Nail Tumors: Clinical Overview

17

Robert Baran

Contents

17.1 Introduction

The diagnostics of disease of the nail and adjacent tissues is frequently aided with the use of radiography, magnetic resonance imaging, and scintigraphy. However, the recent technological advances in sonography, particularly in 3D, have changed the appearance of the diagnosis and consequently the management of nail diseases. Ultrasound is an easy-to-use and more cost-effective tool that has rendered visible what was previously invisible, and clarified what was once difficult to see. It has become an essential tool in dermatological and surgical diagnosis.

17.2 Warts

Common warts are caused by human papilloma viruses of different DNA types. They are benign, weakly contagious, fibroepithelial tumors with a rough keratotic surface. They are most frequently located on the lateral aspect of the proximal nail fold (PNF) and they spread onto the dorsum of the entire fold (Fig. 17.1). Tender nodules beneath the PNF are infrequent [1, 2] and rarely result in longitudinal grooving. Subungual warts affect the hyponychium initially, growing as pseudotumors slowly toward the nail bed and finally elevating the nail plate, that is not often affected although surface ridging may occur. Subungual warts are painful and can mimic a glomus tumor.

Bone erosion from verruca vulgaris has been observed. However, some of those cases may have been keratoacanthomas because the latter, as well as epidermoid carcinoma and verruca vulgaris, are sometimes indistinguishable from clinical signs alone. Therefore, in longstanding warts in the nail area, histologic examination may be necessary to differentiate extensive periungual warts from verrucous Bowen's disease.

On sonography, subungual warts appear as hypoechoic structures mostly of a fusiform shape, associated with thickening of the nail plates and interplate spaces. Warts

R. Baran, MD
Nail Diseases Center, 42, rue des Serbes, Cannes 06400, France

Department of Gustave Roussy Cancer Institute, Villejuif, France
e-mail: baran.r@wanadoo.fr, baran.r@club-internet.fr

X. Wortsman, G.B.E. Jemec (eds.), *Dermatologic Ultrasound with Clinical and Histologic Correlations*,
DOI 10.1007/978-1-4614-7184-4_17,

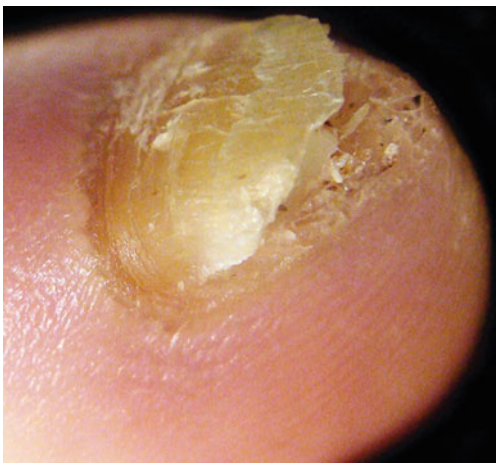

Fig. 17.1 Subungual wart

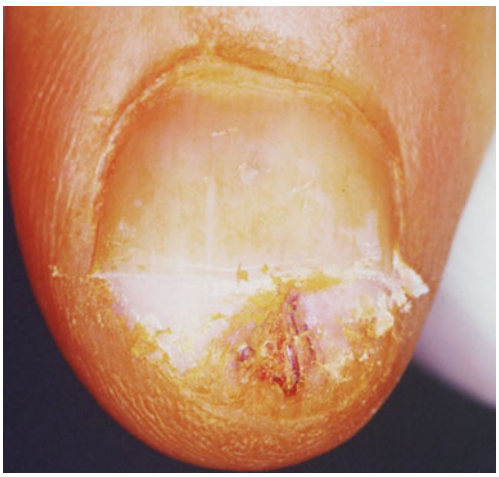

Fig. 17.2 Subungual keratoacanthoma

tend to have a nodular shape and can produce secondary thickening of the nail plates along the same axis when localized in the proximal nail bed. Warts are usually hypovascular, nevertheless, when they involve the hyponychium, they can produce dermal hypervascularity in the affected areas.

17.3 Distal Digital Keratoacanthoma

Subungual and periungual finger keratoacanthoma (KA) can occur as solitary or multiple tumors [3, 4]. KA is a rare, benign, but rapidly growing, seemingly aggressive tumor usually situated below the edge of the nail plate or in the most distal portion of the nail bed. Multiple subungual KAs without KAs on other sites are exceptional [5]. In three cases the distal phalanx of the toe was affected. Spontaneous resolution of KA is rare in the nail area [6].

The lesion can start as a small and painful keratotic nodule, visible beneath the free edge and growing rapidly to a 1–2 cm lesion within 4–8 weeks. Its typical gross appearance as a dome-shaped nodule with a central plug of horny material filling the crater is not often seen subungually, although histology of an adequate biopsy specimen will clearly show the characteristic pattern. Less frequently, the tumor grows out from under the PNF, which becomes inflamed and can cover or surround it with a cushion of swollen tissue (Fig. 17.2). The tumor then erodes the bone which can be seen radiologically as a fairly well-defined crescent-shaped lytic defect of tuft adjacent to the overlying nail bed. Reconstitution of the bony defect can be expected.

Diagnosis of distal digital keratoacanthoma is dependent on the rapid growth, bone erosion, and characteristic histology. Its clinical differentiation from squamous cell carcinoma is nevertheless difficult and the tumor is frequently diagnosed histologically as a squamous cell carcinoma (keratoacanthoma type). On sonography, well-circumscribed masses with mixed echogenicity (anechoic-hypoechoic), cortical remodeling or erosion of the bony margins, as well as associated posterior acoustic enhancement have been described.

17.4 Onychomatricoma

The are four main clinical signs that are striking enough to either make the diagnosis of onychomatricoma or at least to arouse suspicion of the condition that we described 25 years ago [7–19].

