CASE 26

Patient: A 75-year-old Thai retired male banker from Bangkok

Chief Complaint: Multiple erythematous patches and plaques on trunk and extremities for 3 years

Present History: The patient presented with a 3-year history of erythematous patches on his buttock and multiple plaques on his trunk and extremities. A previous skin biopsy revealed cutaneous malignancy and treatment was received from another hospital. New patches and plaques were delveloped on his trunk, buttock and extremities 2 months ago. The lesions gradually increased in size and number. There was no tenderness or itching.

Past History: Hypertention and asymptomatic severe aortic regurgitation for a year. He denied use of herbal or Chinese proprietary medications.

Family History: Unremarkable.

Dermatological Examination (Figure 26.1-3)**:** Multiple well-defined brownish and erythematous hyperkeratotic plaques, irregular borders on trunk and extremities and multiple small yellowish hyperkeratotic papules both palms and soles.

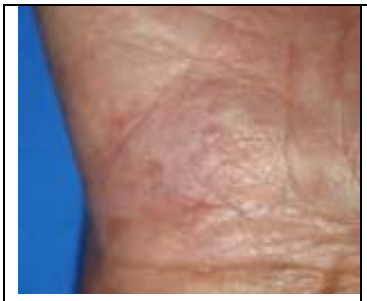

Figure 26.1

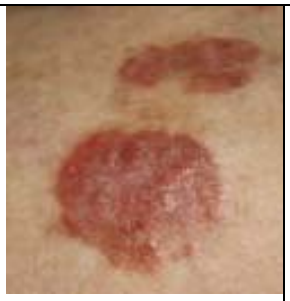

Figure 26.2

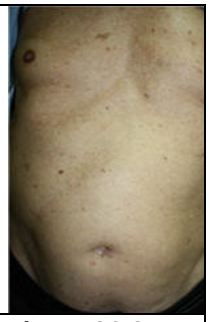

Figure 26.3

Physical Examination:
Vital signs: BP 170/50 mmHg, HR 80 bpm.
General appearance: not pale, no jaundice.
HEENT, Respiratory System, Abdomen: within normal limits.
Cardiovascular system: PMI at 6th ICS lateral to midclavicular line, heaving +ve, DBM and SEM grade III at LUPSB to LLPSB.

Histopathology (S10-4871):
Figure 26.4-5: The skin reveals hyperkeartosis and acanthosis of epidermis. Atypical keratinocytes showing hyperchromatism,pleomorphism are noted accompanying with loss of orderly maturation throughout the epidermis.
Figure 26.6-7: The skin shows nest of atypical kertinocytes invading into the dermis. Keratin pearls formation is noted.

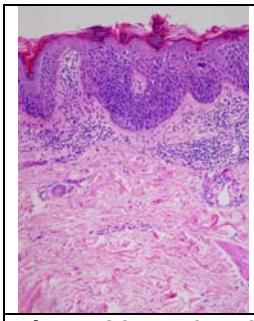

Figure 26.4, H&E 40x

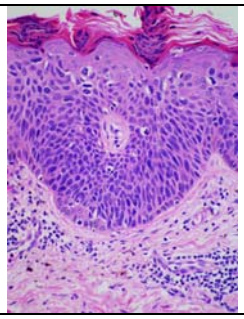

Figure 26.5, H&E 200x

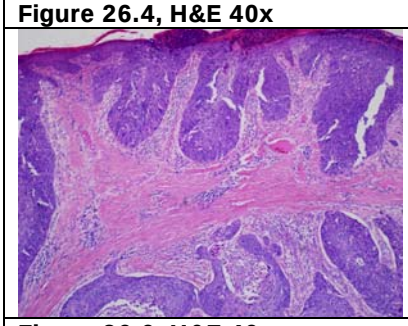

Figure 26.6, H&E 40x

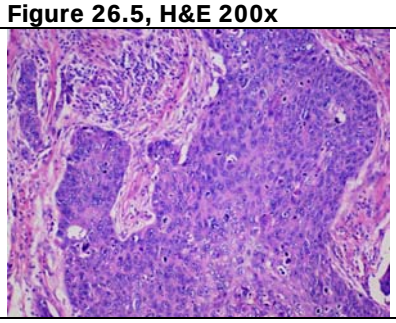

Figure 26.7, H&E 100x

Investigations:

CBC: WBC 5,800/mm^3 Hb13.3 g/dL MCV91 fl
Platelets 230,000/mm^3

LFT: Total protein 74 g/L Alb41.5 g/L AST 19 u/L ALT 29 u/L
ALP 86 u/L GGT 38 u/L
AFP 2.97 ng/ml PSA 1.04 ng/ml

UA: RBC 2-3 WBC 0-1 hyaline cast 0-1 mucous thread few

CXR: no infiltration

Upper abdomen ultrasound: small size of liver, no mass, dilatation of left intrahepatic ducts and common bile duct.

Diagnosis: Chronic arsenicism with Actinic Keratosis and Squamous cell carcinoma

Presenter: Vasinee Kerdvongbundit

Consultant: Penpun Wattanakrai

Treatment: Wide excision
Cryotherapy
Acitretin (10) 1x1 po

Discussion:

Chronic arsenicism is associated with precancerous lesions [arsenical keratosis (ArKs), Bowen disease (BD)], cutaneous malignancies[basal cell carcinoma(BCC), squamous cell carcinoma(SCC) and internal malignancies[lung, urinary bladder, kidney, liver], with latent periods up to 40 years.[1-2]Arsenic-related SCC can arise de novo or from malignant transformation of ArKs and BD. Longer duration of exposure and higher cumulative dose are associated with a higher risk for development of ArkS and other lesions mentioned previously. Systemic manifestations in absence of skin lesions can be found in individuals who drink water contaminated with high arsenic concentration [≥ 50 µg/l]. [3] ArKs typically begins as small, punctuate, yellowish, keratotic

papules most commonly seen on palms and soles in areas of constant pressure or repeated trauma. A diagnosis of ArKs and chronic arsenicism should be considered when numerous characteristic keratoses are seen on the palms and soles or when multiple lesions of BD,SCC or BCC are found in non-sun-exposed regions of the body. Other signs of chronic arsenicism include hyperpigmentation primarily affecting the nipples, axillae, groin, and other pressure points. Within these hyperpigmented patches are often seen small areas of hypopigmentation, resembling "raindrops in the dust." Diffuse alopecia of the scalp may be present. Longer term, patients may develop "blackfoot disease[4-5]," which is a peripheral vascular disorder affecting the lower extremities that eventually results in gangrene. Local treatment options include surgical excision, cryosurgery, curettage, CO_2 laser and topical chemotherapy with 5-FU for ArKs. The cutaneous lesions also respond well to oral retinoids.

Our patient presented with a BD and SCC on non-sun-exposed skin. Moreover there were multiple typical lesions of ArKs. Based on clinical and histological examination the patient was diagnosed with chronic arsenicism. A wide excision was performed for SCC lesion (free margins), Arks and BD were treated with cryotherapy along with use of Acitretin. However, during treatment with Acitretin, multiple new lesions of ArKs and BD continued to occur. Therefore, close follow-up and surveillance of possible malignancies is necessary.

References

1. Wong SS, Tan KC, Goh CL. Cutaneous manifestations of chronic arsenicism: review of seventeen cases. J Am Acad Dermatol 1998;38:179-85.
2. Yu HS, Liao WT, Chai CY. Arsenic carcinogenesis in the skin. J Biomed Sci 2006;13:657-66.
3. Majumdar KK, Guha Mazumder DN, Ghose N, Ghose A, Lahiri S. Systemic manifestations in chronic arsenic toxicity in absence of skin lesions in West Bengal. Indian J Med Res 2009;129:75-82.
4. Tseng CH, Chong CK, Chen CJ, Tai TY. Dose-response relationship between peripheral vascular disease and ingested inorganic arsenic among residents in blackfoot disease endemic villages in Taiwan. Atherosclerosis 1996;120:125-33.
5. Tseng CH. An overview on peripheral vascular disease in blackfoot disease-hyperendemic villages in Taiwan. Angiology 2002;53:529-37.