Acta orthop. Scandinav. 39, 336-345, 1968.

Department of Surgery (Heads: O. Noring, M.D., and H. Paaby, M.D.).
The County Hospital, Nykøbing F, Denmark.

THROMBOSIS OF THE ULNAR ARTERY

By

H. PAABY & F. STADIL

Received 3.vii.67

INTRODUCTION

Thrombosis or aneurysms in the arteries of the hand are not often mentioned in the literature and are perhaps seldom diagnosed. It seems that until the publication of an article by *Kleinert & Voliantis* in 1965, only about 100 cases had been published. (*Butsch & Janes* 1963, *Kleinert & Voliantis* 1965). The disease was first described in the late 18th century by *Guettani* (ref. *Smith* 1962), but apparently attracted little attention. In Scandinavia *v. Rosen* published a case as early as 1934. *Leriche et al.* in 1937 mentioned the disease in their work on arterial resections in the treatment of peripheral vasospasm. For the past 20 to 30 years, several reports of single cases have appeared; some of aneurysms (*Middleton* 1933, *Smith* 1962, *Spittel* 1958, *Trevaskis et al.* 1964, *Zuckerman & Proctor* 1946) and some of thrombosis (*Butsch & Janes* 1963, *Costigan et al.* 1959, *Kleinert & Voliantis* 1965, *Spittel* 1958). These cases were predominantly in the ulnar artery at the level of the carpal bones.

ANATOMY

The arcus volaris profundus receives its main supply from the radial artery, while the arcus volaris superficialis gets its main supply from the ulnar artery. Before ending in the superficial arcus, the ulnar artery sends its ramus profundus to the arcus volaris profundus. The ulnar artery passes deep to the short palmar muscle, but superficially to the

We are greatly indebted to Dr. *P. Taarnhøj*, Head of the Neurosurgical Department, County Hospital, Glostrup and Dr. *H. Eltorm*, M.D., Head of the Radiolagical Department, County Hospital, Glostrup, for kindly placing their records and roentgenograms at our disposal.

ligamentum carpi transversum. Here it is situated just radial to the superficial branch of the ulnar nerve, which is predominantly a sensory nerve. Laterally it runs close to the pisiform bone and to the hamulus of hamatum. Since this location is close to the strong ligaments and bony structures, it might have a bearing on the aetiology.

AETIOLOGY

The reason why thrombi and aneurysms form in this part of the ulnar artery is presumedly because the artery in this area is especially exposed to trauma. It may be a question of repeated minor trauma, as in monotonous work, with consequent pressure of this segment of the ulnar artery against the deeper structures (*e.g.* working with pneumatic tools, gear shifts etc.). Or, it may be a question of a more acute, heavy trauma. Such trauma is described in many of the published cases (*Butsch & Janes* 1963, *Costigan et al.* 1959, *Jackson* 1954, *Kleinert & Voliantis* 1965, *Middleton* 1933, *v. Rosen* 1934, *Smith* 1962, *Trevaskis et al.* 1964, *Zuckerman & Proctor* 1946). Since such trauma is very common, it seems strange that the disease does not occur more often. The explanation might be that the relationship to trauma is not so precise, or that the disease is more common than supposed and that it is often misdiagnosed.

PATHOGENESIS

The formation of thrombi is presumably due to lesions of the intima, and the formation of aneurysms seems to occur mainly through lesions of the tunica media (*v. Rosen* 1934). Aneurysms might also be congenital, but in fact, most aneurysms seem to be of true acquired type and in some cases they are thrombosed.

SYMPTOMATOLOGY

The symptoms may vary considerably. The patient will often complain that a tender, tense tumour has developed in the hypothenar region, a sign which usually appears early in the disease. Besides there are often marked episodes of vasospasm. The patient will exhibit Raynaud's phenomenon and complain of pale and dead fingers, especially, when working, or exposed to the cold. The pallor might be accompanied by aching pain, a sensation of burning and cyanosis. In severe cases, ulceration or gangrene may develop. Neurological symptoms, such as par-

aesthesia and hypaesthesia in the sensory area of the ulnar nerve occurs, perhaps because of the close anatomical relationship between the diseased artery and the superficial branch of the ulnar nerve which may be compressed or irritated by aseptic inflammatory reactions around the thrombosed artery.

DIAGNOSIS

The diagnosis is made from the history, the subjective complaints mentioned above, and a positive Allen-test. This test is nearly always positive when the ulnar artery is thrombosed. If there is a non-thrombosed aneurysm, a swelling can be seen, which disappears when the artery is compressed above the site. In such cases, a bruit is often heard over the swelling.