The first is a yellow longitudinal band of variable width that leaves a single or double portion of normal pink nail on either side (Fig. 17.3a). Splinter hemorrhages can be seen in the yellow area involving the proximal nail region in a characteristic manner and longitudinal ridging is prominent in the affected nail. The second sign is a tendency toward transverse overcurvature of the affected nail portion that becomes more pronounced as the yellow color becomes extended.

The third sign is shown after nail avulsion, exposing a villous tumor (Fig. 17.3b) emerging from the matrix while the nail appears as a thickened funnel, storing filamentous digitations of matrix fitting into the holes of the proximal nail, the fourth sign (Fig 17.3c). The villous projections in the nail plate can be so pronounced that nail trimming may produce bleeding [14]. However, in some cases the clinical presentation can be confusing: longitudinal melanonychia can hide the yellow hue and the PNF can become swollen at its junction with the lateral nail fold. The swelling gives the affected nail the texture of a cutaneous horn, and in some cases, the horn is completely separated from the nail plate. Histological examination establishes the diagnosis [15]. In some cases,

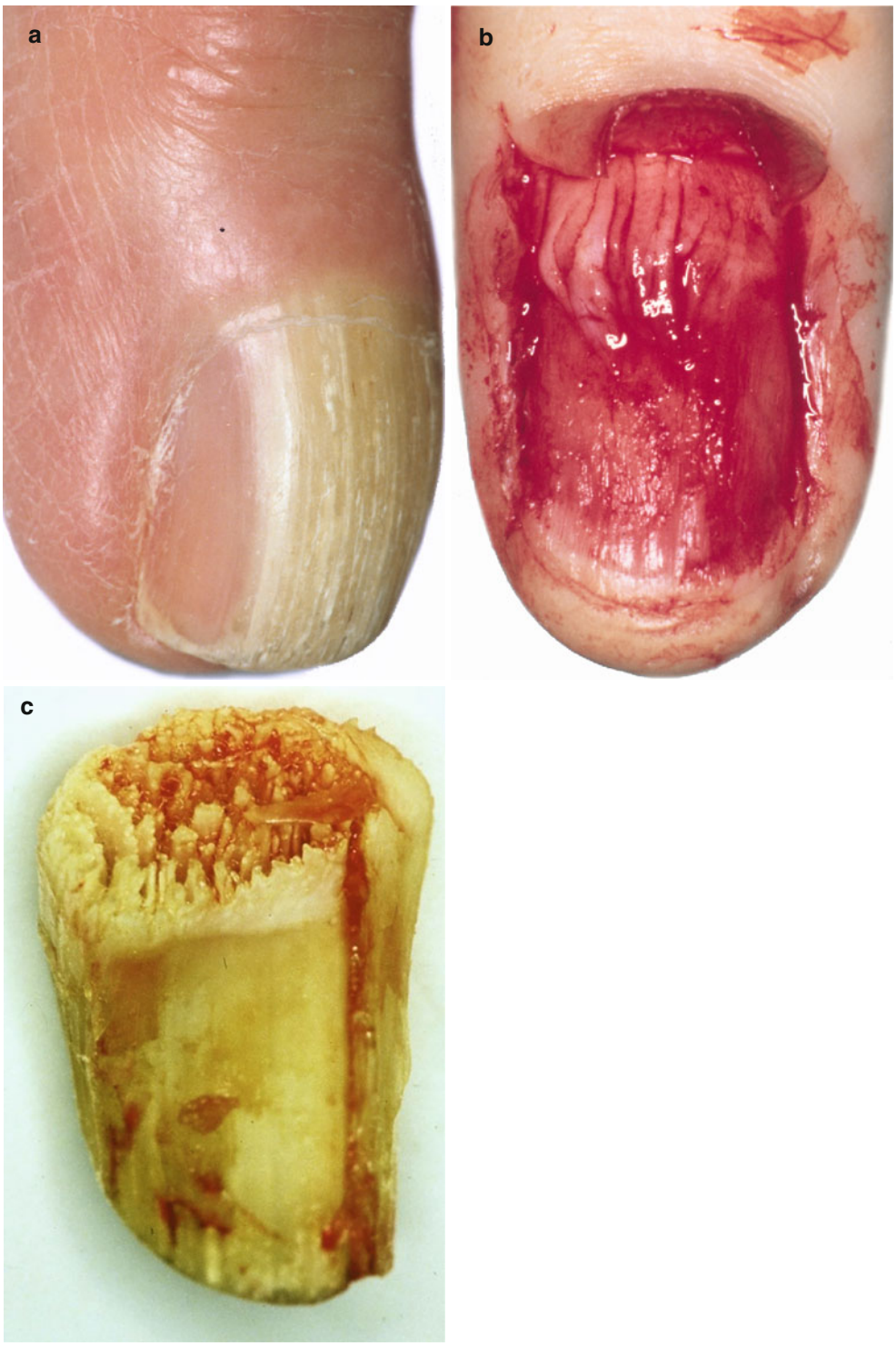

Fig. 17.3 (**a**) Onychomatricoma. (**b**) Digitations derived from the matrix. (**c**) Holes of the proximal nail extremity are clearly visible

the tumor is associated with onychomycosis and longitudinal melanonychia [18].

This fibroepithelial tumor consists of two anatomical zones and three histologic criteria are used for each [10]. The proximal zone is located beneath the PNF with a proximal border starting at the root of the nail and a distal border corresponding to the cuticle. It is characterized by (1) deep epithelial invaginations filled with a thick V-shaped keratogenous zone; (2) a thickened nail plate without cavitation but with an undulating inferior border ending in ungual spurs; and (3) a fibrillar stroma clearly demarcated from the undersurface.

