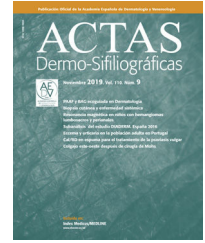




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PRACTICAL DERMATOLOGY

[Translated article] Acne Keloidalis Nuchae: A Therapeutic Challenge for Dermatologists

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KEYWORDS

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Tufted hairs

PALABRAS CLAVE

Folliculitis nuchae
scleroticans;
Alopecia cicatricial;
Pérdida de Cabello;
Pelos en penacho

Abstract Acne keloidalis nuchae (AKN) is a chronic inflammatory dermatosis of the scalp that causes scarring alopecia. The etiology of AKN has been associated with an immune response vs. follicular antigens. AKN is characterized by fibrotic papules and plaques that can converge in tumor-like lesions; subclinical disease has been reported in perilesional areas, which could impact its development. Early diagnosis and treatment are essential to reduce morbidity and preserve and minimize healing. Various treatments have been established including optimal medical therapy, surgical excision, and light sources. An updated description of treatments (algorithm included) used for AKN is suggested based on the clinical lesions.

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Acné queiloideo de la nuca, un reto terapéutico para los dermatólogos

Resumen El acné queiloideo de la nuca es una dermatosis inflamatoria crónica del cuero cabelludo que ocasiona alopecia cicatricial. Su etiología se ha asociado a una respuesta inmune contra antígenos foliculares. Se caracteriza por pápulas y placas fibróticas que pueden confluir en lesiones de aspecto tumoral. Se ha observado enfermedad subclínica en áreas perilesionales que podría influir en su desarrollo. Es fundamental un diagnóstico y tratamiento precoz para disminuir la morbilidad y preservar y minimizar la cicatrización. Se han establecido

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diversos tratamientos que incluyen terapia médica, escisión quirúrgica y fuentes de luz. Se realiza una descripción actualizada de los tratamientos empleados y se propone un algoritmo de tratamiento con base en las lesiones clínicas.

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Introduction

Acne keloidalis nuchae (AKN) is a chronic inflammatory dermatosis of the hair follicle in the occipital and posterior cervical region that results in scarring alopecia. It predominantly affects men, with a 20:1 ratio, particularly those of African descent and with darker skin phototypes.¹⁻³

Etiopathogenesis

The etiology of AKN has been associated with an uncontrolled immune response to follicular antigens that destroys the hair follicle and sebaceous gland, leading to chronic inflammation and fibrosis. Risk factors include increased sensitivity to androgens, seborrhea, curly hair, irritation from friction or shaving (causing ingrown hairs), and certain drugs (i.e., cyclosporine or lithium).^{4,5} However, the exact mechanism of its pathophysiology remains unknown.¹⁻⁶

Comorbidities

Dermatoses directly associated with AKN include hidradenitis suppurativa, pseudofolliculitis barbae, and scalp folliculitis. Pseudofolliculitis barbae has been associated with the growth of keloid scarring plaques, while scalp folliculitis and metabolic syndrome are associated with greater lesion extent.^{7,8}

AKN has a strong association as a cutaneous sign of metabolic syndrome.⁹⁻¹¹ Other conditions coexisting with AKN include acne (14.29%), folliculitis decalvans (20.25-37%), dissecting cellulitis (7%), and pilonidal sinus (1.3%).⁷⁻¹²

Clinical presentation, trichoscopy, and histology

Clinically, AKN is characterized by papules, pustules, and inflammatory, fibrotic, pruritic plaques (Figs. 1 and 2). The chronic and recurrent nature of these lesions leads to keloidal scarring alopecia in individuals without a predisposition to keloid scarring in other areas of the body. Dermoscopy reveals tufted hairs centered in papules, pustules, and crusts on an elevated scarring alopecia plaque (Figs. 3 and 4).^{1,2} Early histological findings show a neutrophilic infiltrate in the isthmus and sebaceous glands, progressing to thick collagen bands and a mixed lymphoplasmacytic and inflammatory infiltrate around the infundibulum.³

