Subfascial venous edema by MRI

Objectification of the central correlate of phlebological pathology?

Summary

For draining the leg of blood exists a venous system. The subfascial veins are the most significant. An insufficiency of the system and the following venous hypertension will be concerned where the mainfunction of the system is situated, in the subfaszial region. By this deficiency of drainage results an extravasal volume overloading (edema) and an increase of the tension of the fascia, what explains the symptoms and the clinical signs. By MRI this edema could be demonstrated and also a therapeutical effect by application of nonelastic compression bandages as designed by Heinrich Fischer.

Zusammenfassung

Das venöse Blut der Beine wird über ein Venensystem zurück zum Herzen transportiert, wobei die subfaszialen Venen mit einem Anteil von ca. 80% die Hauptlast tragen. Eine Insuffizienz im System wird über die venöse Hypertonie somit vorwiegend den subfaszialen Raum betreffen. Dieses Drainagedefizit erklärt über die dabei vorliegende extravasale Volumenüberlastung und die daraus folgende vermehrte Faszienspannung die Beschwerden und die klinischen Befunde als Folge der Ödembildung. Das Ödem konnte im MRT dargestellt und seine therapeutische Beeinflussbarkeit mittels unelastischer Kompressionsverbände nach H. Fischer objektiviert werden.

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Leg vein blood is fed back to the heart via a venous system. The deep, subfascial veins are the main component of this system since they provide 80% of this transport service (1, 2). Blood flow in a central direction is ensured exclusively through the competence of the venous valves and the efficient working of the venous pumps. The functional capacity of valves and of pumps depends on the functional capacity of the fascia (3–8). Blood that reaches the leg via the arteries is transported out of the leg again only if these three structures are working properly.

If there is valvular incompetence or the pumps are not functioning properly, the resulting disturbance in transportation of blood back to the heart leads to venous volume overload, followed by venous hypertension with a further reduction in reabsorption capability in the capillary region (9–11).

In colloquial language, and therefore of immediate general understanding, the reabsorption deficit means inadequate "drainage", i.e. the tissues become "boggy". Harvey spoke of "Suffocation by an excess" (12). It corresponds clinically to the state of venous congestion and in pathophysiological terms it is called "oedema". That means an extravasal volume overloading as a consequence of the impaired reabsorption capability. Due to the functional importance of the subfascial veins, this will be predominantly relevant at a subfascial level, although it affects all organs of the leg. Because of the spatial restriction of the subfascial space caused by the inelastic structure "fascia", this condition of increased fascial tension correlates well with the typical complaints of congestion such as a feeling of heaviness and tightness in the legs or calf cramps.

If the oedema also extends in an epifascial manner, it may be clinically apparent as a "thick leg" (13). Patients may experience a tendency for the legs to swell and, on inspection, the contours of the malleolar region (Bisgaard's link) and proximal calf insertion are obliterated. A conspicuous increase in leg circumference can also occur.

The theoretical continuation of this physiological and pathophysiological cascade, which leads to the logical explanation of this subfascial oedema, would enable this condition to be recognised as the starting point of further complicating venous diseases: on the one hand, the tendency for thrombosis through the accompanying venous dilatation according to Poiseuille's law, and on the other, inflammatory phenomena such as stasis dermatitis or even necrosis, i.e. an ulcer, through stasis-related nutritional deficiencies. The existing persistent venous hypertension would also explain the development of epifascial venous incompetence.