There are two histologic differential diagnoses that can be discussed [15]. In the first, longitudinal sections in the

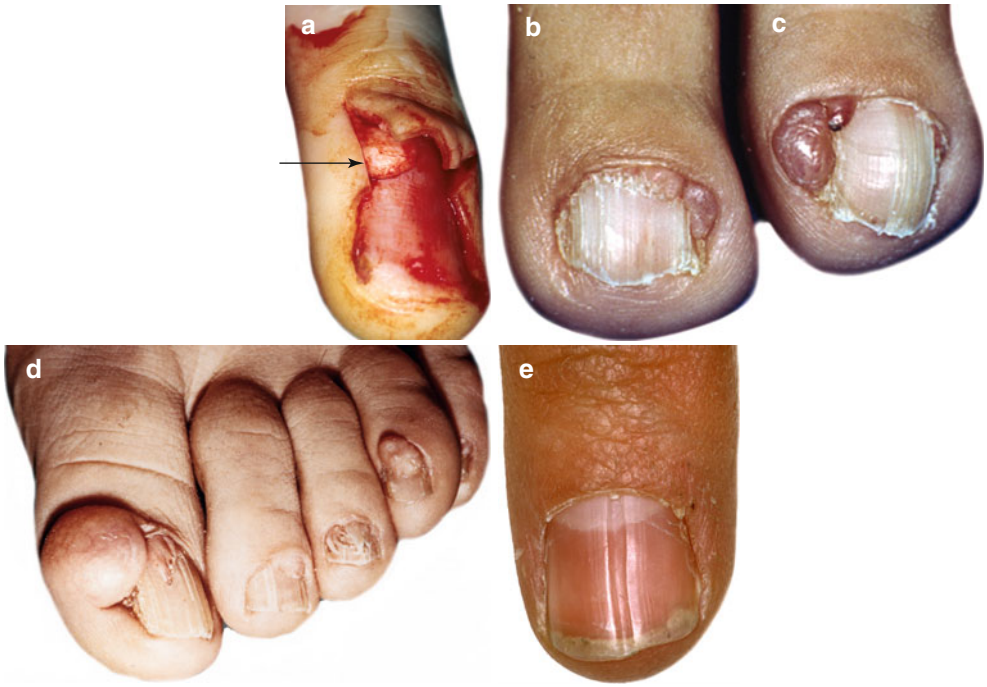

Fig. 17.4 (**a**) Fibrokeratoma (*arrow*) (**b**–**d**) Koenen's tumors (**e**) Koenen's tumor with longitudinal groove demonstrating presence of a very tiny tumor in front of the cuticle

structure are reminiscent of a fibrokeratoma. However, a diagnosis of fibrokeratoma of the nail matrix can be excluded on the basis of the multiplicity of fibroepithelial digitations, absence of a horny corn at the distal border of the thickened nail plate, and the presence of cavitation filled with serous fluid. In the second, the stroma of the lunular segment of the onychomatricoma can suggest a fibroma. However, the latter can be ruled out on the basis of the hyperplastic and onychogenic nature of the epithelium. Histologically, an ungual fibroma compresses the matrix epithelium and results clinically in thinning of the nail plate in the form of a longitudinal groove [18]. This type of tumor can bleed after extensive trimming of the distal nail margin.

On sonography, onychomatricoma presents with an eccentric location in the nail bed and affects one of the matrix wings. Hyperechoic linear dots are described within the hyperechoic tumor that also sends projections into the intraplate space and matrix region. Remodeling or erosive changes in the bony margin, hypervascularity, or expansion in the PNF have been reported.

17.5 Fibrous Tumors

17.5.1 Acquired Ungual Fibrokeratoma

Acquired ungual fibrokeratoma (Fig. 17.4) is probably a variant of acquired digital fibrokeratoma [20] and garlic clove fibroma [21]. Classically, they are acquired, benign, spontaneously developing, asymptomatic nodules with a hyperkeratotic tip and a narrow base that occur mostly in the periungual area or elsewhere on the fingers. They can be double and even triple and reach a considerable size. Most ungual fibrokeratomas emerge from beneath the PNF, growing on the dorsum of the nail where it causes a sharp longitudinal depression. Some of the lesions originate from within the matrix and grow in the nail plate to eventually emerge in the middle of the nail; these intraungual fibrokeratomas are also called "dissecting ungual fibrokeratomas" because they divide the nail plate [22]. Subungual fibrokeratomas that arise from the nail bed are rare.

17.5.2 Acquired Ungual Fibrokeratoma and Koenen's Tumors

Koenen's periungual fibromas develop in about 50 % of cases of tuberous sclerosis (epiloia or Bourneville-Pringle disease) (Fig. 17.4a, b, c), which is a dominantly inherited multisystem disease affecting the central nervous system, eyes, skin, cutaneous appendages, kidneys, heart, blood vessels, and bones. Two major gene loci have been identified where mutations can cause the tuberous sclerosis complex with apparently indistinguishable phenotypes: *TSC1* at 9q34 and *TSC2* at 16p13.3 [23].

In the Koenen's tumors that we have examined, we were able to distinguish two portions: (1) a small distal segment with loose collagen and many blood vessels, and (2) a larger proximal part built up of dense collagen bundles and fewer capillaries [24]. It thus appears that Koenen's tumor can be considered as a particular type of fibrokeratoma that can be

