DEFINITIONS

1. **Polyarteritis nodosa** is one of several diseases in which the underlying lesion is inflammation of the blood vessel wall - i.e. vasculitis or, since it is usually the arteries or arterioles which are affected, polyarteritis.

2. The term **polyarteritis** or **periarteritis** describes an inflammation of the full thickness of the arterial wall - i.e. adventitia, media and intima. The term **vasculitis** extends this definition to include all types of blood vessel. An important practical point is that the inflammatory changes are not continuously present along the length of a vessel, so that histological preparations may miss the affected portion of the vessel and fail to demonstrate the vasculitis.

CLINICAL MANIFESTATIONS

3. The overt presentation is usually preceded by a prodromal illness lasting weeks or months consisting of malaise, anorexia, intermittent fever, night sweats and considerable weight loss. Myalgia and arthralgia often accompany this phase. Hypertension is not a major feature at presentation, but becomes more prominent as a sequel to renal injury. The diagnosis may be very difficult before focal signs develop. The different forms of polyarteritis involve the various parts of the body in similar ways although some organs are involved more commonly in specific forms of arteritis.

4. A wide range of skin lesions is found in 20-50% of patients but the majority are non specific. Specific lesions include skin necrosis, subcutaneous nodules with or without ulcerations and necrosis, and livedo reticularis. Cutaneous nodules are more common with the granulomatous vasculitides. They are found most commonly on extensor aspects of the elbow, then on the fingers and thumb. On the digits, they are often multiple and symmetrical over the distal phalanx. They are also found on the scalp as immobile indurated lesions. Nodules may ulcerate and become infected. Indolent, chronic ulceration of the skin is common with rheumatoid vasculitis.

5. The non-specific lesions include macular erythema, erythema multiforme, erythema nodosum, petechial haemorrhage, subungal or splinter haemorrhages, purpura which may be macular or papular, urticaria in the form of vesicles, bullae or pustules and alopecia. All these lesions may show morphological evidence of vasculitis.

AETIOLOGY

6. The cause of most cases of polyarteritis is unknown but there are some associations which suggest possible mechanisms.

7. Although the general clinical setting of polyarteritis suggests that immunopathogenetic mechanisms cause the tissue damage, other possible mechanisms - e.g. cytotoxins produced by infectious organisms with a secondary inflammatory response - cannot be excluded.

8. Occasionally, the illness follows administration of drugs which may act as an antigen or hapten and initiate an immune response. Those most commonly implicated include sulphonamides, penicillin, gold salts, amphetamines and, more rarely, drugs used by addicts. The evidence that drugs play an aetiological role is, however, circumstantial and it must be remembered that many patients have a prodromal period with symptoms which may be interpreted as an infection for which the incriminated drug is given.

9. More obvious examples of a foreign antigen are those due to infection, such as hepatitis B virus which is assumed to cause an immune complex disease. Evidence of hepatitis B infection is found in less than 10% of cases, contrary to early reports suggesting infection in 30%. Polyarteritis has also been described following streptococcal infections.

CONCLUSION

10. **Polyarteritis nodosa** is a condition of inflammation of blood-vessel walls. The aetiology is unknown, although certain theories listed above have been advanced.

REFERENCE

Wolff S M. Polyarteritis Nodosa Group. In: Wyngaarden J B, Smith L H and Bennett J C (Eds). Cecil Textbook of Medicine. Philadelphia. W B Saunders Company. 19th Ed. 1992. p1539-1541.

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