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surface and involving approximately 30% of the articular surface.

The comminuted osteoarticular fragment could not be fixed and was excised. The UCL was repaired to the palmar base of the proximal phalanx with a suture anchor. The RCL was repaired to the metacarpal head using a docking technique. The dorsal capsule was repaired with nonabsorbable sutures. The joint was stable and was pinned in a neutral position. The sagittal bands were then repaired.

IP joint range of motion was started with the hand in a thumb spica splint. The transarticular pin was removed after 4 weeks and active range of motion exercises were started. The MP joint dislocates palmarwards because of hyperflexion of the joint as the flexed phalanx is forced palmarwards (Mata et al., 1991; Miyamoto et al., 1986; Shingal, 1974). This was reproduced in a cadaveric study (Wood and Dobyns, 1981). Palmar displacement of the thumb MCP joint cannot be reproduced unless both the dorsal capsule and the volar plate are disrupted (Miyamoto et al., 1986). In our patient, the combined fracture and associated ligament injuries (radial and ulnar collateral ligaments of the MCP joint and sagittal bands) caused the instability.

Previously reported cases also had a torn dorsal capsule and ulnar collateral ligament. Many were due to motor vehicle accidents where the metacarpophalangeal joint became hyperflexed and radially deviated. Most also report a partial tear of the extensor pollicis longus. In our case there was a complete avulsion of this tendon's attachment which was repaired with the dorsal capsule. Many cases of palmar dislocation cannot be successfully reduced because there is interposition of torn dorsal capsule, volar plate, or extensor tendons (Miyamoto et al., 1986; Wood and Dobyns, 1981) and open reduction is required if closed reduction fails (Mata et al., 1991).

Conflict of interests

None declared.

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The five-fingered hand anomaly might be considered as a variant of the atypical mirror hand

Dear Sir,

The five-fingered hand anomaly has been classified on morphological grounds as a type of triphalangeal thumb. Buck-Gramcko (1998), based on the morphology of the anatomical structures of the triphalangeal thumb, described a teratogenic sequence of the anomaly from the most rudimentary triangular middle phalanx to a fully developed five-fingered hand. However, recent advances in the understanding of developmental processes have shown that morphologically similar structures may result from different embryological processes.

Miura (1976) and Wood (1976) have recognized two groups of triphalangeal thumbs. The first group consists of those with an intercalated phalanx. The intercalated phalanx might be delta, rectangular, or normal in shape. The thumbs are opposable and the thenar musculature is developed. The chief complaints are about angular deformity and/or the extra length of the thumb. The second group has a fully developed finger instead of a thumb. The finger is in the same plane as the other fingers. The hand appears to be five-fingered. There is no thenar musculature and the patients have side to side lateral pinch instead of typical opposition. The epiphyseal plate of the first metacarpal in the opposable triphalangeal thumb is at the proximal end and in the non-opposable triphalangeal digit is at the distal end (Miura, 1976). Miura (1976) suggested that some cases of non-opposable triphalangeal thumb, which are referred to as a five-fingered hands, might be considered to be an absent thumb with variable duplication of the index finger.

The treatment of the opposable triphalangeal thumb is by correction of the angular deformity and length reduction, whereas pollicization has been recommended for the treatment of five-fingered hands (Buck-Gramcko, 1998; Wood, 1976). Therefore, it might be appropriate to distinguish between the five-fingered hand and the four-finger hand with the triphalangeal thumb (Lamb et al., 1983).

The apical ectodermal ridge (AER) is responsible for proximodistal development of the embryonic limb bud and secretes a group of fibroblast growth factors (FGFs) that regulate the normal limb outgrowth (Al-Qattan et al., 2009). The zone of polarizing activity (ZPA), located on the posterior margin of the limb bud, controls the radioulnar patterning through the mediation of sonic hedgehog (Shh). The complex interrelations between the ZPA and the AER are a part of a feedback loop. Shh signals from the ZPA regulate the activity of the AER. The AER gives positive feedback to the ZPA by FGF signals. The presence of abnormally high Shh signals on the radial (preaxial) side, as produced by transplantation of the ZPA, produces ulnar dimelia and mirror hand (Al-Qattan et al., 2009).

The five-fingered hand anomaly might be produced by altering the delicate feedback regulations between the ZPA and the AER activities after development of the forearm. Progressive reduction of FGFs from the AER leads to truncation and decreases limb volume, affecting the radius and thumb. The development of the thumb is not dependent on the Shh signals (Al-Qattan et al., 2009). However, the presence of ectopic Shh signals on the radial side of the index finger leads to finger duplication (Al-Qattan et al., 2009).

Typical ulnar dimelia has some symmetry around the midline axis. The features of atypical ulnar dimelia include a forearm that contains both a radius and an ulna, absence of the thumb and preaxial polydactyly, which might be not necessarily be symmetrical around the midline (Al-Qattan et al., 1998). Al-Qattan et al. (1998) have described a case of atypical mirror hand with an ulna and a hypoplastic radius and found a case of mirror hand with five digits and two cases of atypical mirror hands with a well-formed radius and ulna in their review of previous studies. Mirror image polydactyly without ulnar duplication might be considered as a variant of the mirror hand anomaly. It seems that the five-fingered hand anomaly might be considered to be a variant of the atypical mirror hand anomaly rather than a four-fingered hand with a triphalangeal thumb.

Conflict of interests

None declared.

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Acute-on-chronic carpal tunnel syndrome after correction of wrist drop using tendon transfers

Dear Sir,

A 51-year-old man presented with inability to extend his right wrist and fingers since all the extensor tendons of the wrist and fingers had been severed by a straw cutter 38 years previously. He had received no surgical treatment except for primary skin closure at the time of injury. Under general anaesthesia, multiple tendon transfers were performed. The donor tendons were the palmaris longus, the ring flexor digitorum superficialis (FDS) and flexor carpi ulnaris. The recipient tendons were the extensor pollicis longus, extensor carpi radialis brevis and extensor digitorum communis respectively. The ring FDS tendon was routed through the interosseous membrane. Postoperatively, a short arm splint was applied in 30° of wrist extension.

Immediately after the operation, the patient complained of severe pain and tightness of the hand. He had very little active movement of the fingers and passive extension of the fingers was very painful. Examination showed hypoaesthesia in the distribution of the median nerve and the two-point discrimination increased to 15 mm in the index finger and thumb. The splint was immediately changed from wrist extension to the neutral position, resulting in the temporary relief of the severe pain. However, some pain and paraesthesiae in wrist extension lasted 3 weeks after the operation despite reduced wrist swelling. The electrophysiological findings were consistent with a median nerve lesion in the wrist.

The median nerve was explored at 3 weeks after the tendon transfers, finding that the flexor retinaculum was hypertrophied and the median nerve was enlarged from 2 cm proximal to the proximal edge of the flexor retinaculum to its distal edge (Fig 1). The thickened median nerve was tight and pressed against the deep structures of carpal tunnel when the wrist was extended passively, but tension in the nerve was relieved by flexing the wrist. The FDS tendon routed through the interosseous membrane was not compressing the median nerve decompression. Ten months later, the patient was symptom-free, and nerve conduction studies and