Trichoscopy reveals areas of erythema and perilesional scaling, considered subclinical disease markers potentially



Figure 1 Initial stage of AKN showing fibrotic lesions with a papular and nodular appearance centered on follicles in the nuchal area.

influencing AKN growth. Histologically, these areas correspond to lymphoplasmacytic infundibulo-isthmic infiltrates and fibrosis.¹³

AKN severity is classified using a 3-level system that allows for clinical description of lesions and treatment outcome evaluation (Table 1).¹⁴

Treatment

Treating AKN poses a therapeutic challenge for dermatologists. Early intervention is crucial to prevent plaques and tumor-like lesions. General recommendations include avoiding frequent shaving, reducing friction, and using antiseptic cleansers to prevent secondary infection.¹⁻¹⁵

Currently, various treatments aim to reduce inflammation and fibrosis, including optimal medical therapy, surgical



Figure 2 Papules, pustules, and inflammatory nodules; in the sagittal region of the nuchal area, these merge to form a plaque with a keloid scar-like appearance surrounding tufted hairs.

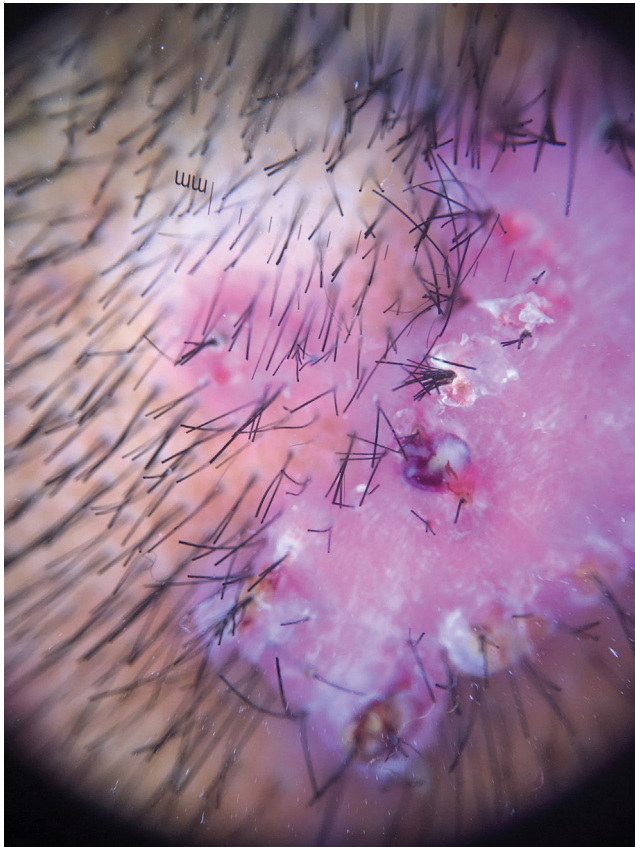


Figure 3 Trichoscopy showing tufted hairs centered on papules and pustules that converge into a scarring alopecia plaque.



Figure 4 Trichoscopy showing tufted hairs centered on fibrotic-looking papules arranged in clusters.

excision, and light-based interventions.³ However, no specific therapeutic algorithm exists.

Treatment options for AKN are approached practically based on an objective assessment of the clinical stage (Table 2).

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Table 1 Classification system for AKN in 3 levels.

Level I (primary distribution) ^a		Level II (secondary) type of lesion	Level III (tertiary) scalp condition
<i>Class</i>			
I. Sagittal width < 3 cm within demarcation lines	Result treatment ^b No likelihood of wide scarring (scar < 0.5 cm).	Discrete papules or nodules	Positive associated disease
II. Sagittal width 3–6.5 cm within demarcation lines	<55% probability vs class III (scar 0.5–2.5 cm).	Fused papules or nodules	
III. Sagittal width > 6.5 cm or breaches demarcation lines but limited to the nuchal area	>7 times probability vs class II (scar >2.5 cm or dehiscence).	Plaques	Negative associated disease DC and FD
IV. Exceeds the nuchal area		Tumoral masses ^c	

Source: Adapted from Umar et al.¹⁴

DC: dissecting cellulitis; FD: folliculitis decalvans.