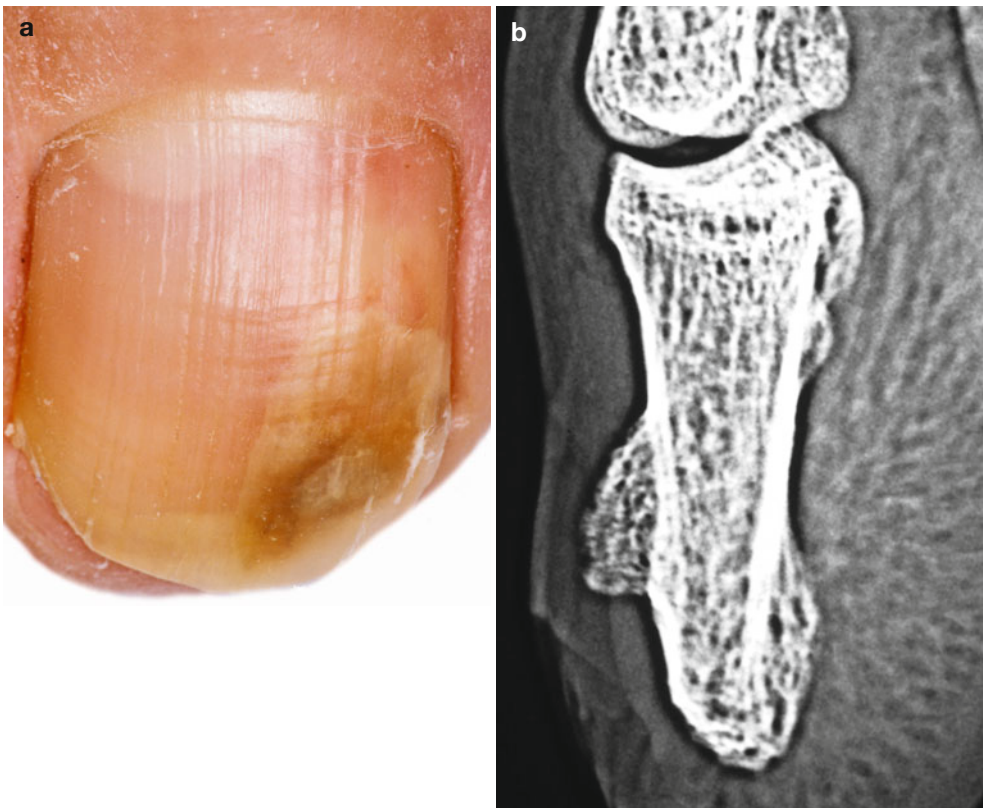

Fig. 17.5 (**a**) Distal subungual exostosis. (**b**) Exostosis radiograph

subdivided according to its clinical appearance, its location, and its origin into the following groups:

1. Fibrokeratomas originating from the dermal connective tissue. These fibrokeratomas are post-traumatic or appear spontaneously and are usually located on the fingers (acquired digital fibrokeratoma).
2. Fibrokeratomas originating from the PNF or the surrounding connective tissue. These fibrokeratomas are located in the nail fold [25] and can be hereditary [26] (tuberous sclerosis) or acquired (for example, garlic-clove fibroma).

Trauma is thought to be a major factor initiating acquired periungual fibrokeratoma. Biopsy is mandatory for the diagnosis of nail tumors because pseudo-fibrokeratoma should be considered as a clue for Bowen's disease [27].

On sonography, fibromas present uniform hypoechoic nodular or oval structures. Their location is commonly eccentric within the nail bed and they can affect the matrix region including its wings. They can present in a variety of sizes and usually, in large sized tumors, a remodeling of the bony margin can be detected.

Moreover, fibrous tumors can secondarily involve the lateral nail fold going from the dorsal aspect to the ventral aspect and attaching into the corresponding flexor sheath. On color Doppler ultrasound, fibrous tumors are usually hypovascular, with the exception of angiofibroma that can present with small sized vascular bundles with low velocity arterial and venous blood flow within the tumoral lesion.

17.6 Exostosis and Osteochondroma

Subungual exostoses are not true tumors, but rather outgrowths of normal bone or calcified cartilaginous remains. It is still unclear whether subungual osteochondroma [28] is a different entity [29] Subungual exostoses are rarely reported: there were only 60 subungual exostoses in a series of 6,034 benign osseous lesions [30]; however, they can be considerably under-diagnosed and underreported. They are bony growths that are painful with pressure and can elevate the nail. They are particularly frequent in young people and located mostly in the dorso-medial aspect of the tip of the great toe, although subungual exostoses can also occur in the lesser toes or less commonly in the thumb or index fingers [31].

Subungual exostoses start as small elevations of the dorsal aspect of the distal phalanx and can eventually emerge from under the nail edge or destroy the nail plate. If the nail is lost, the surface becomes eroded and secondarily infected, sometimes mimicking an ingrown toenail or even a melanoma, which can cause walking to be painful; however, solitary subungual exostoses have never been observed as undergoing malignant degeneration. Some authors believe that a history of trauma is only occasionally found in subungual exostosis [32].

The triad of pain (the leading symptom), nail deformation (Fig. 17.5a), and radiographic features (Fig. 17.5b) are usually diagnostic. The exostosis is an ill-defined trabeculated osseous growth with an expanded distal portion covered with radiolucent fibrocartilage.

Osteochondroma, commonly evoking the same symptoms as subungual exostosis, is believed to have a male predominance. Its onset is usually between the ages of 10–25 years, and there is also often a history of trauma [28]. Its growth rate is slow and radiographs shows a well-defined circumscribed pedunculated or sessile bone growth projecting from the dorsum of the distal phalanx near to the epiphyseal line, therefore, nail dystrophy can be pronounced [33]. A bony tumor with a hyaline cartilage cap can be seen histologically, however, on the basis of the histopathological pattern, de Palma et al. [29] have stated that most subungual bone masses exhibit the features of conventional osteochondromas and not of subungual exostoses independent of their location at the distal phalanx. It must be differentiated from primary subungual calcification (particularly seen in elderly women), secondary subungual calcification due to trauma or psoriasis [34], and primary osteoma cutis [35].

Subungual exostoses appear on ultrasound as linear echoic structures with posterior shadowing artifact, frequently seen and dependent on the reflective properties of the calcium component. These exostoses usually connect with their origin in the hyperechoic line of the bony margin of the distal phalanx. When they are associated with cartilaginous tissue, they are called osteochondroma, and a hypoechoic cap can be detected surrounding the calcified hyperechoic component. Hypoechoic ill-defined tissue can also be seen in the periphery of the exostoses as a part of secondary inflammatory and scarring reaction.

