 102/64 mmHg with respect of the nycthemeral rhythm, i.e. this is a normal examination.

ECG Holter: Nothing abnormal to note.

The brain MRI is also in favour of a:



single cerebral metastasis.

We have received the conclusion from the analysis of the biopsy from the bronchial tumoral mass in the right inferior lobe – the diagnosis is the one of a **bronchial adenocarcinoma**.

Our patient has been directed towards a specialized therapeutic care by neurosurgeons and oncologists in an interregional oncological centre. She has had neurosurgery, pulmonary surgery, chemotherapy and radiotherapy (on pulmonary level and on cerebral level).

One year later this woman was living at her home with permanent aid for the activities of the everyday life. Her mobility was reduces because of a right residual spastic hemiparesis and her Karnofsky Performance Score was 50 %.

***Note: SUV** = **S**tandardized **U**ptake **V**alue; it is a parameter that quantifies the ^{18}F-FDG fixation during a positron emission tomography.*

In order to know more

The cerebral metastases

The cerebral metastases are, unfortunately, a problem that becomes more and more frequent in the everyday neurological practice; hence it is necessary to know them well in order to manage them in an optimal way.

And not only the cerebral metastases are a frequent problem but they are also a clinical problem that has various clinical presentations and the cerebral metastases could

- be the inaugural presentation of the primary cancer,
- present themselves during the initial diagnostic and / or the initial therapeutic care for a known somatic cancer; they can manifest themselves during the follow-up of the primary cancer or even
- be discovered in an incidental way, for example when a brain imaging study is being done after a head trauma.

There are cancers that do not give cerebral metastases and there are cancers that give cerebral metastases. Among the cancers that give cerebral metastases there are five types of cancers that are most frequently on the basis of this problem:

- the lung cancers;
- the breast cancers;
- the cancers of the digestive system;
- the cancers of the genitourinary system;
- the melanomata.

These five types of cancers altogether give at least ¾ of all cerebral metastases.

The number of the cerebral metastases is another aspect of this medical problem. In principle it can be said that there are cancer types that give only one cerebral metastasis (a single metastasis) and that there are other cancer types that give multiple cerebral metastases although this rule is not absolute:

- single metastases: mostly in case of renal cancers, breast cancers and cancers of the digestive system;
- multiple metastases: readily by the melanomata and by the lung cancers.

The localisation of the metastases intracranially / intracerebrally could be different:

- intraparenchymal cerebral metastases: their typical localisation is at the junction between the cortex and the subcortical white substance;
- dural cerebral metastases;
- leptomeningeal cerebral metastases.

As far as the spatial distribution of the cerebral metastases is concerned it could also be said that most metastases in the brain are supratentorial and only approximately 20 % of them are localized caudally to the cerebellar tentorium.

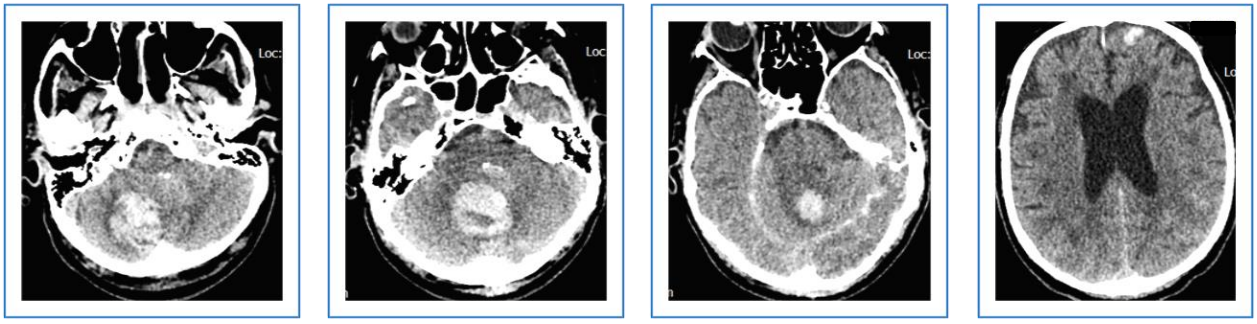
055. The spontaneous cerebellar haematoma: a real neurological and neurosurgical emergency

This 81-years-old patient has been completely independent in his everyday life up to this morning when he has been found lying on the ground in his house with an afebrile confusion not being able to stand up by himself as if he was drunk. In addition, he had a wound on his forehead.