Although the significance of these ideas cannot be discussed in detail here, they show the relevance of subfascial oedema (as a palpable clinical correlate) and were the reason why the aim of the present in-
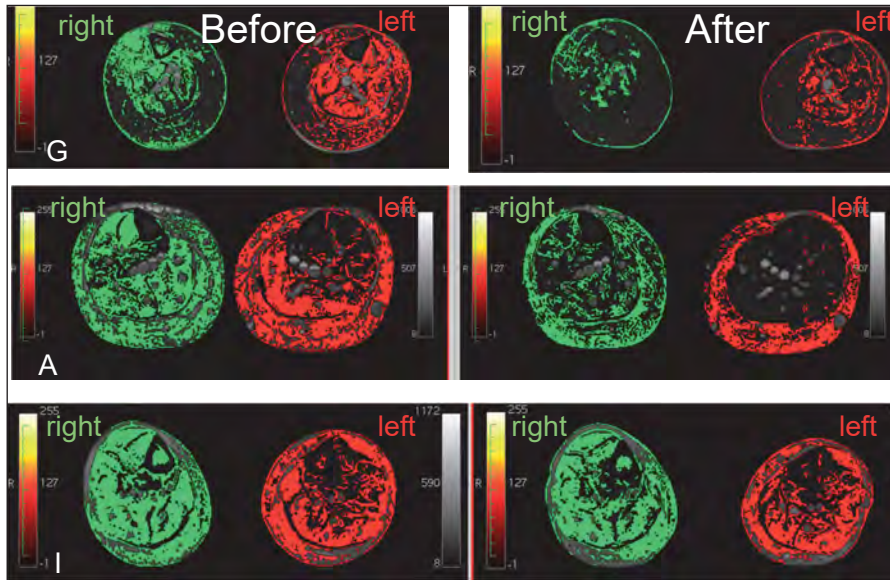
Fig. 1 T2-weighted MRI image of the lower legs of patients A, G and I before and after treatment of both legs. Water content on the right coloured green, left red. The water content is reduced after treatment.

vestigation was to prove the existence of the oedema by demonstrating it.

It was therefore my idea that it should be possible to demonstrate this oedema using MRI, because this technique is based on the resonance of water protons. With magnetic resonance imaging, atomic nuclei – generally hydrogen nuclei in the body – are excited to resonate. The varying content of hydrogen atoms in tissues provides the contrast in the sectional images. These are composed of three-dimensional imaging points, called voxels. There are different degrees of greyness for the various structures that can be coloured for better visualisation (▶ Fig. 1, ▶ Fig. 4).

In an MRI study, Duewell et al. distinguished between "swollen legs", lipoedema, lymphoedema and phleboedema, all of which show marked characteristics in MRI (14). Venous oedema – here, however, post-thrombotic – can be clearly differentiated from the other causes of thick legs, predominantly endofascial and also epifascial.

Haarverstad et al. showed very similar MRI images in a study of leg oedema (15). Although again post-thrombotic, they also found accumulations of oedema particularly in the subfascial compartments.

The visualisation of phleboedema in these two studies is identical to the oedema seen in my investigation and confirms that

this oedema can be demonstrated. The MRI images of lymphoedema or so-called lipoedema in the two studies are completely different in terms of the position subfascial or epifascial, structure and distribution in the different MRI-weightings.

Patients in whom, in addition to their symptoms (a feeling of heaviness and tightness in the legs, restless legs, calf cramps), the palpation findings (heavyness and tissue tension) of subfascial oedema are present, received soft, afferent-stimulating shoe inserts for active correction of poor foot statics. They were also recommended to wear feet-friendly shoes to improve foot and body posture and so optimise the fascial structures in the sense of the "Chinese finger" described by Askar (8) and also to take brisk walks several times a day to activate the muscle pumps. At the same time they were treated with inelastic lower leg compression bandages as described by Heinrich Fischer (16).

Aim

The aim of this study was to
● Demonstrate this substantial correlate of this pathophysiological view,
● Demonstrate the substrate of the palpation findings of the calf and also to

● Demonstrate the effect of treatment with inelastic compression bandaging on the calf as described by Heinrich Fischer.

The hypothesis was that by showing different quantities of water in the calf in the comparative measurements before and after treatment, it would be possible to document the fact of oedema and that it is treatable.

Material and methods

Five patients (two men, three women, average age 70.6 years) diagnosed with "manifest subfascial venous oedema" underwent MRI scans before and after treatment with inelastic compression bandages on the calf. The diagnosis was made according to history, inspection and palpation.

T2-weighted MRIs with fat saturation were carried out. T2-weighting means high signal intensity of all water protons. All other tissues show low signal intensity in T2-weighted images. Only fat would show a high signal intensity, which is why fat saturation was chosen. If only water spins show high signal intensity, it should be possible to measure the water content of tissue – the oedema – by counting all voxels above a threshold value.

The signals in MRI images are not standardised as in CT. The MRI signal depends on the person in the scanner, the receiving coil, the position of the coil and of the patient relative to the magnet and so forth. Threshold values were set by measuring corresponding signals of the soleus muscle. The mean signal intensity and the standard deviation in a region of interest (ROI) in the soleus muscle were measured and we defined the lower threshold as the mean value from the ROI minus one SD and the upper threshold as unlimited. The voxels within the thresholds were counted and we declared the sum of all voxels as the water content of the lower leg.