17.7 Myxoid Cysts

Myxoid cysts are cystic lesions that are usually connected to the distal interphalangeal (DIP) joint and extend into the periungual or subungual regions. They most commonly affect the PNF but also may extend into the nail bed secondarily compressing the ungual matrix [36] (Fig. 17.6).

They appear on ultrasound as round or oval-shaped anechoic structures that produce posterior acoustic reinforcement and lack of inner blood flow. Extension of the mucoid cyst into the nail bed and the usually tortuous anechoic connecting tract with the DIP joint can be assessed. They are commonly associated with osteoarthritis and synovitis of the DIP joint, and thickening and irregularities of the nail plate can be detected in the same axis of the ungual compression.

17.8 Pyogenic Granuloma

Pyogenic granuloma is a benign eruptive hemangioma which typically occurs following a minor penetrating skin injury. It begins around the nail with a minute red papule that rapidly grows to the size of a pea or even a cherry. Its surface can become eroded with necrosis of the overlying epidermis. Crusting can mimic a malignant melanoma, although the typical collarette can usually be seen. Pyogenic granuloma is most commonly located at the PNF but can develop distally in the hyponychium region (Fig. 17.7) or in the nail bed associated with onycholysis [37] of the toe, often resulting from prolonged frictional trauma. The matrix can also be the site of this tumor after a penetrating wound of the nail plate, with tenderness and a ready tendency to bleed being characteristic features. Extensive granulation tissue resulting from an ingrown toenail can mimic a periungual pyogenic granuloma, and has also been observed in patients treated with aromatic retinoids [38], indinavir [39], cyclosporine [40], as well as with the new anti-cancer agents that act by inhibiting the epidermal growth factor receptor or

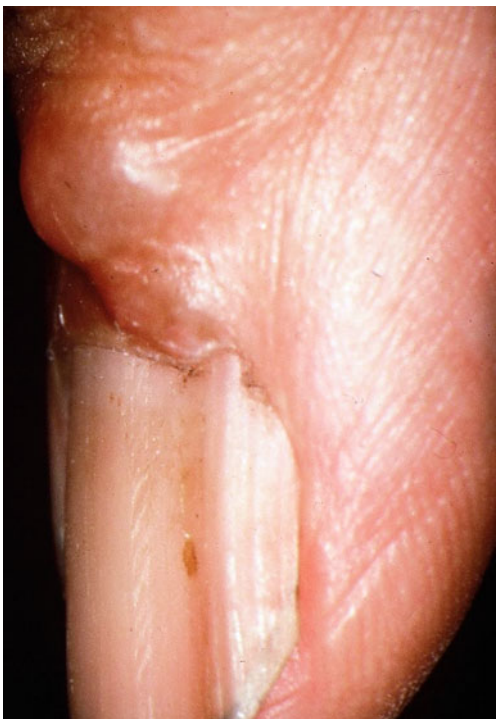

Fig. 17.6 Myxoid pseudocysts

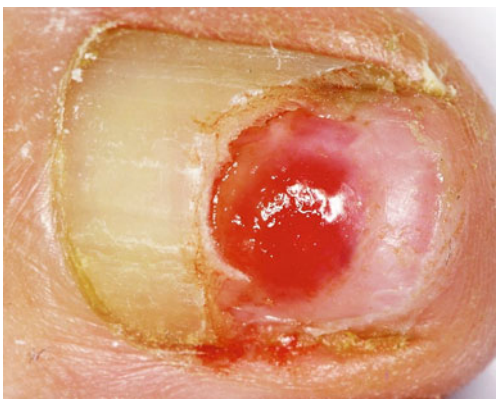

Fig. 17.7 Pyogenic granuloma

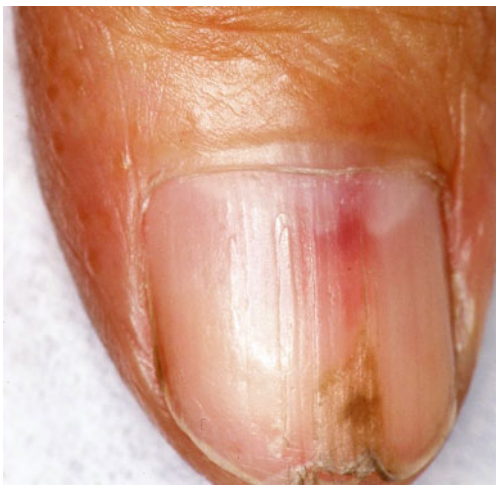

Fig. 17.8 Glomus tumor with longitudinal erythronychia

its transduction: Iressa (ZD1839) [41], Cetuximab (C225) [42], and Gefitinib [43].

They appear on ultrasound as poorly defined hypoechoic structures that displace the nail plate and enlarge the nail bed. One or both nail layers of the nail plate can be thickened and/or wavy in shape, and variable degrees of blood flow can be detected within granulomas going from hypovascularity to hypervascularity (telangieltatic variant).

17.9 Glomus Tumor

Seventy-five percent of glomus tumors occur in the hand, most commonly in the fingertips and particularly in the subungual area. One to two percent of all hand tumors are glomus tumors [44], with seven cases of glomus tumors being reported in von Recklinghausen's neurofibromatosis [45].

Glomus tumors are characterized by intense, often pulsating, pain that can occur spontaneously or be provoked by the slightest trauma and exacerbation (such as placing an ice cube on the nail), and even changes in temperature, particularly from warm to cold, can trigger pain radiating up to the shoulder. The pain can sometimes be worse at night, however, a tourniquet placed at the base of the digit stops the pain, with a blood pressure cuff inflated to 300 mmHg before or immediately after minor trauma abolishing the pain response [46].