The only known medical problem he has is a well-known and well-equilibrated arterial hypertension.

The initial examination confirmed the confusion and showed a paralysis of the horizontal gaze towards the right with the right eye, dysarthria and dysmetria for both right limbs.

The next logical step is the realization of a brain imaging examination and at first, a brain CT without contrast medium has been carried out. It showed the following result:

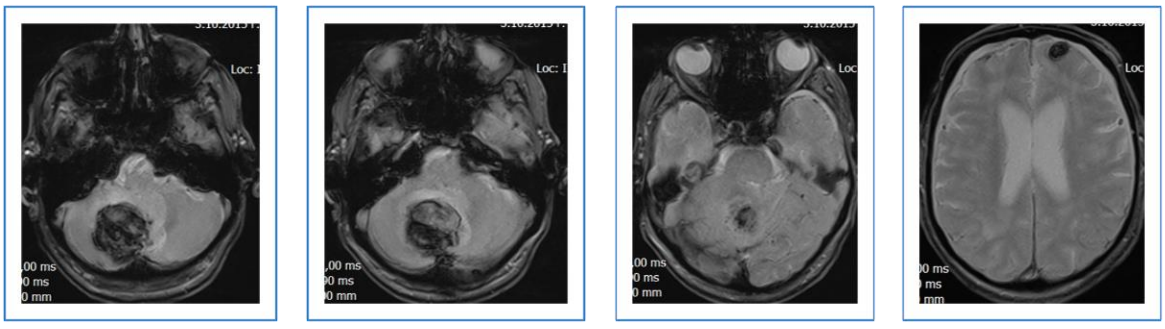


Presence of haematoma in the right cerebellar hemisphere with a subfalcine herniation that leads to a compression of the brainstem and of the fourth ventricle. In addition, contusion of the parenchyma of the right frontal lobe with a minimal posttraumatic subarachnoid haemorrhage.

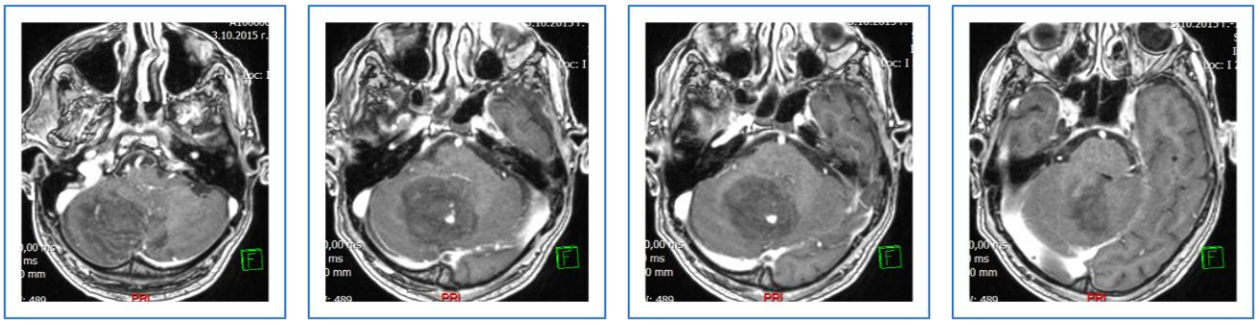
Presence of a cortical and subcortical atrophy, presence of a leukoencephalomalacia.

The application of the contrast medium did not bring further details.

The patient has immediately had a brain MRI, too:



The MRI T2* shows the locations where there have been bleedings in the cerebral tissue.