^a Distribution of predominant lesions relative to an area defined by the occipital prominence and the inferior tips of the mastoid processes.

^b Probability of scar stretching, hypertrophic scarring, or wound dehiscence.

^c Tumoral masses present in 22% of Class II and 42% of Class III cases. Distribution: Lesions are predominant relative to the area defined by the occipital prominence and the lower tips.

Table 2 Treatment of reported AKN based on the type of lesions.

Type of lesion	Proposed treatment
Fibrotic papules	Topical corticosteroids (clobetasol propionate 0.05%, betamethasone valerate 0.12%) Intralesional corticosteroids (triamcinolone acetonide 2.5–40 mg/mL) Imiquimod 5% cream ± pimecrolimus 1% cream Excision of affected follicular units (FUE) Laser therapy ^c
Fibrotic plaques ^a	Laser therapy ^c Cryotherapy
Thick fibrotic plaques, tumor masses, or refractory to treatment	Surgical excision CO ₂ laser excision or electrosurgery Radiotherapy
Bacterial superinfection	Clindamycin 1% for mild cases Oral tetracyclines (minocycline, doxycycline) for more severe cases
Active or inflammatory phases ^b	Topical retinoids Oral retinoids

^a Fibrotic plaques up to 3 mm thick (from the skin to the surface).

^b Refractory lesions treated with antibiotics.

^c Long-pulse and long-wavelength laser therapy: Alexandrite 755 nm, diode 810 nm, ND-YAD 1064 nm, Er:YAG 2490 nm.

Optimal medical therapy

Topical corticosteroids

Topical and intralesional corticosteroids are the most widely used treatment modalities. A few cases have been reported using oral corticosteroids.¹²

The study by Callender described the efficacy of high-potency corticosteroids, such as 0.05% clobetasol propionate and 0.12% betamethasone valerate. An 8-week regimen of clobetasol applied every 12 h for 2 weeks, alternating with 2 weeks off, resulted in a 60% reduction in papulopustular lesions and decreased symptoms such as burning, itching, or pain. A mild rebound occurred within the first 2-week cycle. No significant difference was observed when using

betamethasone every 12 h for 4 week.^{1,3,15,16} However, there is limited literature supporting its use.

Intralesional corticosteroids

Ogunbiyi and Brahe described the use of triamcinolone acetonide at concentrations ranging from 2.5–5 mg/mL up to 40 mg/mL. Concentrations > 20 mg/mL are recommended for lesions > 3 cm. This treatment is indicated for mild-to-moderate lesions, administered every 4 weeks. Dilution reduces the risk of atrophy, and combining it with lidocaine improves patient comfort.^{1,3,15}

Adotama et al. suggested injecting triamcinolone into the active area with a 1 cm margin to include subclinical affected areas (0.1 mL of triamcinolone at 5–10 mg/mL

concentrations with 1 cm intervals). This technique improved keloid lesions within 7 weeks and may help prevent lesion development compared with intralesional injection alone. However, side effects such as atrophy, telangiectasias, and hypopigmentation need further monitoring.¹⁷

Antibiotics

Ogunbiyi has described antibiotic use for superinfections, noting their antimicrobial and anti-inflammatory effects.

Topical antibiotics are considered for mild pustular superinfections. Clindamycin 1% is the most widely used, while other options include erythromycin 2%, benzoyl peroxide, and fusidic acid. These can be combined with steroids or topical retinoids, potentially following acne treatment guidelines.^{1,18}

Oral antibiotics have been reported for moderate-to-severe superinfections. Evidence supports the use of tetracycline, doxycycline, minocycline, trimethoprim-sulfamethoxazole, erythromycin, azithromycin, amoxicillin, and cephalexin.