Glomus tumors are seen through the nail plate as small, bluish to reddish-blue spots several millimeters in diameter, rarely exceeding 1 cm in diameter. An erythematous focus that does not totally blanch with pressure, and is associated with sharp pain, probably represents a glomus tumor. Longitudinal erythronychia that can be associated with a distal fissure is a classic presentation of a glomus tumor (Fig. 17.8). A glomus tumor can sometimes cause a slight rise in surface temperature which can be detected by thermography; dynamic telethermography shows the lesion at

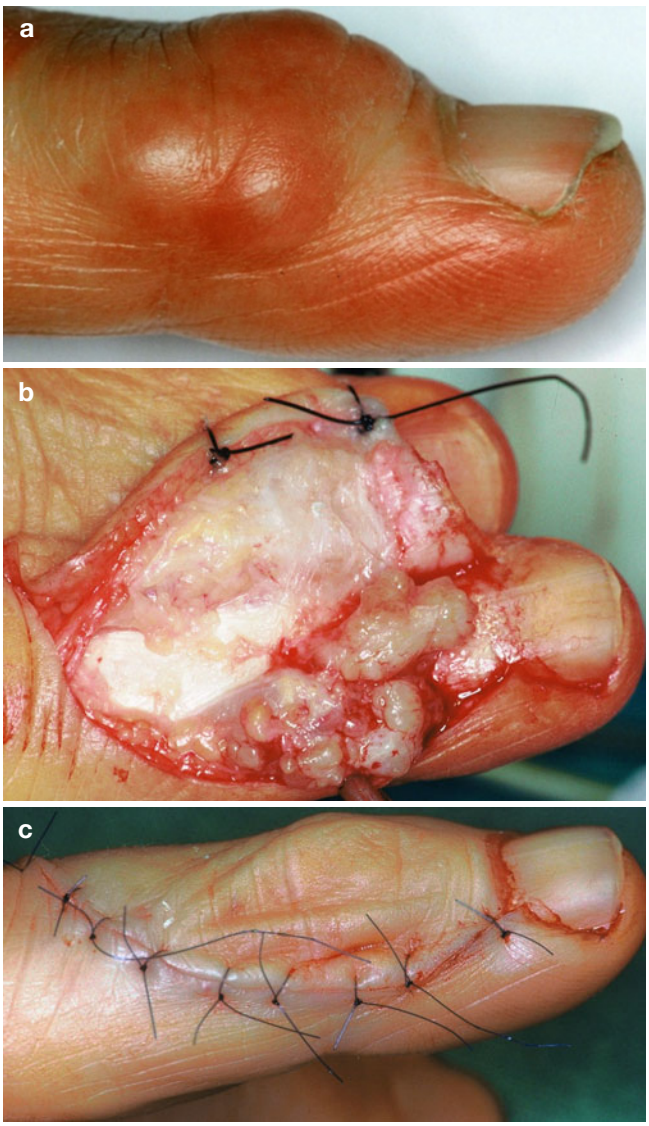

Fig. 17.9 (**a**) Giant cell tumor. (**b**) Exposed lesions. (**c**) After removal of the tumor

about three times its actual size [47]. One half of glomus tumors cause minor nail deformities with ridging and fissuring being the most common. Subungual hyperkeratosis with onycholysis is rare. About 50 % cause a depression on the dorsal aspect of the distal phalangeal bone or even a cyst visible on radiographic study [48], and an intraosseous location is unusual. Probing and transillumination may help to localize the tumor if it is not clearly visible through the nail as arteriography is no longer performed; this would reveal a star-shaped telangiectatic zone useful for diagnosis and localization of the tumor if it cannot be localized clinically or on radiography, although magnetic resonance imaging may be preferable because it offers the highest sensitivity and a better assessment of the extent of the lesion [49].

Glomus tumors appear as hypoechoic nodules on ultrasound, centrally located and frequently with increased vascularity. Variable peak arterial systolic velocities can be detected

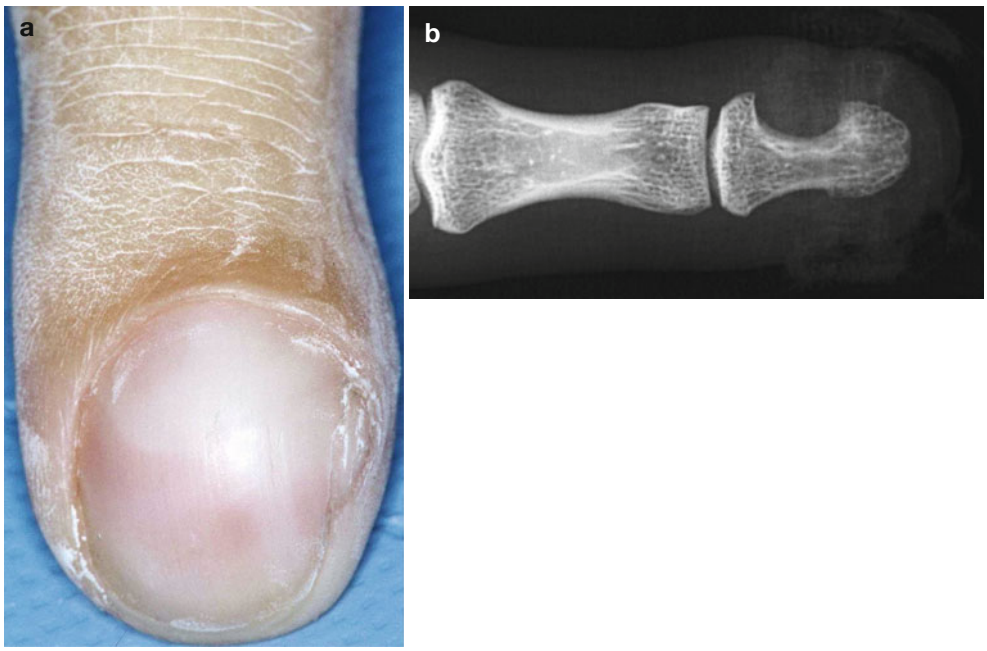

Fig. 17.10 (**a**) Perineurioma. (**b**) Perineurioma radiograph

within the intratumoral blood flow, and have been reported to be as low as 3.7 cm/s and as high as 21.1 cm/s, the latter exceeding the peak systolic velocity that had been described for the normal tibial artery (16 ± 10 cm/s). It is common to see remodeling of the bony margin of the distal phalanx beneath the tumor, probably reflecting the slow growth pattern of the tumor. On ultrasound, proximal locations of glomus tumors are described as more frequent than distal locations; therefore, pre-surgical knowledge of the location of the tumor can benefit the choice of the incision site.