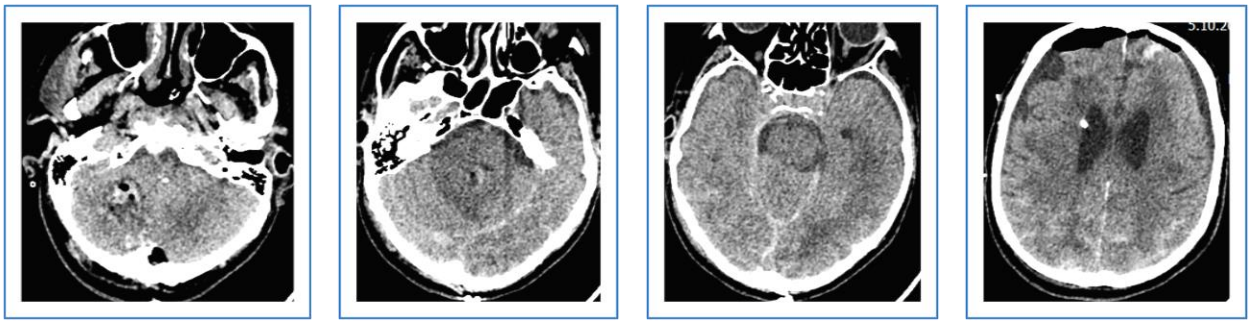


The sequence T1 with contrast medium injection shows well the compression of the above-mentioned cerebral structures and leads to the doubt that there exists an arterio-venous malformation (please note the contrast medium intake in the lesioned zone on the 2nd and on the 3rd images).

At this moment this man's diagnose is clear: a spontaneous intraparenchymal haematoma of the right cerebellar hemisphere with a minimal head trauma and a minimal contusion of the right frontal lobe. This contusion of the right frontal lobe parenchyma has occurred when right after the formation of the cerebellar haematoma this man has lost balance and has fallen. Suspicion of the presence of an arterio-venous malformation as the cause of this cerebellar haematoma.

The patient has been cared for in the neurosurgical reanimation ward and because the initial normal CGS score of 15 points it has been decided to await with the eventual neurosurgical operation. This patient's neurological status has been continuously surveyed and it showed a quick and inexorable degradation with a GCS as low as 8 points and the appearance of a respiratory insufficiency and of a tachycardia (suffering of the brainstem).

Therefore, it was decided to evacuate the cerebellar haematoma without any delay, so a neurosurgical operation has been carried out this same 2nd day of in-hospital treatment. This operation has been carried out without any problem and right after it the respiratory insufficiency and the tachycardia disappeared, what allowed this patient's extubation. His GCS improved to 14 points. At 24 hours after the neurosurgical operation a control head CT has been done:



This control CT without contrast medium showed the good evacuation of the cerebellar haematoma with disappearance of the mass effect and so with the disappearance of the brainstem compression. Please, note the presence of air bubbles in the zone where the haematoma has been situated and the presence of air into the skull (pneumoncephalus): these are normal phenomena after a neurosurgical intervention on the level of the skull.

During the operation (after the evacuation of the haematoma) an external CSF derivation has been created through the anterior horn of the right lateral ventricle. The surveillance of the intracranial pressure showed the absence of development of a hydrocephalus, what allowed the removal of this external CSF derivation two days later.

This patient's postsurgical period has been marked mostly by the presence of inhalation pneumopathies as the consequence of a major dysphagia following the transitory compression of his brainstem.

A Proteus mirabilis strain has been isolated from this patient's bronchial secretions and that allowed the realization of an appropriate antibiotic therapy that has been accompanied by symptomatic measures (aerosols, humidification of the air, respiratory rehabilitation). In addition, this man benefited from speech therapy and he could recover a normal deglutition in several weeks.

Once the patient's clinical state has been stabilized, he got an intensive complex rehabilitation.

A control brain MRI has been carried out 6 weeks after the neurosurgical intervention in order to search for the eventual arterio-venous malformation. This MRI search showed a negative result.

Finally, this man had a normal life without any residual neurological deficits for many years after this medical problem.

In order to know more

The spontaneous cerebellar haematoma (SCH)

The cerebellar haematomata represent about 10 % of all brain parenchyma haematomata.

They could be spontaneous ones and traumatic ones (they will not be discussed here).

