A CASE OF UNILATERAL CLUBBING OF THE FINGERS

WITH A SUMMARY OF THE LITERATURE

BY

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The first observer to describe this rare condition was Ogle in 1859. Others to describe and collect cases were Béclère (1901), Castex and Mazzei (1939), Groedel (1907), Hatzieganu (1923), Högler (1920), Loucaides (1932), Mendlowitz (1938), Pigney (1920), Souques (1919), and Walsh and Alldredge (1934). A case observed recently is now described.

Case History

A male quarry worker aged 54 was admitted complaining of pain in the right arm and a lump on the right side of the neck for six months. At first the lump was not painful, but it gradually increased in size and pain occurred, first in the right upper arm and later in the forearm. The patient also noticed a change in the shape of his fingers. There was no cough or dyspnoea, and no history of syphilis was admitted.

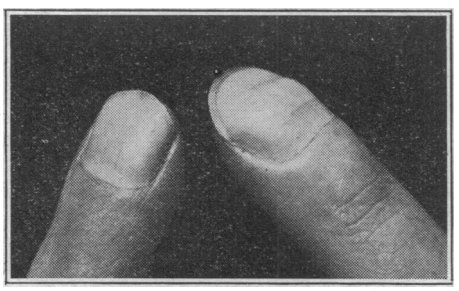

FIG. 1.

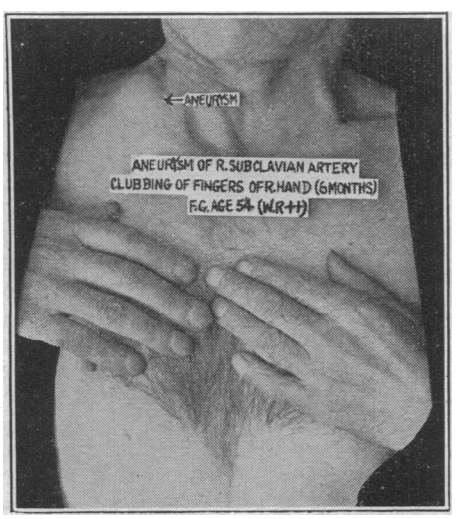

FIG. 2.

On examination a pulsatile swelling, 2½ by 2¾ inches, was seen just above the right clavicle; a systolic thrill and murmur were present over the swelling. There was pitting oedema and venous engorgement of the right upper limb. The right hand was constantly warmer than the left. There was pronounced finger clubbing of the right hand and curvature of the nails; the left hand was normal (Fig. 1). The power in the right upper limb was poor compared with the left (the patient was right-handed). The sensation and reflexes were normal in both arms, and the circumference of the arms 3 inches above the elbow was: right 8½ inches, left 9 inches (measured after oedema had subsided with rest and one month's treatment). The pulse at the right wrist was less forceful than that at the left. Blood-pressure readings were: right arm 118/84, left arm 124/88. The heart showed no enlargement clinically, but there was a systolic murmur over the aortic area. The pupils reacted to light and accommodation; the knee-jerks were present. No tracheal tug was observed. The sedimentation rate (Cutler) was 25 mm. in one hour, and the Wassermann reaction was strongly positive. Radiographs showed: (1) extensive soft-tissue shadow above the right clavicle, but nothing abnormal in the chest; (2) slight decalcification of both hands, right greater than left; (3) slight decalcification of bones of right forearm.

The case was diagnosed as syphilitic aortitis, with right subclavian aneurysm and secondary unilateral clubbing of the fingers of the right hand; and rest, potassium iodide, and mercury were prescribed. Later bismostab 0.2 gramme was given weekly. The aneurysm gradually decreased to about three-quarters of its original size, and the oedema and much of the pain and venous dilatation disappeared.

The patient, when seen six months later, had voluntarily discontinued treatment for three months; his aneurysm was much larger, and the clubbing more pronounced (Fig. 2). He has recently been operated on successfully, the aneurysm having been tied off both distally and proximally. It is too early to assess any change in the clubbing, but all his symptoms have disappeared.

Discussion

Clubbing of the fingers occurs bilaterally in various conditions, chiefly in cardiac and pulmonary disease. Other states, such as cirrhosis of the liver, ulcerative colitis, sprue, amoebic dysentery, and post-thyroidectomy myxoedema, occasionally present clubbing (Mendlowitz, 1938). It therefore seems difficult to deduce a single cause, and circulatory, chemical, and nervous factors may all play a part.

