TABLE 72-10 HYPOPIGMENTATION (PRIMARY CUTANEOUS DISORDERS, LOCALIZED)

	Clinical Characteristics	Wood's Lamp Examination (UV-A; Peak = 365 nm)	Skin Biopsy Specimen	Pathogenesis	Treatment
Idiopathic guttate hypomelanosis	Common; acquired; 1–4 mm in diameter Shins and extensor forearms	Less enhancement than vitiligo	Abrupt decrease in epidermal melanin content	Possible somatic mutations as a reflection of aging or UV exposure	None
Postinflammatory hypopigmentation	Can develop within active lesions, as in subacute cutaneous lupus, or after the lesion fades, as in atopic dermatitis	Depends on particular disease Usually less enhancement than in vitiligo	Type of inflammatory infiltrate depends on specific disease	Block in transfer of melanin from melanocytes to keratinocytes could be secondary to edema or decrease in contact time Destruction of melanocytes if inflammatory cells attack basal layer of epidermis	Treat underlying inflammatory disease
Pityriasis (tinea) versicolor	Common disorder Upper trunk and neck (shawl-like distribution), groin Young adults Macules have fine white scale when scratched	Golden fluorescence	Hyphal forms and budding yeast in stratum corneum	Invasion of stratum corneum by the yeast *Malassezia* Yeast is lipophilic and produces C_9 and C_{11} dicarboxylic acids, which in vitro inhibit tyrosinase	Selenium sulfide 2.5%; topical imidazoles; oral triazoles
Vitiligo	Acquired; progressive Symmetric areas of complete pigment loss Periorificial—around mouth, nose, eyes, nipples, umbilicus, anus Other areas—flexor wrists, extensor distal extremities Segmental form is less common—unilateral, dermatomal-like	More apparent Chalk-white	Absence of melanocytes Mild inflammation	Autoimmune phenomenon that results in destruction of melanocytes—primarily cellular (circulating skin-homing autoreactive T cells)	Topical glucocorticoids; topical calcineurin inhibitors; NBUV-B; PUVA; transplants, if stable; depigmentation (topical MBEH), if widespread
Chemical- or drug-induced leukoderma	Similar appearance to vitiligo Often begins on hands when associated with chemical exposure Satellite lesions in areas not exposed to chemicals	More apparent Chalk-white	Decreased number or absence of melanocytes	Exposure to chemicals that selectively destroy melanocytes, in particular phenols and catechols (germicides; adhesives) or ingestion of drugs such as imatinib Release of cellular antigens and activation of circulating lymphocytes may explain satellite phenomenon Possible inhibition of KIT receptor	Avoid exposure to offending agent, then treat as vitiligo Drug-induced variant may undergo repigmentation when medication is discontinued
Piebaldism	Autosomal dominant Congenital, stable White forelock Areas of amelanosis contain normally pigmented and hyperpigmented macules of various sizes Symmetric involvement of central forehead, ventral trunk, and mid regions of upper and lower extremities	Enhancement of leukoderma and hyperpigmented macules	Amelanotic areas—few to no melanocytes	Defect in migration of melanoblasts from neural crest to involved skin or failure of melanoblasts to survive or differentiate in these areas Mutations within the c-*kit* protooncogene that encodes the tyrosine kinase receptor for stem cell growth factor (kit ligand)	None; occasionally transplants

Abbreviations: MBEH, monobenzylether of hydroquinone; NBUV-B, narrow band ultraviolet B; PUVA, psoralens + ultraviolet A irradiation.