As far as the spontaneous cerebellar haematomata are concerned, they are subdivided into:

- primary ones: they are due to the a long-lasting and not well equilibrated arterial hypertension (their anatomopathological basis are the Charcot – Bouchard's microaneurysm);
- secondary ones: the following can be mentioned:
  - arterio-venous malformations;
  - tumours;
  - amyloid angiopathy;
  - medications: anticoagulants; sympathicomimetics;
  - the presence of a spontaneous intracranial hypotension;
  - after supratentorial or spinal neurosurgery.

According to their localisation the spontaneous cerebellar haematomata are subdivided into:

- midline SCH or SCH that are quite near to the midline;
- SCH in one of the cerebellar hemispheres (lateral position).

The localisation of the SCH is important both because the clinical symptomatology is different and also because the best treatment option is frequently different.

As far as the clinical symptomatology is concerned in case of SHC its beginning is usually brutal or at least quick with the installation of headaches, sometimes of nausea, vertigo and disturbed balance. The level of consciousness could be normal or, since the very beginning, lowered.

Among the localizing signs it is possible to observe the ataxia of one or of both ipsilateral limbs, a (peripheral or central) facial palsy and a palsy of the ocular movements. In principle, these signs are ipsilateral to the haematoma side.

The differential diagnosis is being done with the ischemic strokes of the cerebellum and of the brainstem, with the spontaneous subarachnoid bleeding and with the acute subdural haematoma.

The final diagnosis is being established by brain imaging studies: CT and / or MRI.

The medical care for patients with spontaneous cerebellar haematoma should be done in specialized wards – neuro-vascular wards and neurosurgical wards.

The standard approach is the surgical treatment with the possibility for an initial non-surgical treatment for the lateralized SCH and for the SCH of small size (< 30 mm in diameter).

The appropriate initial therapy coupled with adequate postsurgical treatment and surveillance and also with a complex rehabilitation allows that most patients with SCH survive and remain independent, so that they have a normal life after the end of their treatment.

The Mount Fuji sign

The Mount Fuji sign is established by brain imaging studies (CT or MRI) and it can be observed in case of tension pneumocephalus (existence of a mechanism of unidirectional valve that allows the entering of air into the skull but that does not allow this air to exit the skull). This could be observed after neurosurgery, head trauma, head irradiation or spontaneously.

The frontal lobes are compressed by the air and they resemble a cone (the cone of the Mount Fuji).

The clinical picture is represented by headaches, lowering of the level of consciousness and signs of focal neurological deficits.

The treatment of this condition is by neurosurgery.

061. A patient with a Guillain – Barré syndrome who is getting worse after an initial improvement

This 53-years-old woman comes for an urgent neurological consultation on the 25th of September, sent by our colleagues from the nearby rehabilitation clinic.

The reason for the urgent neurological consultation is the fact that this patient:

- who has been diagnosed on the 02nd of August this same year in an university hospital with a Guillain – Barré syndrome from ascendant sensorimotor type with generalized flaccid tetraparesis with a generalized muscle weakness of 3/5 and with a diffuse pain syndrome,
- who has been treated from the 08th to the 12th August with polyvalent immunoglobulins i.v. in the dose of 0,4 g/kg body weight per day and who has done a good clinical recovery and who has resumed walking and
- who has been admitted for functional rehabilitation 12 days ago

presents a neurological degradation since the last 3 days.

So, there is a lapse of time of 51 days (i.e. 7 weeks and 2 days) between the beginning of the Guillain – Barré syndrome and the beginning of the relapse of the same problem.

The medical history of the actual deterioration shows:

1. The relapse has been preceded by pains in the right upper limb that have lasted only a few hours and the patient has felt the weakness appear rapidly both in her upper and lower limbs.
2. During the three days between the beginning of the relapse and the consultation the patient had an abnormal sensitivity over her right cheek, a metallic dysgeusia and a troubled vision. These deficits have gradually faded away and they are not present now.
3. There has been neither an acute bronchitis nor diarrhoea during (at least) the three weeks that preceded the relapse of the paralysis.
4. Just after the relapse of the disease there was some difficulty urinating but it is not present anymore.

She is embarrassed by her incapacity to walk but most of all by the quite unpleasant pains that make her suffer even although she takes two different antineuropathic painkillers.