(The radiological study was conducted by Prof. Dr. R. Janka of the Institute of Radiology, Erlangen University.)

The Fischer bandage (16–19) is a dressing with inelastic gauze bandages that is shaped precisely to the lower leg with dif-

fering pressure distribution. It is fixed to the leg using a paste. Inelasticity is the opposite of elasticity and is characterised by a lack of stretch and hence also no restoring force. In combination with brisk walking, very high working pressures around 100 mmHg are generated (20, 21). This treatment was carried out until palpable removal of congestion stasis.

Results

The MRI scans before and after treatment with inelastic lower leg compression bandages showed a reduction in water content. This corresponded to a decrease in oedema (► Fig. 1).

In numerical terms, there was a volume reduction from the nuclear spin values (► Table 1 and ► Fig. 2) that corresponded to the leg circumferences measured before and after treatment (► Fig. 3).

The relevance was also clear in one patient who only allowed his right leg to be treated: the coloured visualisation of water content and the values measured on the left untreated leg remained the same, whereas the volumes (voxels) and externally measured circumference of the right leg decreased in parallel to the coloured visualisation of the fluid content (► Fig. 4).

Conclusions

The anatomical and physiological structure of the venous system of the legs means that the deep veins in the subfascial space have a very high functional significance.

However, because a system of veins is responsible for the transportation of venous blood back to the heart, all structures
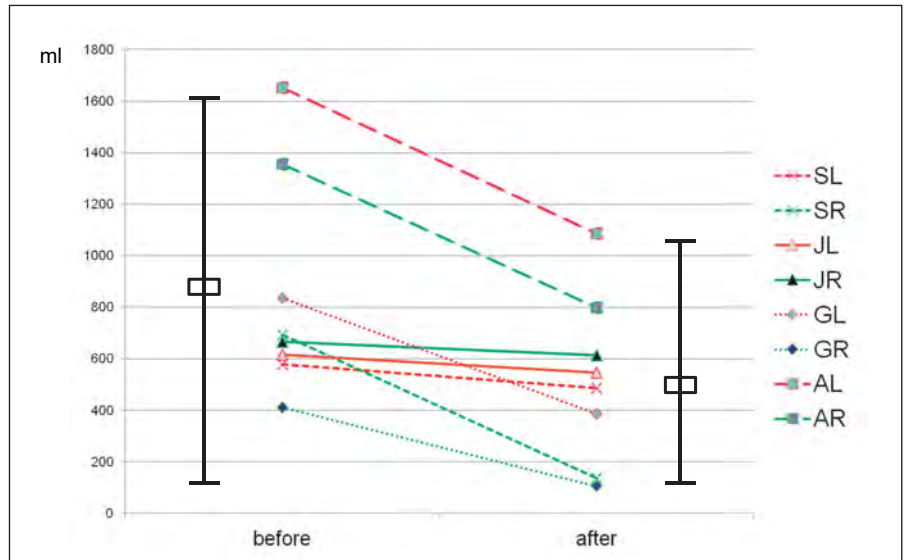

Fig. 2 Amount of fluid in ml in the lower leg measured from the sum of voxels in patients A, G, I and S before and after treatment. (Patient S only right leg treated); AL = Patient A left leg; AR = Patient A right leg; GL = Patient G left leg; GR = Patient G right leg; IL = Patient I left leg; IR = Patient I right leg; SL = Patient S left leg; SR = Patient S right leg