Tetracyclines are considered the first-line therapy for their anti-inflammatory properties, such as doxycycline at doses of 50–100 mg/day twice daily for 12–16 weeks.¹⁸ Rifampin, clindamycin, and dapsone have also been used to control superinfections.¹² These can be combined with topical and intralesional corticosteroids.^{1,3,15}

Retinoids

Retinoid use is likely based on their efficacy in treating acne vulgaris, given their comedolytic (topical), anti-inflammatory, and follicular occlusion-preventing effects.^{1,3,15}

Topical retinoids include tretinoin 0.025–0.1%, adapalene 0.1–0.3%, and tazarotene 0.05%. Their efficacy depends on concentration, with reported success rates ranging from 35% to 80% in treatments lasting 12–16 weeks.^{1,3,15,18,19}

Oral isotretinoin has been used at doses of 0.25 mg/kg per day, showing marked improvement at 6 months. It is recommended when there is no response to treatment with tetracyclines and topical or intralesional corticosteroids.³

Isotretinoin is typically initiated at doses of 0.5–1 mg/kg per day, depending on patient tolerance during active phases, and maintained at doses of 0.25–0.4 mg/kg per day. The total cumulative dose is 120–150 mg/kg, with recurrence rates up to 65%. In severe cases, it may be used alongside oral corticosteroids.^{1,3,15,18,20}

Imiquimod

The mechanism of action of imiquimod is related to the stimulation of inflammatory cytokines, inhibition of angiogenesis, and activation of apoptosis, leading to the death of fibroblasts.

Figueroa and Lorenz reported cases in which daily use of imiquimod resulted in clinical improvement of fibrotic papular lesions after 8 weeks.²¹ In another study, Barr et al. demonstrated the efficacy of imiquimod combined

with pimecrolimus over 8 weeks, with no subsequent recurrence.²²

Laser therapy

The mechanism of action of laser therapy involves the destruction of hair follicles within lesions. It is limited to folliculocentric papular lesions, isolated discrete nodules, or plaques with a vertical height of up to 2–3 mm, ensuring greater penetration depth corresponding to hair follicle length.

Umar describes the use of pulsed and long-wave lasers, including 755 nm alexandrite, 800 nm diode, and Nd:YAG. The latter penetrates deeper (5–7 mm) compared with others and is less absorbed by melanin, causing less damage in darker phototypes. Hair follicles are a potential source of new AKN lesions, and their treatment reduces disease progression.²³

Alopecia is an expected effect of laser therapy in AKN. Umar proposed a severity-based classification system to predict the area of posterior hairline displacement. The classification ranges from Level I up to IV based on the extent of AKN lesion spread in relation to the occipital notch (Table 3).²³

Laser therapy has been proposed as effective for long-term disease control and, together with surgery, is considered for cases where conservative treatments fail or as an adjuvant aftersurgery to prevent recurrence. It can be combined with topical corticosteroids in refractory cases. Laser therapies described for AKN include:

- 755 nm Alexandrite (12–16 J/cm² fluence, 18 mm spot size, 3 ms): In the study by Tawfik et al., 6 sessions were conducted at monthly intervals, with greater efficacy observed in papular lesions than in keloids.²⁴
- 810–819 nm diode: Studies by Maranda and Umar reported efficacy in treating inflammatory and fibrotic papules after the 4th session, with sessions every 1–1.5 months and 90–95% lesion improvement.^{3,15}
- 1064 nm Nd:YAG (25–35 J/cm² fluence, 10–13 mm spot size, 35 ms): Effective for darker phototypes, it achieves an 82% reduction in lesion number and size between the 3rd and 6th sessions.^{15,23,25,26}
- 2490 nm Er:YAG (5 Hz, 3 mm spot size, 300 ms, 800–980 mJ pulse energy, partially overlapping mode): Gamil et al. validated Er:YAG laser as an effective, minimally invasive option for early and late AKN lesions, with superior efficacy in advanced lesions vs Nd:YAG.²⁵
- CO₂ Laser (10,600 nm): Used for surgical excision of plaques and tumor lesions in AKN under local anesthesia. CO₂ laser evaporation using a defocused beam with 130–150 J/cm² fluence has been associated with recurrence in some reported cases.³