The clinical neurological examination shows:

1. Absence of any clinical problem of the cranial nerves.
2. Dysmetria on both sides for the "finger-nose" test, D > G.
3. Perturbation of the deep sensitivity in both inferior limbs (with her eyes closed she cannot determine well which one of her toes is being pinched and in what direction it is being moved).
4. Absence of dys- or of hypaesthesia for the touch but during the ENMG the electric stimulations have been perceived as extremely unpleasant and even painful.
5. Partial hypotrophy of her both extensor digitorum brevis muscles, tibialis anterior muscles and, in a lesser degree, of the lumbrical muscles of her both hands.
6. The study of the periosteal reflexes and of the deep tendon reflexes shows the presence of the radial reflexes (+) and of the biceps brachii reflexes (+/–). The other reflexes of both upper limbs and all these reflexes in the lower limbs are not present at all.
7. Absence of pyramidal reflexes.
8. Generalized weakness that is estimated as it follows: both sternocleidomastoids are at 4+/5, the shoulders: 4/5 on the right and 4+/5 on the left, the flexion and the extension of the forearm on the arm: 4/5 on the right and 4 +/5 on the left, the same findings for both hands: 4/5 on the right and 4+/5 on the left; the thighs are at 4/5 on the right and at 4+/5 on the left, the flexion and the extension of the

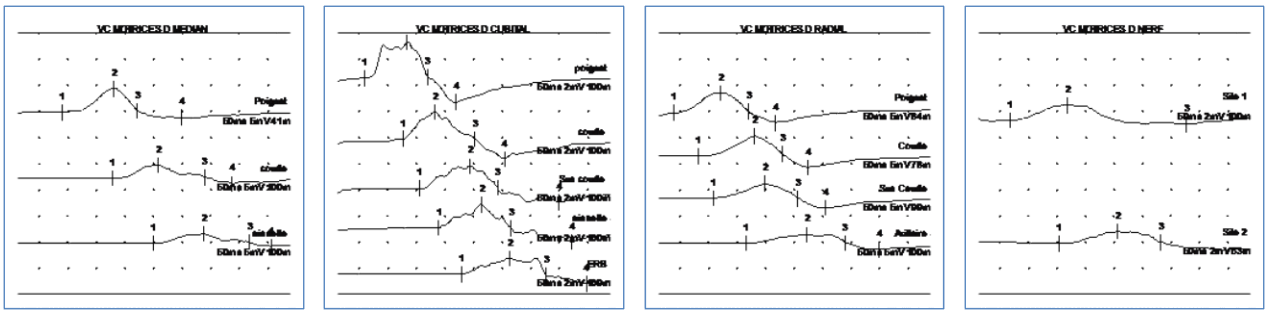
legs are at 4+/5 for the right and at 4/5 for the left, the dorsiflexion of the feet and of the great toes are at 0/5 on the right and at 3/5 on the left and the plantar flexion of the feet and of the toes is at 4/5 on the right and at 4+/5 on the left.

9. Standing up and hence walking are not possible.

The ENMG result is as it follows:

MOTOR CONDUCTION STUDY

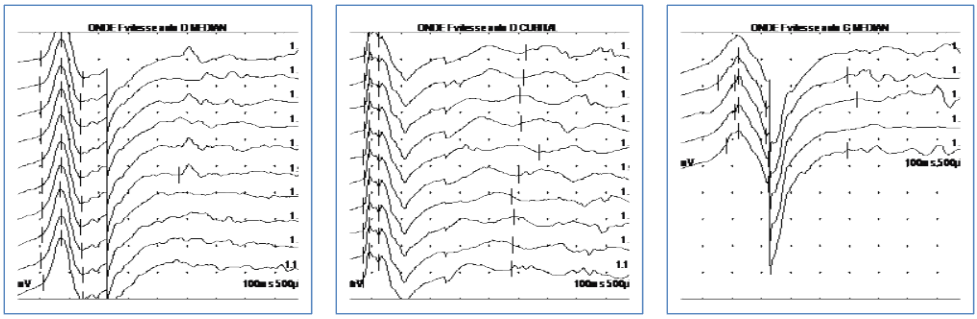
Nerve / Sites	Latency ms	Amplitude 1-2 mV	Surface mVms	Decrease S %	Distance cm	Velocity m/s
RIGHT MEDIAN NERVE ON THE ABDUCTOR POLLICIS BREVIS MUSCLE						
Wrist	8,95	**4,6**	25,4	100		
Elbow	17,85	**2,6**	17,1	**67,3**	25	**28,1**
Erb's point	25,00	**1,8**	12,9	**50,8**		
LEFT MEDIAN NERVE ON THE ABDUCTOR POLLICIS BREVIS MUSCLE						
Wrist	11,90	**3,1**	24,3	100		
Elbow	24,25	**2,4**	21,2	87,4	25	**20,2**
Erb's point	30,25	**1,9**	12,7	**52,5**	31	51,7
RIGHT ULNAR NERVE ON THE HYPOTHENAR MUSCLES						
Wrist	5,95	**2,8**	18,1	100		
Below the elbow	12,70	**2,1**	12,6	**69,8**	21,5	**31,9**
Above the elbow	15,50	**1,8**	13,2	**73,1**	6,4	**22,9**
Arm	19,00	**1,8**	10,6	**58,7**	13	**37,1**
Erb's point	22,85	**1,3**	12,8	**70,6**	18	**46,8**
RIGHT RADIAL NERVE ON THE EXTENSOR DIGITOROM MUSCLE						
Wrist	4,00	4,1	27,3	100		
Elbow	8,15	3,8	23,7	86,9	8,3	**20,0**
Arm	10,80	2,7	20,3	74,6	10	**37,7**
Erb's point	16,55	1,6	15,9	58,2	29	50,4
RIGHT TIBIAL NERVE ON THE FLEXOR HALLUCIS BREVIS MUSCLE						
Ankle	24,90	**0,2**	1,8	100		
Behind the knee	53,20	**0,1**	1,3	**71,6**	34	12,0
RIGHT EXTERNAL POPLITEAL NERVE ON THE TIBIALIS ANTERIOR MUSCLE (because no response from the extensor digitorum brevis muscle)						
Below the fibular head	6,85	**1,3**	14,9	100		
Above the fibular head	15,35	**1,0**	9,3	62	7	**8,2**
LEFT EXTERNAL POPLITEAL NERVE ON THE TIBIALIS ANTERIOR MUSCLE (because no response from the extensor digitorum brevis muscle)						
Below the fibular head	5,75	**1,7**	11,7	100		
Above the fibular head	8,45	**1,0**	6,6	**56,1**	6,5	**24,1**



From the left to the right: Motor conduction velocities on the right median, ulnar and radial nerves and on the left external popliteal nerve showing conduction blocks and temporal dispersion of the signal. Both these findings are the ENMG equivalent to demyelination on these motor nerves.

F WAVE automatic velocity

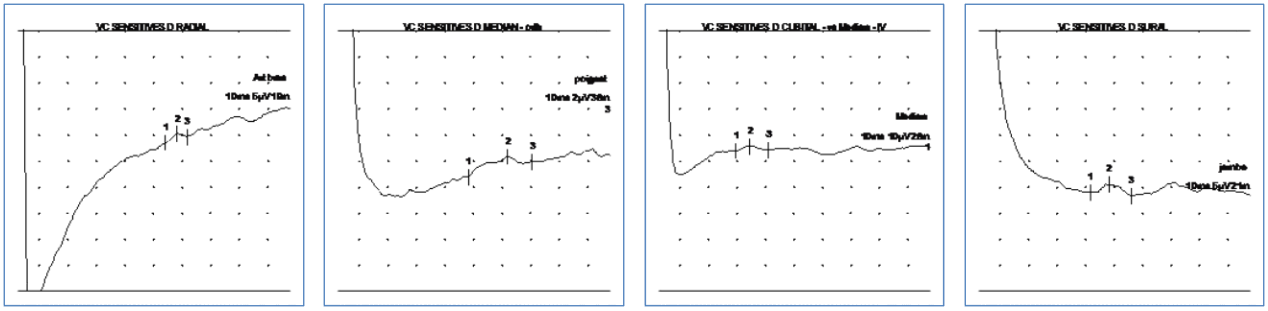
Nerve	Latency F min ms	Distance cm	Velocity F m/s	F-M min ms
RIGHT MEDIAN	57,15	77	**34,0**	46,35
RIGHT ULNAR	57,55	77	**31,2**	50,35
LEFT MEDIAN	59,40	77	**35,7**	41,40