Cases reported in the Literature

Reporter	Observer	Accompanying Lesion
Béclère (1901)	Béclère	Aneurysm of R. subclavian artery
Castex and Mazzei (1939)	Castex and Mazzei	Innominate and R. subclavian aneurysm
,,	,,	Aneurysm of L. subclavian artery
Groedel (1907)	Groedel	Aneurysm of descending arch—compression of L. subclavian artery
,,	Bernhardt	Aneurysm of L. subclavian artery
,,	Berent	,, ,, ,,
,,	Ogle	Aneurysm of R. subclavian artery
,,	Canton	Aneurysm of subclavian artery
,,	Thomas Smith	,, ,, ,, ,,
,,	Osler	Intrathoracic aneurysm (exact site not given)
,,	,,	,, ,, ,, ,,
Hatzieganu (1923)	Hatzieganu	Aneurysm of R. brachio-cephalic trunk
Högler (1920)	Joachim	Aneurysm of aortic arch
,,	Ebstein	Aneurysm of R. subclavian artery
,,	Klausa	Old dislocation of shoulder
,,	Forster	Aneurysm of aortic arch and subclavian artery
,,	Hatiegan	,, ,, ,, ,,
Loucaides (1932)	Loucaides	,, ,, ,, ,,
Mendlowitz (1938)	Mendlowitz	R. subclavian aneurysm
,,	,,	Aortic aneurysm
,,	,,	Phlebectasia
,,	,,	Carcinoma of R. upper lobe bronchus
Pigney (1920)	Pigney	Dilatation of ascending aorta and aortic arch
,,	De Jong	Bullet which passed through axillary vascular region. No exact details of lesion
,,	Baur	Aneurysm of L. subclavian artery
Souques (1919)	Souques	Phlebectasia
Walsh and Alldredge (1934)	Walsh and Alldredge	Aneurysm of R. innominate artery
,,	Barney Brooks	Aneurysm of R. axillary artery
,,	Stanley Smith	Aneurysm, site unquoted

Of the 29 cases of unilateral clubbing of the fingers reported in the literature, 24 occurred with aneurysms, chiefly of the subclavian arteries (see Table). In the remaining 5 pressure was observed on the vessels and nerves of the affected limb. This pressure seems to be the only factor common to all the 29 cases, but it is not clear whether it was on the arteries, veins, nerves, or lymphatics, or on a

combination of these, as the clinical signs accompanying the clubbing varied considerably. In the case reported above there was, clinically, obstruction of the vessels as shown by oedema, venous dilatation, pulse, and blood pressure. Subsequently, pressure on the nerves gave rise to pain.

Ogle (1859) thought that the clubbing was due to the presence of an increased amount of venous blood in the affected limb and that this was caused by interference with the axillary circulation. Other observers thought it was due to interference with the sympathetic nerves running with the vessels, but as it is now known that sympathetic nerve fibres do not run down the whole length of the arteries this theory seems untenable.

Mendlowitz (1938) investigated the circulation in the arms of 4 cases of unilateral clubbing and found no evidence of a circulatory cause. He compared the maximum heat elimination and blood pressure gradient from brachial to digital areas in both the normal and affected arms and found that no significant changes in the blood flow had occurred in the affected finger-tips. However, only 4 cases were investigated, and the results of the experiments were not very conclusive. Local anoxaemia due to circulatory embarrassment still remains a very probable cause of clubbing.

Chemical causes, such as toxaemia, seem very probable when bilateral cases are considered. Thus, bronchiectasis, a disease with general toxaemia, is commonly accompanied by bilateral clubbing. However, if general toxaemia were always the cause, unilateral clubbing should never occur. There remains the possibility that the localized pressure, for instance from an aneurysm, might precipitate incipient clubbing on one side, and that the other side might eventually show clubbing if the patient lived long enough. It is interesting to note that cases of an aneurysm causing pressure and pronounced clubbing in one hand have sometimes shown slight clubbing of the other hand (Béclère, 1901 ; Hatzieganu, 1923). It seems unlikely that pressure on the nerves of the arm could be the cause of clubbing, as although nervous symptoms are common in unilateral cases they do not occur in bilateral cases.