Phototherapy

Its utility is based on the anti-inflammatory, immunosuppressive, and antifibrotic effects of UVB light (290–320 nm). Okoge et al. reported its effectiveness in papular lesions with usage 3 times a week for 8–16 weeks. The initial dose was determined by the minimal erythema dose and

Table 3 Severity levels and prediction of the new hair implantation line with the use of laser therapy.

Levels	Limits	New hair implantation line
Level I	Lesions in the sagittal area defined by the horizontal line that borders the mastoid process	New hairline indistinguishable from the normal posterior hair implantation line
Level II	Lesions in the upper half described above, below the level of the occipital notch	New posterior line higher than typical hairlines
Level III	Lesions extend above the occipital notch or slightly above it	Very high posterior line at the occipital notch, thinning hair is accepted in the area to treat rather than complete baldness if lesion improvement is achieved
Level IV	Generalized scalp skin condition	Patient with total alopecia or very thin hair in the treated area

Source: Adapted from Umar.²³

230 increased by 20% per session as tolerated, up to week 8 and
231 maintained until week 16. Lesions decreased by 34% and 49%
232 at weeks 8 and 16, respectively, with improved appearance,
233 though less effective compared to laser treatment.^{15,27}

234 **Surgical treatment**

235 Surgical treatment is recommended for extensive AKN
236 lesions and cases refractory to other treatments. Effectiveness
237 depends on complete removal of hair follicles to
238 prevent recurrence and excision of lesions down to the sub-
239 cutaneous tissue.

240 According to Umar’s clinical classification of AKN, the
241 sagittal width of lesions relative to the nuchal area predicts
242 esthetic outcomes post-excision (Fig. 5). Lesions with
243 a sagittal width > 3 cm are more likely to result in wide or

244 dehiscent scars.^{14,28} The association with dissecting cellulitis
245 and folliculitis decalvans has been linked to wide scars
246 and dehiscence due to the deterioration of tissue laxity.¹⁴

247 For lesions with a vertical width < 3 cm, secondary intention
248 healing is preferred. Lesions > 3 cm sagittal width,
249 located in the lower nape or extending beyond the nuchal
250 area, require tension sutures. Lesions 2.5–3 cm sagittal
251 width in the upper nuchal area are ideal for primary closure.
252 After excision and primary closure, trichophytic closure
253 allows hair growth through the scar, resulting in a camou-
254 flaged scar.²⁸ Glenn et al. reported a 35% recurrence rate in
255 patients with primary closure or skin grafts.²⁹

256 Elliptical excision allows for healing within 3 months (6–8
257 weeks on average), with better contraction and esthetic
258 results compared with pear or semicircular excisions. Recur-
259 rence is linked to inadequate excision of affected follicles.²⁹
260 Umar et al. propose a bat-shaped excision for esthetically