ALLEN-TEST

This test is performed by compressing the radial artery at the wrist, while the patient forcibly opens and closes the hand a few times. Normally, a slight transitory ischaemia will be seen, which disappears quickly when the hand is kept still and compression maintained. When the ulnar artery is not functioning due to thrombosis, the pallor will persist for a long time during continued compression of the radial artery, and marked ischaemic pallor will be seen. In such cases, the test is said to be positive. During this final part of the examination, the fingers should not be hyperextended, since such hyperextension will result in a false positive reaction.

Positive results of the Allen-test will be sufficient to make the diagnosis. Further evidence can be obtained by brachial arteriography. Many authors emphasize that this is hardly necessary and a few authors warn against this method because of the possibility of provoking vasospasm, which might lead to an exacerbation of the disease. One participant in a discussion at the Mayo Clinic (*J. W. Lord,* quoted by *Butsch & Janes* 1963) mentioned a case where gangrene developed after arteriography.

TREATMENT

The treatment of this disease varies. It may be by conservative methods, using vasodilators, anti-coagulants or perhaps even steroid drugs and cessation of smoking. In addition, conservative surgical treatment such as sympathectomy, performed after a good response to injection of the

stellate ganglion, has been shown to be effective, but not uniformly so, because sympathectomy will eliminate vasoconstriction and does not stimulate the vasodilating apparatus (*Butsch & Janes* 1963).

Finally, there is radical surgical treatment consisting of resection of the injured part of the ulnar artery followed by ligation of the ends. The ultimate objective is (*Leriche* 1937) to remove the part of a vessel which is thought to provoke the vasospasm and to encourage the formation of collaterals. This treatment has been used effectively in the majority of reported cases. Furthermore, surgical treatment may be conservative with multiple arteriotomies and endarterectomies, or resection of the thrombosed area and end-to-end anastomosis of the vessel. This latter method has been successfully used by *Kleinert & Voliantis* (1965) in 9 cases. The authors did not follow up the patients arteriographically, but reported a good result in a case where the Allen-test again had proved positive.

It is still debatable whether this treatment is superior to resection and ligation.

CASE REPORTS

The disease is more common than what is indicated by the number of single cases reported. During the period of about one year, we observed 6 cases which are described below.

Case 1. A 49 year old labourer, whose mother had a tendency to vasospasm in the fingers. In 1959 the second right finger was amputated after an accident. On admission to hospital on 6th December 1965, he complained of prickling pain of about one year's duration in the middle and right fingers of his right hand. The attacks occurred 1 to 3 times a day and lasted from 5 minutes to 2 hours. During the attacks the fingers were pale and the spasms were followed by paraesthesia and pain. Cold and hard work provoked the attacks. No symptoms were seen in the other hand or in the lower extremities. At the onset of symptoms, the patient noticed swelling of the hand and lower forearm. Vasodilators were tried, but without success. In his work, he had been using pneumatic tools. Before he was admitted to hospital he had been incapable of working for about 9 months.

Physical findings: Slight arteriosclerotic changes at ophthalmoscopy. No evidence of costoclavicular arterial compression. Slight cyanosis in the middle and ring fingers of the right hand. No trophic changes. Allen-test was positive on the right side and negative on the left.

The diagnosis of thrombosis of the ulnar artery was based on the criteria mentioned above. The patient had been treated with infiltration of the stellate ganglion with a good, but transitory response. Transferred to the neurosurgical department, County Hospital, Glostrup, he was examined with brachial arteriography, which showed evidence of thrombosis at the level of the carpal bones (Figure 1).

A right thoracic sympathectomy of the the 2., 3., and 4. ganglion was performed with an immediate and good relief of the symptoms. After the operation some

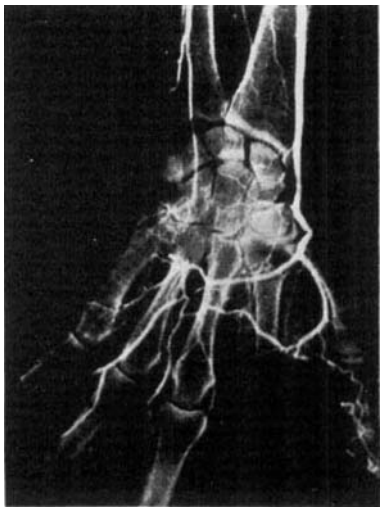

Figure 1. Brachial arteriogram showing occlusion of the ulnar artery at the carpus (Case 1).

neuralgic pains developed, due probably to a slight contusion of the brachial plexus during operation. Four months after operation, when the patient was reexamined, the neuralgic pains had subsided and the vasospastic symptoms had disappeared, but the Allen-test was still positive. The prognosis is thought to be good. If the symptoms recur, regional resection of the ulnar artery will be offered.