17.10 Giant Cell Tumor

Giant cell tumors are neoplasms derived from the tendon sheath or the joint synovia. They are the second most common subcutaneous tumors of the hand and occur more frequently in women than they do in men. On the digits, giant cell tumors usually occur on the dorsum of the DIP joint and appear as solitary, often lobulated slow-growing, skin-colored and smooth-surfaced nodules that tend to feel firm and rubbery (Fig. 17.9a–c). The tumors may enlarge to the size of a cherry and may cause pain on flexion by virtue of their dimensions. Only rarely do the tumors interfere with the nail unit in the region of the lateral nail fold. Periodic inflammation and drainage may occur, but in contrast to malignant synovioma, no calcification is demonstrable on radiographs [50], and giant cell tumors in a subungual location are unusual [51]. A cystic-appearing lesion adjacent to the nail can cause a wide longitudinal groove in the nail plate [52], and a giant cell tumor involving the lateral nail fold and nail bed can interfere with nail growth [53].

17.11 Perineurioma of the Nail

Perineuriomas can be distinguished by their positive immunoreactivity for epithelial membrane antigen and lack of reactivity to the S-100 protein and α-smooth muscle actin. They appear clinically as swelling, clubbing (Fig. 17.10a, b), or dystrophy of the nail and can mimic other tumoral entities such as fibrous tumors or subungual exostoses.

Poorly defined hypoechoic eccentric masses have been reported when using ultrasound. Perineuriomas can involve the matrix region (particularly one of the wings) and the ipsilateral nail fold, presenting on color Doppler ultrasound as a hypovascular tumors. Remodeling of the bony margin has not been described, although this finding could be hypothetically present in larger size tumors. To our knowledge, neither involvement of the inter-plate space nor hyperechoic dots within the perineuriomas have been reported to date. Some authors were fortunate enough to see three cases of perineurioma in the nail apparatus [54–56]; however, these benign tumors, derived from neural tissue, are extremely rare in this location. Additionally, they differ from other common neurogenic tumors such as schwanomas or neurofibromas.

References

1. Holland TT, Weber CB, James WD. Tender periungual nodules. Arch Dermatol. 1992;128:105–10.
2. Laüchli S, Eichmann A, Baran R. Swelling of the proximal nail fold caused by underlying warts. Dermatology. 2001;202:328–9.
3. Baran R, Goettmann S. Distal digital keratoacanthoma: a report of 12 cases and review of the literature. Br J Dermatol. 1998;139:512–5.
4. Baran R, Mikhail G, Costini B, Tosti A, Goettmann S. Distal digital keratoacanthoma. Two cases with a review of the literature. Dermatol Surg. 2001;27:575–9.