From the left to the right: Study of the F waves on the right median and ulnar nerves and on the left median nerve. Everywhere it is possible to observe an increase in the time of appearance of the F wave and hence a slow velocity of proximal conduction below the inferior limit of the normal what is the equivalent to a proximal demyelination (on the sensory and / or the motor spinal nerves).

ENSORY CONDUCTION STUDY

Nerf / Sites	Site	Latency 1 ms	Amplitude µV	Distance cm	Velocity m/s
RIGHT MEDIAN NERVE – orthodromic Wrist	Palm	4,80	**1,6**	8	**16,7**
LEFT MEDIAN NERVE – orthodromic Wrist	Palm	3,20	**2,6**	8,3	**25,9**
RIGHT ULNAR NERVE Wrist	Palm	2,95	**1,9**	8,3	**28,1**
RIGHT SURAL NERVE Calf	External ankle	4,15	**2,2**	8,5	**20,5**
LEFT SURAL NERVE Calf	External ankle	4,70	**1,7**	10	**21,3**
RIGHT RADIAL NERVE Dorsum of the hand	Forearm	5,40	**1,7**	8,5	**15,7**
LEFT RADIAL NERVE Dorsum of the hand	Forearm	1,55	8,7	8,8	56,8
RIGHT SUPERFICIAL PERONEAL NERVE Lateral leg	Dorsum of the foot	5,15	**1,3**	13	**25,2**



From the left to the right: Sensory conduction velocities on the right radial, median, ulnar and sural nerves. Everywhere there is a significant slowing of these velocities what is the equivalent of demyelination along these nerves.

Conclusions:

1. Arguments in favour of demyelinating lesions of the sensory and motor nerves of all four limbs that concern both the peripheral nerves and their roots.
2. On the motor fibers these demyelinating lesions show also the presence of temporal dispersions of the signal as well as of conduction blocks.
3. **The ENMG results coupled with the clinical picture are in favour of a relapse of the Guillain – Barré syndrome**.

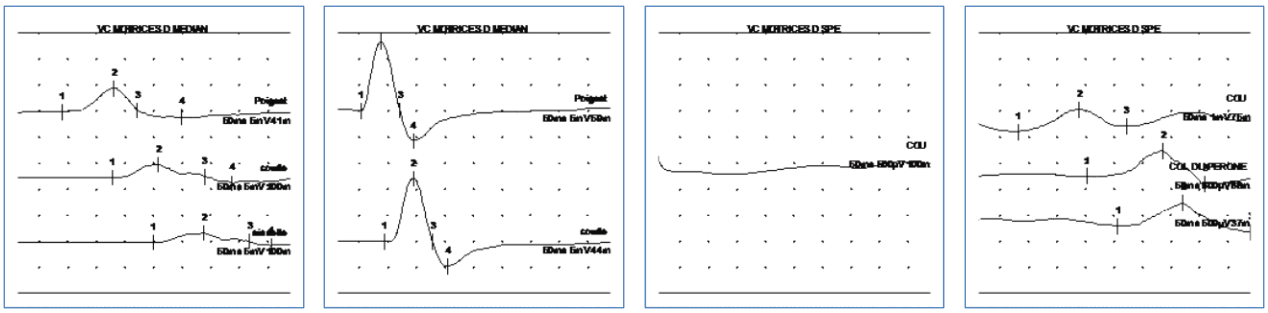
At this moment we have to answer what should be the treatment for this problem?

The treatment of the relapse of the Guillain – Barré syndrome is the same as for the initial episode: polyvalent immunoglobulins or plasma exchange.

In addition, standard preventive measures against the eventual complications of a prolonged bedrest and (continuation) of the functional rehabilitation should be undertaken.