Case 2. A 66 year old man, who is a small holder. Six months before admission to hospital he was operated on because of benign pyloric stenosis. Three months before presentation, without preceding trauma, he noticed an increasing, somewhat tender swelling ot the base of the left hypothenar region. The swelling persisted for about 2 months. No signs of vasospasm.

Physical signs: On Februar 15, 1966, before admission to hospital, a slight tender lump of 2 by 1 cm in size was found at the base of the left hypothenar region. It was deep-seated, not movable and not circumscribed. The diagnosis was thought to be a deep-seated ganglion. When admitted to hospital on 1st March 1966 the lump and tenderness had decreased. He was operated upon three days later, using a bloodless field. It was then found that the ulnar artery was thrombosed and a small embolus proximal to the thrombosed area was noticed. The pathological changes were spread only in the area of the ramus superficialis. The artery was normal where the profound branch was given off. A slight inflammatory reaction, oedema, discoloration of the fat and a little serous fluid was found around the vessel. The superficial branch of the ulnar nerve was intact. Distal to the thrombosed area, the artery was likewise normal (Figure 2). A small arteriotomy was done. The degenerated intima was removed together with the embolus. After suture of the incision with atraumatic silk no. 00000 the blood flow was excellent through the artery, so that regional resection was not performed. In the post-operative phase the patient had a little stiffness of the fingers, which improved steadily with exercise and physiotherapy, and a slight paraesthesia in the ulnar area which subsided spon-

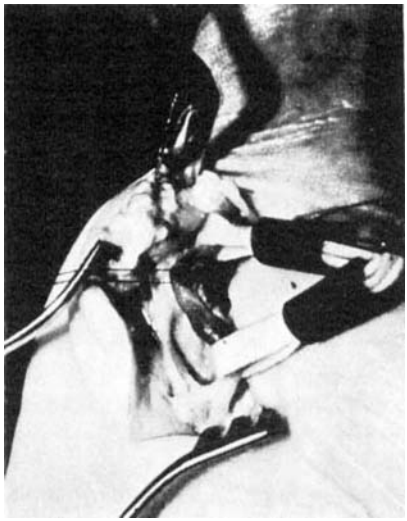

Figure 2. Operative findings in Case 2 showing occlusion of the ramus superficialis of the ulnar artery. The thrombosed area is seen between the slings, ulnarly the superficial branch of the ulnar nerve is exposed.

taneously. These symptoms were not present before the operation. The Allen-test had not been performed before operation because vasospasm had not been present and the disease had been misdiagnosed as a ganglion. One month later the positive Allen-test showed evidence that the artery had thrombosed again. If vasospastic symptoms recur, we shall consider reoperation and resection of the diseased part of the artery. This patient has no history of heavy or repeated minor trauma. He is a farmer, however, accustomed to heavy work and he is lefthanded.

Case 3. A 26 years old man, of low intelligence. On 20th May 1966 he sustained a heavy blow against the left hand on the volar side of the wrist. X-ray revealed no fracture. Swelling and ecchymosis were present, and the patient was treated by his general practitioner for sprain of the wrist. One week later there was some stiffness of the fingers together with tingling and hypaesthesia on the volar aspects of the three ulnar fingers. The patient was treated with ultrasonic waves and finger exercises. The condition of his fingers varied during this period. He complained of coldness of the fingers, which were from time to time cyanotic, but actual vasospastic attacks were difficult to trace because of his poor intellect. Pulsation was felt in the radial artery at the wrist, but not in the ulnar artery. 5 months after the trauma, further exacerbation of the symptoms with attacks of cyanosis, pains and swelling occurred. Slight hyperhidrosis and some atrophy of the skin were present, no radiological signs of Sudeck's atrophy were seen. The patient was again admitted to hospital and 6 injections of the stellate ganglion were given. Percutaneous brachial arteriography showed filling of the arcus volaris profundus from the radial artery, but no filling in the arcus volaris superficialis. Nevertheless, there was filling of the very ulnar part of the superficial branch of the ulnar artery. Injections of the stellate ganglion were continued and anti-coagulating therapy was started simultaneously. After 2 months, there was improvement and at the last follow up there were no signs of Sudeck's atrophy.