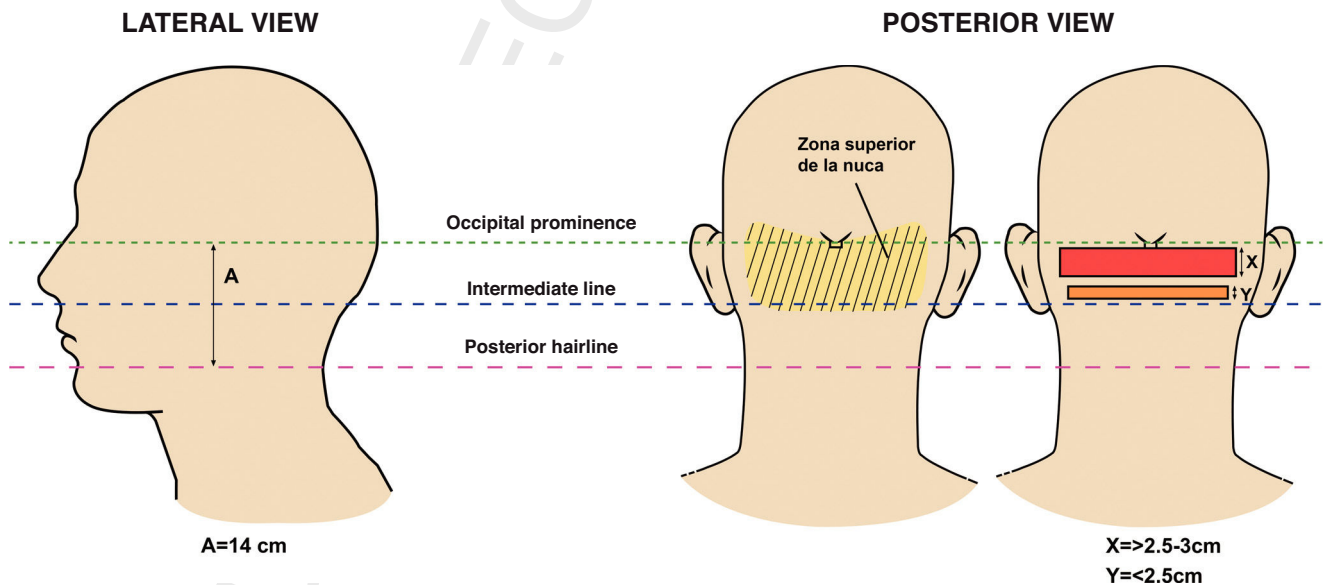


Figure 5 Lateral and posterior views indicating the anatomical references of the nuchal area—upper line demarcated by the occipital protuberance and lower line by the posterior hairline—delineating treatment areas. Figure adapted from Umar et al.²⁸

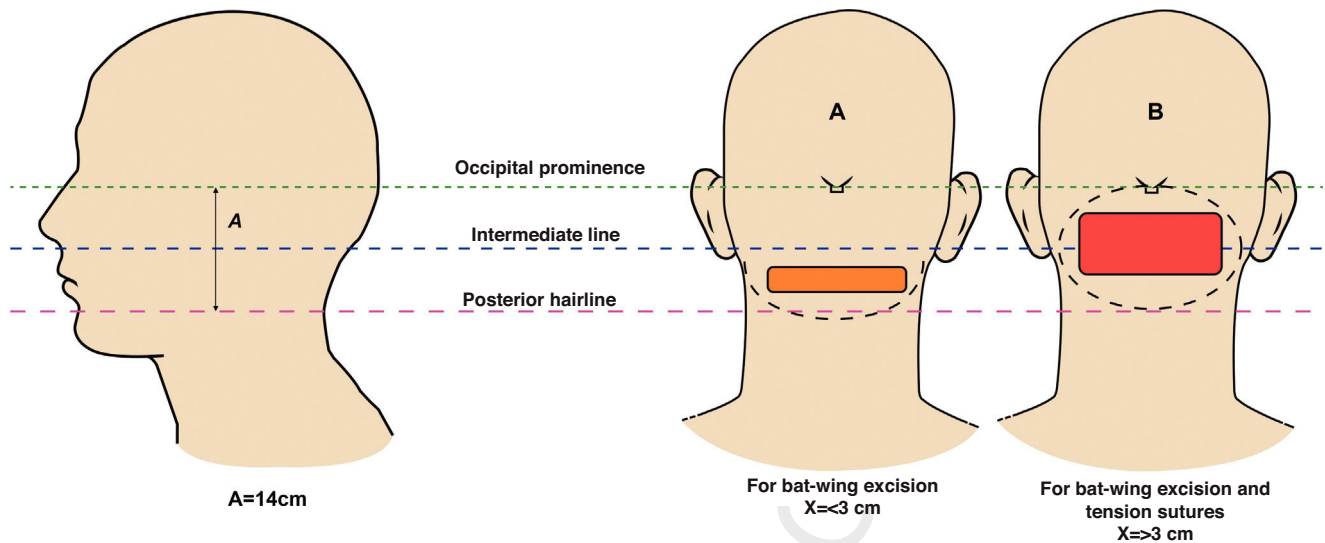


Figure 6 Lateral and posterior views of the head showing reference points for bat-shaped excision alone (a) and with tension sutures. Adapted from Umar et al.³⁰