Of course, this treatment has been immediately begun and its effect was spectacular: after the first two days of perfusion of the polyvalent immunoglobulins the subjective sensory troubles and the pains disappeared and after the 3rd day of this treatment the patient could stand up and walk again.

At 4 months after the beginning of the relapse of her Guillain – Barré syndrome this patient's clinical state was practically normal and the results from her control ENMG were significantly better:



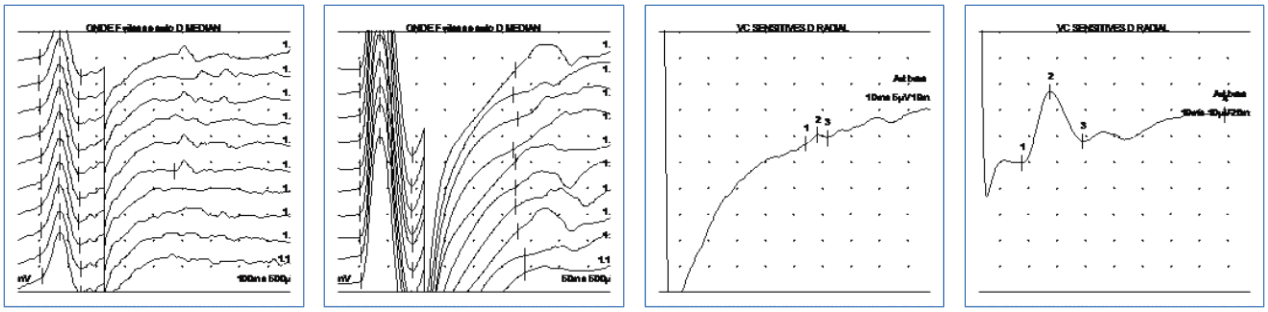
From left to the right, motor conduction velocities:

1st image: Responses of the couple right median nerve – right APB at the beginning of the relapse.

2nd image: the same couple nerve – muscle (without the axillary stimulation) 4 months later.

3th image: No responses on the right CPN – extensor digitorum brevis at the beginning of the relapse.

4th image: Reappearance of the motor responses in the same couple 4 months later.



From the left to the right (Note: The speed of scanning on the 2nd image is twice quicker as compared to the one on the 1st image):

1st image: F waves on the right median nerve at the beginning of the relapse: the velocity of proximal conduction is 34.0 m/s.

2nd image: F waves of the same nerve 4 months later: the velocity of proximal conduction now is already 59.8 m/s.

3rd image: Sensory responses of the right radial nerve at the beginning of the relapse.

4th image: Sensory responses of the same nerve 4 months later.

Finally, 10 months after the beginning of her Guillain – Barré syndrome and at almost 8 months after its relapse our patient had normal familial and professional life.

In order to know more

The ENMG study of the peripheral nerve in case of (suspicion of) Guillain – Barré syndrome should be done of at least 4 motor nerves with the study of the F waves and of 3 sensory nerves.

As the ENMG study during the very early stage of the Guillain – Barré syndrome could be normal, it is in fact perfectly legal to repeat this study a few days later if the diagnostic doubt is still present.

In order to establish a definite ENMG diagnosis of a Guillain – Barré syndrome we need the presence on at least two motor nerves of the two following criteria of demyelination:

1. Focal slowing, temporal dispersion and / or conduction block.
2. Absent F waves or F waves with a prolonged minimum latency (slowed maximal velocity) and an absent H reflex.

The Guillain – Barré syndrome can both relapse and reappear.

The relapse in case of a Guillain – Barré syndrome is defined as the initial improvement of a Guillain – Barré syndrome followed by the appearance of a neurological deterioration during the 8 weeks that follow the beginning of the disease.
Approximately 5 % of the cases of Guillain – Barré syndrome that are being treated by polyvalent immunoglobulins or by plasma exchange undergo a relapse.

The reappearance of a Guillain – Barré syndrome is defined as the appearance of two or more Guillain – Barré syndromes separated by a minimal interval of:

- more than 2 months if the previous episode has not left any residual deficits or
- more than 4 months if the previous episode has left any residual deficits.

In the medical literature there are descriptions of reappearance of a Guillain – Barré syndrome up to 43 years after the previous episode.

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