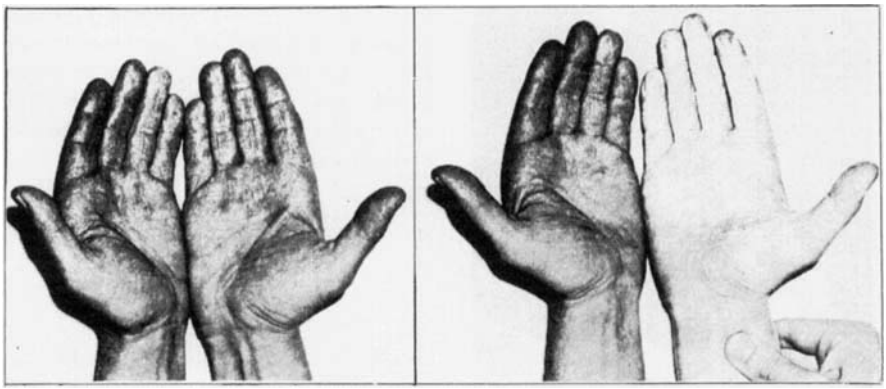

Figure 3. Positive Allen-test. The hands before and during compression of the right radial artery (Case 4).

Symptoms: The Allen-test was equivocal, but the arteriographic findings pointed to a traumatic thrombosis located peripherally in the superficial branch of the ulnar artery. Very recently the patient had a new excacerbation of the symptoms. He was admitted to hospital and operated upon on the 17th of May 1967. The diagnosis was confirmed at operation, the superficial branch of the ulnar artery was partly thrombosed, and resection of the diseased part of the artery was performed. The primary result after the operation was very promising.

Case 4. A 44 years old man, a brewer's driveer, had for 2 years had a lump of 1 by 2 cm in size at the base on the palmar side of this right middle finger. For 21 months there were no symptoms. Three months before the operation on 3rd November 1966, he began to have vasospastic attacks in the right middle ring and little fingers. The attacks were provoked by cold, and work during which he carried heavy wooden beer containers, whose edges always pressed against the ulnar part of the carpus.

The tumour was removed on November 3rd, 1966. It was a benign fibroma. The vasospastic attacks persisted unchanged. No sensory disturbances were found. The Allen-test was positive on the right side (Figure 3), but not on the left. He was right-handed.

Two and a half months later he was seen again. The Allen-test still was positive, but the symptoms had decreased considerably. When working he now avoided pressure against the base of the hypothenar region. This was slightly inconvenient, but he refused further operative treatment. No acute trauma could be traced as the cause of the disease.

Case 5. A 54 years old police sergeant. One year before presentation he had had a direct trauma against the volar aspect of the wrist, followed by swelling and ecchymosis of booth sides of the hand. X-ray showed nothing abnormal. For the next few months paraesthesia in the area of distribution of the ulnar nerve developed, together with weakness in the ring and little fingers as well as vasospastic attacks in the middle, ring and little fingers. During the attacks ischaemia was severe, with

irregular cyanosis of the pulp of the ring finger. The attacks were provoked by cold or by use of the hand, especially, when pressure was applied to the hypothenar area. For example, the patient stated that the was unable to handle an axe or a chisel for any length of time. During this period he had tenderness in the region of the hamulus of hamatum and also suffered from vasospastic attacks.

Physical signs: No neurological symptoms and no signs of reflex dystrophy were found. There were paleness and low skin temperature of the right middle, ring and little fingers, with slight cyanosis on the pulp of the ring finger together with small ecchymoses at the free edge of the nail. Since the vasospasm had started, the patient had noticed that the ecchymoses occurred particularly when the nail was cleaned with a nailfile. The Allen-test was positive.

The patient was able to work, but with some inconvenience. In accordance with his own wishes, no treatment was given.

Case 6. A man, 29 years old ,carter, fractured his right carpus 11 years ago. Six years later he fractured the right ring finger. On arrival at hospital on 22nd February, 1967, he complained that for three months he had had attacks of pain and coldness in the third finger of his right hand. He had had no recent trauma. The symptoms were provoked by cold. 7 weeks before admission, his own doctor made an incision in the pulp of the finger believing that he was suffering from a pulp space infection. This was not borne out. The wound was very slow in healing. The patient went to work regularly until admission to hospital.