Fig. 3 Largest circumference of the lower leg of patients A, G, I and S before and after treatment. (Patient S only right leg treated); AL = Patient A left leg; AR = Patient A right leg; GL = Patient G left leg; GR = Patient G right leg; IL = Patient I left leg; IR = Patient I right leg; SL = Patient S left leg; SR = Patient S right leg
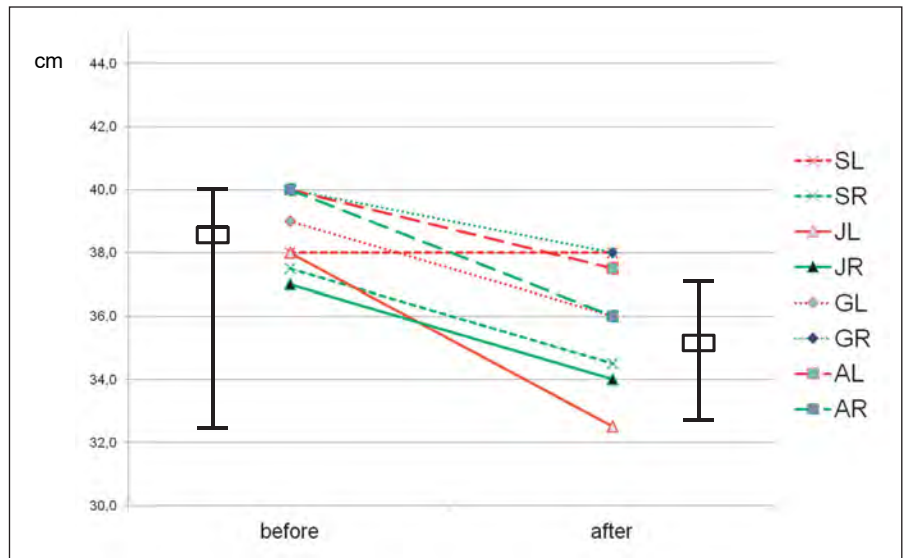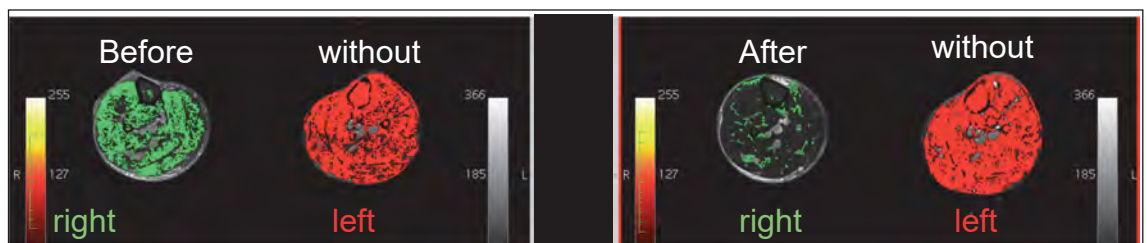

Fig. 4 T2-weighted MRI image of the lower leg of patient S before and after treatment of only the right leg, water content on the right coloured green, left red. The water content is markedly reduced in the right leg, but is unchanged on the left, untreated leg.

Tab. 1 Table showing volumes in ml measured from the sum of voxels of the MRI lower leg images of patients A, G, I, and S before and after treatment and of the maximum lower leg circumferences measured with a tape measure before and after treatment.

Name	Time	Volume right (ml)	Volume left (ml)	Circumference right (cm)	Circumference left (cm)
A	before	1355	1652	40	40
A	after	797	1085	36	37.5
G	before	411	835	40	39
G	after	104	384	38	36
I	before	664	615	37.5	38
I	after	613	544	34	32.5
S	before	692	579	37.5	38
S	after	136	(without) 484	34	(without) 38

of the leg are affected if the system is incompetent.

It appears logical that any incompetence of the system is predominantly manifest in the area chiefly responsible for its functioning, namely in the subfascial space.

This idea would accord with patient symptoms that can be explained by the fascial tension (22) of the subfascial region overloaded with fluid due to the subfascial stasis. It would also match the palpable finding of congestion in the deep veins of the calf.

I therefore assume that subfascial oedema is the pathophysiological correlate of diseases of the leg veins. I hypothesise that the oedema demonstrated in this MRI study is the pathophysiological correlate of what Browse has already called "The painful deep-vein syndrome" (23) and is also the substrate that Partsch attempted to differentiate as "The painful deep-vein syndrome" and "Pseudo-thrombophlebitis" (24). Subfascial oedema is the correlate of what Petter (25) described as symptoms of deep leg vein incompetence.

As long ago as the early 1970s, scintigraphic investigations by Haid and Partsch showed an increase in subfascial lymph transport in patients with inelastic compression therapy after thrombosis (26, 27) and thus provided an indirect demonstration of subfascial oedema.

This could be demonstrated because the subfascial space was already "boggy" due to the subfascial stasis (reabsorption deficit) that led to the thrombosis.

Conflict of interests

The author declares that there are no conflicts of interest.

Ethical guidelines

The preparation of this manuscript involved no studies in humans or animals.

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