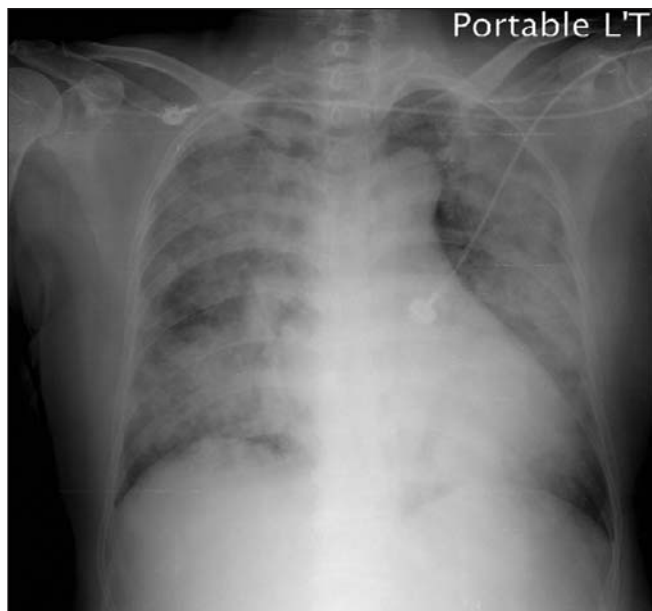


CLINICAL VISTAS BRIEFS

What's your call?



Chest radiograph of a 57-year-old woman who presented with progressive dyspnea and profuse pink frothy sputum that began 6 hours after the onset of left hemiplegia.



Foot radiograph of a 70-year-old man with intermittent sharp pain in his left first metatarsal.



A 72-year-old man with a 15-year history of diabetes mellitus presented with an asymptomatic, whitish-yellow plaque on the sole of his right foot. The lesion measured 7 cm × 4.5 cm and had a verrucous surface and a peripheral hyperkeratotic collarette.

See page 250 for diagnoses.

CLINICAL VISTAS BRIEFS

Neurogenic pulmonary edema

The patient presented with progressive dyspnea that began after a right middle cerebral artery infarction. She had no history of loss of consciousness or obvious aspiration. A chest radiograph obtained on admission showed diffuse homogeneous infiltrates compatible with a non-specific diagnosis of pulmonary edema. An electrocardiogram and an echocardiogram appeared normal, and cardiac enzyme levels were within the normal range. A CT scan performed 3 days after admission showed an extensive middle cerebral artery infarct (Appendix 1, available online at www.cmaj.ca/cgi/content/full/177/3/249/DC1).

The diagnosis of neurogenic pulmonary edema is based on the occurrence of the edema after a neurologic insult and on the exclusion of other plausible causes.¹ Although a number of central nervous system injuries are associated with neurogenic pulmonary edema, the most common is subarachnoid hemorrhage, which accounts for more than two-thirds of reported cases. The incidence of neurogenic pulmonary edema is 23% following subarachnoid hemorrhage, 20% following severe head injury and about 33% among patients with status epilepticus. More rare causes include multiple sclerosis, brain tumour, encephalitis, cervical spine injury and ischemic stroke.^{1,2} The exact pathophysiology of neurogenic pulmonary edema is unclear, but it probably involves an adrenergic response to the cerebral insult, which leads to increased pulmonary hydrostatic pressure and increased lung capillary permeability related to the inflammatory response.^{1,2}

Neurogenic pulmonary edema characteristically presents within minutes to hours after a neurologic insult and usually resolves within 72 hours. Treatment ranges from supportive to endotracheal intubation with mechanical ventilation. The use of dobutamine, osmotic or loop diuretics and α -adrenergic blockers has been described. The condition is likely underdiagnosed; thus, the full distribution of outcomes is unclear. Without

rapid diagnosis and appropriate management, the mortality is high. Physicians should consider neurogenic pulmonary edema when caring for patients with acute respiratory distress following a neurologic insult. In our case, the neurogenic pulmonary edema resolved 2 days after the patient was admitted to hospital, and she was discharged to a nursing home 2 weeks later.

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Carcinoma cuniculatum

The size of the lesion had gradually increased over a 6-year period. A previous diagnosis of a plantar wart had been made, and repeated cryosurgery had been performed. Physical examination showed no evidence of inguinal lymphadenopathy. The patient had no history of infectious diseases or cutaneous malignant tumours. The differential diagnosis included carcinoma cuniculatum and diabetic ulcer. The lesion was surgically excised, and carcinoma cuniculatum was confirmed by histopathology (Figure 1).

Carcinoma cuniculatum (verrucous carcinoma) is an exoendophytic type of

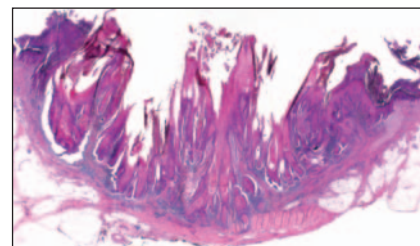


Figure 1: Histopathologic findings of carcinoma cuniculatum. Note the presence of hyperkeratosis, parakeratosis, acanthosis and nests of well-differentiated squamous epithelium with slight atypia extending into the dermis. The lesion was surrounded by chronic inflammatory cells.

low-grade squamous cell carcinoma that often presents as a slowly enlarging papillated tumour.¹ In addition to the foot, it may also occur in the oral and anogenital regions.² The clinical differential diagnosis includes plantar wart, amelanotic melanoma and sarcoma, and the histopathologic differential diagnosis includes keratoacanthoma and pseudoepitheliomatous hyperplasia.¹⁻³ The standard treatments for carcinoma cuniculatum are surgical excision and Mohs' technique (serial excision for microscopic analysis), both of which are associated with a high cure rate and a low recurrence rate. Alternative treatments include curettage and electrodesiccation, cryosurgery, carbon-dioxide laser therapy and radiotherapy.³ Typically, the tumour remains indolent for years, although it may extend into the subcutaneous tissue or metastasize.^{1,2} Early diagnosis and treatment of carcinoma cuniculatum are crucial.

In our patient, no recurrence was seen 36 months after treatment. The presence of multiple, burrow-like openings on the surface of the lesion and a hyperkeratotic collarette should raise the index of suspicion for carcinoma cuniculatum.

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