5. Haneke E. Multiple subungual keratoacanthomas. XII Int cong dermatol surg Munich. Zbl Haut-Geschl Kr. 1991;159:337–8.
6. Sinha A, Marsh R, Langtry J. Spontaneous regression of subungual keratoacanthoma with reossification of underlying distal lytic phalanx. Clin Exp Dermatol. 2005;30:20–2.
7. Baran R, Kint A. Surgical treatment of a filamentous tufted tumour in the matrix of a funnel-shaped nail – a new entity. Zbl Haut-Geschl Kr. 1991;159:337.
8. Baran R, Kint A. Onychomatricoma. Filamentous tufted tumour in the matrix of a funnel-shaped nail: a new entity. Br J Dermatol. 1992;126:510–5.
9. Van Holder C, Dumontier C, Abimelec P. Onychomatricoma. J Hand Surg. 1999;24B:120–1.
10. Fraga GR, Patterson JW, McHargue CA. Onychomatricoma. Report of a case and its comparison with fibrokeratoma of the nail bed. Am J Dermatopathol. 2001;23:36–40.
11. Nagamatsu S, Tozawa A, Nakuoka H, Ohtsuka H, Hashimoto K. A case of onychomatricoma. JPN J Plast Reconstr Surg. 2002;45:875–80.
12. Laxmisha C, Thappa DM, Karthikeyan K, Jayanthi S. Onychomatricoma. Indian J Dermatol. 2003;48:94–5.
13. Ko CJ, Shi L, Barr RJ, Mölne L, Termesten-Bradel A, Headington JT. Ungioblastoma und unguioblastic fibroma – an expanded spectrum of onychomatricoma. J Cutan Pathol. 2004; 31:207–311.
14. Raison-Peyron N, Alirezai M, Meunier L, Barnéon G, Meynadier J. Onychomatricoma: an unusual cause of nail bleeding. Clin Exp Dermatol. 1998;23:138.
15. Perrin C, Goettmann S, Baran R. Onychomatricoma: clinical and histopathologic findings in 12 cases. J Am Acad Dermatol. 1998;39:560–4.
16. Fayol J, Baran R, Perrin C, Labrousse F. Onychomatricoma with misleading features. Acta Dermato Venereol. 2000;80:370–2.
17. Goettmann S, Drapé JL, Baran R, Perrin C, Haneke E, Bélaïch S. Onychomatricome: 3 nouveaux cas, intérêt de la résonance magnétique nucléaire. Ann Dermatol Venereol. 1994;121:S145.
18. Haneke E, Fränken J. Onychomatricoma. Dermatol Surg. 1995;21:984–7.
19. Kint A, Baran R, Geerts ML. The onychomatricoma: an electron microscopic study. J Cutan Pathol. 1997;24:183–8.
20. Bart RS, Andrade R, Kopf AW, Leider M. Acquired digital fibrokeratomas. Arch Dermatol. 1968;97:120–9.
21. Steel HH. Garlic-clove fibroma. J Am Med Assoc. 1965;191:1082–3.
22. Haneke E. Intraoperative differential diagnosis of onychomatricoma, Koenen's tumours, and hyperplastic Bowen's disease. J Eur Acad Dermatol Venereol. 1998;11:S 199.
23. Sampson JR, Harris PC. The molecular genetics of tuberous sclerosis. Hum Mol Genet. 1994;3:1477–80.
24. Kint A, Baran R. Histopathologic study of Koenen tumours. Are they different from acquired digital fibrokeratoma? J Am Acad Dermatol. 1988;18:369–72.
25. Colomb D, Racouchot J, Jeune R. Les lésions des ongles dans la sclérose tubéreuse de Bourneville isolées ou associées aux tumeurs de koenen. Ann Dermatol Syph. 1976;103:431–7.
26. Webb DW, Clarke A, Fryer A, Osborne JP. The cutaneous features of tuberous sclerosis: a population study. Br J Dermatol. 1996;135:1–5.
27. Baran R, Perrin C. Pseudo-fibrokeratoma of the nail apparatus: a new clue for Bowen disease. Acta Derm Venereol. 1994;74:449–50.
28. Apfelberg DB, Druker D, Maser M, Lash H. Subungual osteochondroma. Differential diagnosis and treatment. Arch Dermatol. 1979;115:472–3.
29. De Palma L, Gigante A, Specchia N. Subungual exostosis of the foot. Foot Ankle Int. 1996;17:758–63.
30. Dahlin DC, Unni KK. Bone tumors. In: Dahlin DC, Unni KK eds, Dahlin's Bone tumors, 4th ed. Springfield: Charles C Thomas; 1986. p. 18–30.
31. Baran R, Sayag J. Exostose sous-unguéale de l'index. Ann Dermatol Venereol. 1978;105:1075–6.
32. Norton LA. Nail disorders. J Am Acad Dermatol. 1980;2:451–67.
33. Kim SW, Moon SE, Kim JA. A case of subungual osteochondroma. J Dermatol. 1998;25:60–2.
34. Fischer E. Subunguale verkalkungen. Fortschr Röntgenst. 1982;137:580–4.
35. Burgdorf W, Nasemann T. Cutaneous osteomas: a clinical and histopathologic review. Arch Dermatol Res. 1977;260:121–35.
36. Wortsman X, Jemec GB. Ultrasound imaging of nails. Dermatol Clin. 2006;24:323–8.
37. Richert B. Frictional pyogenic granuloma of the nail bed. Dermatology. 2001;202:80–1.
38. Baran R. Retinoids and the nails. J Dermatol Treat. 1990;1:151–4.
39. Bouscarat F, Bouchard C, Bachour D. Paronychia and pyogenic granuloma of the great toes in patients treated with indinavir. VEJ Med. 1998;338:1776–7.
40. Higgins EM, Hughes JR, Snowden S, Pembroke AC. Cyclosporin-induced periungual granulation tissue. Br J Dermatol. 1995;132:829–30.
41. Lee MW, Seo CW, Kim SW, et al. Cutaneous side effects in non-small cell lung cancer patients treated with iressa (ZD1839), an inhibitor of epidermal growth factor. Acta Derm Venereol. 2004;84:23–6.
42. Busam KJ, Capodieci P, Motzer R, Kiehn T, Phelan D, Halpern AC. Cutaneous side-effects in cancer patients treated with the antiepidermal growth factor receptor antibody C225. Br J Dermatol. 2001;144:1169–76.
43. Nakano J, Nakamura M. Paronychia induced by gefitinib, an epidermal growth factor receptor tyrosine kinase inhibitor. J Dermatol. 2003;30:261–2.
44. Rettig AC, Strickland JW. Glomus tumor of the digits. J Hand Surg. 1977;2:261–5.
45. Sawada S, Honda M, Kamide R, Niimura M. Three cases of subungual glomus tumors with von Recklinghausen neurofibromatosis. J Am Acad Dermatol. 1995;32:277–8.
46. Hildreth DH. The ischemia test for glomus tumor: a new diagnostic test. Rev Surg. 1970;27:147–8.
47. Corrado EM, Passareti U, Messore L, Lanza F. Thermographic diagnosis of glomus tumour. Hand. 1982;14:21–4.
48. Van Geertruyden J, Lorea P, Goldschmidt D, de Fontaine S, Schuind F, Kinnen L, et al. Glomus tumours of the hand. A retrospective study of 51 cases. J Hand Surg. 1996;21:257–60.
49. Van Ruyssevelt CE, Vranckx P. Subungual glomus tumor: emphasis of MR angiography. AJR Am J Roentgenol. 2004;182:263–4.
50. Wright CJE. Benign giant-cell synovioma. An investigation of 85 cases. Br J Surg. 1951;38:257–71.
51. Abimelec P, Cambiaghi S, Thioly D, Moulonguet I, Dumontier C. Subungual giant cell tumor of the tendon sheath. Cutis. 1996;58:273–5.
52. Batta K, Tan CY, Colloby P. Giant cell tumour of the tendon sheath producing a groove deformity of the nail plate and mimicking a myxoid cyst. Br J Dermatol. 1999;140:720–81.
53. Richert B, André J. Latero-subungual giant cell tumor of the tendon sheath: an unusual location. J Am Acad Dermatol. 1999; 41:347–8.
54. Baran R, Perrin C. Subungual perineurioma: a peculiar location. Br J Dermatol. 2002;164:125–8.
55. Baran R, Perrin C. Perineurioma: a tendon sheath fibroma-like variant in a distal subungual location. Acta Derm Venereol. 2003;83:60–1.
56. Wortsman X, Merino D, Catalan V, Morales C, Baran R. Perineurioma of the nail on sonography. J Ultrasound Med. 2010;29:1379–82.