Physical signs: Pallor and coldness were noticeable in the middle, ring and little fingers. No swelling. The middle finger was not completely healed. The Allen-test was negative on the left side and positive on the right. Good pulsation in the radial and ulnar arteries was felt at the wrist. No neurological symptoms. At examination on admission, no pulsation was felt in the radial artery when the arm was elevated 120°. A bruit was heard over the subclavian artery. When reexamined 2 days later all signs of costoclavicular arterial compression had disappeared, but the Allen-test was unaltered. Radiological examination of the cervical vertebral showed nothing abnormal.

However, for about two weeks before admission, the symptoms had been subsiding quickly. The patient was rather impatient and wished to return to his work and was discharged a few days later.

This patient had signs of costoclavicular arterial compression as well as of stenosis of the ulnar artery below the wrist. The circulatory status of this patient could have been investigated with advantage with arteriography. However, we did not perform this examination because of its possible risks, and because of the remission of the symptoms. We believe, however, that the symptoms and the physical findings definitely point to thrombosis of the ulnar artery distal to the wrist.

DISCUSSION

The disease can be confused with some systemic diseases. *Allen, Barker & Hines* (1962) point out that a distinction should be made between idiopathic Raynaud's disease and the Raynaud's phenomenon. In Ray-

naud's disease vasospasm and cyanosis are not related to another disease. Raynaud's phenomenon is a symptom of such diseases as post-traumatic Sudeck's atrophy, costoclavicular arterial compression, scalenus anterior syndrome, occlusive disease of an artery, intoxications (lead, ergot) or occur in association with systemic diseases (scleroderma, lupus erythematosus disseminatus, rheumatoid arthritis, dermatomyositis, paroxysmatic haemoglobinuria, myelomatosis, chronic leukaemia and idiopathic cryoglobulinuria).

The characteristic feature of Raynaud's phenomenon in this disease is that it is unilateral and confined to single fingers (ulnar ones only).

It is differentiated from neurovascular compression by the hyperabduction-test or Anson-test. In scleroderma, the skin shows characteristic changes. In its later stages the disease may be characterized predominantly by Raynaud's phenomenon, while in its early stages, where the lesions of the artery are recent, it may simulate a tumour of the hand (case 2). The disease is often misdiagnosed as Sudeck's atrophy (case 5, referred to examination suspected of this, and case 3), but this diagnosis can often be ruled out by the typical radiological changes and by changes in the skin. Eventually, the picture can be dominated by an associated irritative neuritis of the ulnar nerve, showing paraesthesia in the ulnar area, as in case 4. If such a patient has a co-existing tumour of the hand, the disease will easily be misdiagnosed, especially if attention is not focused on the vasospastic attacks. These seem to be the dominating symptoms in the later stages of the disease.

If the disease is of recent origin, active treatment may be postponed, and if the actual region of the hand can be spared, some patients might be able to perform their usual work (cases 3, 4 and 5). When the vasospasm becomes more pronounced, operation should be done, and in our opinion preferably with resection and ligation or endarterectomy.

SUMMARY

A description of thrombosis of the ulnar artery is given. Like the formation of aneurysms in this artery, the disease often seems to be of traumatic aetiology. The anatomical basis, the aetiology and the pathogenesis are discussed. 6 cases are reported, the diagnosis was verified by operation, arteriography and particularly by a positive Allen-test. It is emphasized that the disease is more common than generally supposed and that it is often misdiagnosed. Treatment in the later and more severe stages is operative.

RESUME

Description d'une thrombose de l'artère cubitale. Comme la formation d'un anévrisme dans cette artère, la maladie semble souvent avoir une étiologie traumatique. Il est discuté de la base anatomique, de l'étiologie et de la pathogénèse. 6 cas sont rapportés. Le diagnostic a été vérifié par l'opération, l'artériographie et en particulier par un test Allen positif. Il est souligné que la maladie est plus fréquente qu'on ne le suppose généralement et qu'elle est souvent mal diagnostiquée.

ZUSAMMENFASSUNG

Eine Beschreibung der Thrombose der a.ulnaris wird gegeben. Die Erkrankung scheint ebenso wie die Bildung eines Aneurismas in dieser Arterie oft auf traumatischer Grundlage zu entstehen. Die anatomische Basis, die Athiologie und die Pathogenese werden besprochen. 6 Fälle werden berichtet. Die Diagnose wurde durch die Operation, die Arteriographie und besonders durch eine positive Allen-Probe bestätigt.

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