261 acceptable posterior hairlines, recommended for lesions
262 within the nuchal area. No recurrence was observed during
263 1.9 years of follow-up (Fig. 6).³⁰

264 Extensive excision may be considered in patients with
265 large tumor lesions. Staged removal is preferable, as noted
266 by Galarza et al.³¹ Using negative-pressure wound therapy
267 and split-thickness skin grafting, Labib et al. observed an
268 80% faster healing rate compared to healing by secondary
269 intention, with complete healing in less than two months.
270 At the 8-month follow-up, no evidence of new lesions was
271 observed.³² Electrosurgery is an option that allows for excision
272 and hemostasis simultaneously, which is similar to
273 CO₂ laser excision (130–150 J/cm²). With electrosurgery, no
274 recurrence was observed at 8 months, unlike with CO₂ laser
275 therapy.^{3,33}

276 Treatment involving follicular unit extraction, punch,
277 and healing by secondary intention may be considered for
278 papular lesions < 5 mm (fibrotic papules), with variable effi-
279 cacy depending on the removal of intralesional hair. In
280 their case report, Umar and Khanna observed improve-
281 ment after the first session, with remission maintained
282 for 6–12 months after extraction and acceptable esthetic
283 outcomes.^{34,35}

284 Postoperative therapies may include the use of intralesional
285 or topical corticosteroids, laser therapy, or radiother-
286 apy to prevent recurrence or the appearance of hypertrophic
Q2 scars (Fig. 7).

Other therapies

287 Cryotherapy may be a useful alternative when primary
288 closure is not feasible. Treatment involves 2 cycles of
289 freeze–thaw for 15 s (spot freezing).^{1,3,35}

290 Local radiotherapy is a treatment modality for suc-
291 cessfully managing disease refractory to conservative
292 treatments, particularly when surgery and laser therapy are
293 not viable options. Its use at low doses to eliminate intralesional
294 hair not reachable by laser (broad lesions) reduces
295 signs and symptoms of inflammation, with sessions of 2–3 Gy
296 at 6 MeV over 10–20 sessions. Millán-Cayetano et al. report
297 that total doses of 30 Gy do not reach the threshold to
298 cause permanent alopecia (43 Gy). By avoiding high radi-
299 ation doses, complications such as edema, telangiectasias,
300 and skin cancer are minimized. Additionally, tissue remain-
301 ing after treatment is more likely to respond well to surgery,
302 with fewer complications related to wound healing. How-
303 ever, potential adverse effects must be considered. The
304 estimated risk of skin cancer development by age 70 is 44
305 per 10,000 cases with a dose of 40 Gy.^{36–38}

306 Lobato-Berezo et al. reported various treatments pre-
307 viously unmentioned, such as photodynamic therapy, JAK
308 inhibitors (baricitinib or tofacitinib), and biological thera-
309 pies like infliximab and adalimumab. The response to these
310 treatments was mild in 25.75% of cases, moderate in 18.57%,
311 and complete in 37.14%.¹²