Finally, it may be noted that in 2 of the reported cases the clubbing disappeared wholly or partially after operation on the aneurysm (Hatzieganu, 1923 ; Högler, 1920). This is convincing evidence that pressure is the primary factor in unilateral cases. Local circulatory embarrassment follows this pressure, causing anoxaemia and, hence, probably the clubbing. Anoxaemia from circulatory embarrassment or general toxaemia in bilateral cases may well account for the clubbing, so conforming with the final factor in unilateral cases.

Summary

A case of unilateral clubbing of the fingers is described.

The cases previously reported are tabulated.

The difficulty of explaining the exact causation is stressed and the possibilities are discussed.

I wish to thank Prof. E. J. Wayne for permission to publish this case, and Dr. L. C. D. Hermitte for the photographs.

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Medical Memoranda

Cirrhosis of Liver and Perforated Gastric Ulcer in an Infant of 6 Months

The only type of infantile cirrhosis which I can trace in medical literature is one which occurs in India. It was first described by Twining in 1832, and its morbid anatomy was investigated by Gibbons in 1891. A full account of this disease is given by Sir Upendranath Brahmachari and Phanindranath Brahmachari in volume 8 of the *British Encyclopaedia of Medical Practice*. I can find no record of a similar condition in this country, nor of any case in which the terminal event was a perforated gastric ulcer, though Brahmachari refers to gastric and intestinal haemorrhage as a common complication. A non-syphilitic cirrhosis, originating as an intralobular and progressing to a multilobular cirrhosis, occurring in an infant of this age and terminating in a perforated pyloric ulcer at 6 months must be very uncommon in this country ; and if the brother's liver was also cirrhotic the condition must be, as in the Indian cases, familial. Two other children appear to be healthy.

CASE RECORD

A male child aged 5½ months was sent to me by Dr. Bruce of Newport. The history was that he weighed 9 lb. at birth, and that, beyond a somewhat intense icterus which persisted for some days, the puerperium was normal. It was found, however, that the infant did not thrive on the breast. Dried milk preparations did not result in improvement, and on these the stools were fatty in appearance. On sweetened condensed milk he thrived for a time, but on resuming dried milk he lost weight. It was found that the liver was very large, extending to the umbilicus. No free fluid was detected in the abdomen, and there was no enlargement of the spleen. There had been no jaundice since the neo-natal period. A former child of the same family had also had a much enlarged liver, and had died of pneumonia at 2 years of age. A provisional diagnosis of von Gierke's (glycogen) disease was made.

The blood sugar was first investigated. The fasting blood sugar was 100 mg. per 100 c.cm. ; one hour after a feed of 5 oz. ostermilk it was 225 mg., and after two hours 160 mg. Next the effect of adrenaline was observed, for in von Gierke's disease this drug does not raise the blood sugar. On this occasion the fasting blood sugar was 140 mg. ; 5 oz. of adrenaline 1 in 1,000 raised this to 160 mg. The urine contained glucose and acetone. The blood count was: red cells, 3,700,000 per c.mm. ; haemoglobin, 80% ; white cells, 12,400 per c.mm. (polymorphs 29%, lymphocytes 70%, large mononuclears 1%). The Wassermann reaction was negative. I concluded that this was not a case of von Gierke's disease, but a true diabetes mellitus with hepatomegaly, and decided to try the effect of 3 units of soluble insulin twice a day. On the second day the infant suddenly collapsed and died at 11.15 a.m.—four hours after his third dose of insulin. Just before death he passed a quantity of blood per rectum. At no time did he seem to have any pain.

Post-mortem Examination.—The abdomen contained about 8 to 10 oz. of cloudy fluid. The liver had the typical appearance of multilobular cirrhosis. On raising the liver I discovered the cause of the cloudiness of the fluid in the abdomen, for a perforated ulcer of the pylorus on the greater curvature was exuding partially digested milk, and this, mingling with the straw-coloured ascitic fluid, produced the turbid appearance. The small and large intestines were full of blood. The spleen was normal in size. The liver weighed 22 oz. The pathological report on the liver was as follows: "Sections show considerable cirrhosis ; this is to some extent of multilobular type, but numerous delicate fibrous bands are seen surrounding most of the individual lobules, and conforming more to a biliary (Hanot's) cirrhosis, though there is no evidence of active bile-duct regeneration."

My thanks are due to Dr. Bruce for his account of the history of this case, and to Dr. Douglas Thornton for his report on sections of the liver, as also to his staff for pathological investigation.

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