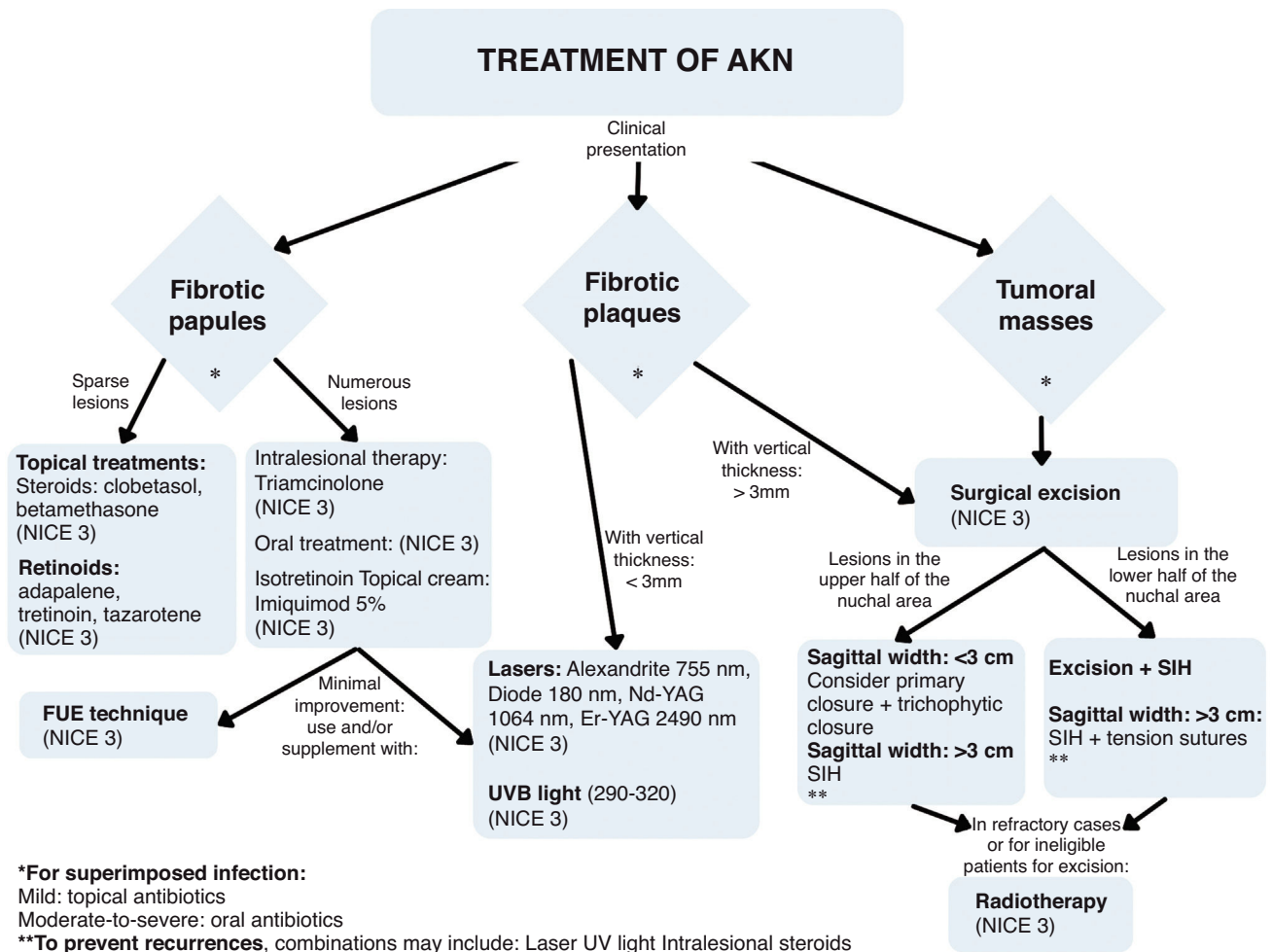


Figure 7 Proposed treatment algorithm for AKN based on stage at clinical presentation.

Conclusions

AKN treatment is a therapeutic challenge. Multiple treatments with varying benefits should be considered based on lesion type, severity, and extent, aiming to reduce inflammation and fibrosis while achieving acceptable esthetic outcomes.

Surgical modalities combined with energy-based and medical therapies could ensure better long-term outcomes tailored to lesion morphology. More randomized controlled studies are needed to support treatment efficacy.

Conflicts of interest

None declared.

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