Sir,

Purpura fulminans (PF) is a rare disorder characterized by thrombosis of dermal vessels and skin necrosis with or without consumptive coagulopathy (1). PF is usually classified into three types: haemostasis-initiated PF or neonatal PF; acute infectious PF; and idiopathic PF (2). However, accumulated evidence has shown that there may be another type of PF: drug-induced PF (3–5).

Disseminated intravascular coagulation (DIC) exists in most, but not all, cases of PF. Some cases have only cutaneous manifestations but mild coagulation changes. The management of PF is difficult and varies with the clinical types of PF. Intravenous heparin inhibits intravascular clotting and reduces consumption of anticoagulant factors. The role of systemic corticosteroid therapy is controversial.

We report here a case of drug-induced PF in a patient with rheumatoid arthritis (RA) who administered a pain-relieving traditional Chinese medication (TCM) called Anluotong Pian.

CASE REPORT

A 50-year-old Chinese woman presented in 2008 with a 10-day history of rapidly progressive painful ecchymoses, haemorrhagic bullae and skin necrosis (Fig. 1A–C). She was apyrexial. She had a 3-year history of recurrent symmetric arthritis of...

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Fig. 1. Ecchymoses, haemorrhagic bullae or skin necrosis on (A) thighs, (B) lower legs and (C) upper arm. Intravascular thrombosis involving small and larger vessels of (D) dermis and (E) subcutaneous tissue (haematoxylin and eosin (H&E) stain, original magnifications ×200). Lesions on the upper arm (F) resolved completely after administration of heparin and antibiotics. Lesions on (G) thighs and (H) lower legs resolved with atrophic scars.
Also many unidentified ingredients in the drug. There were no previous reports on adverse drug reactions of Anluotong Pian. We suggest the drug as the causative agent of the present PF case because: (i) there was a definite history of administering the drug; (ii) the incubation period was one week; (iii) there were no prodromal infections; (iv) the patient administered no other drugs simultaneously; (v) there was an excellent response to corticosteroids; (vi) there was no relapse after withdrawal of the suspected drug and corticosteroids.

Our patient’s typical clinical and histological manifestations are highly suggestive of a drug-induced PF, although she had normal serum protein C and protein S levels and no DIC. The manifestations of our PF case were similar to those of the first propylthiouracil-induced PF case (3). However, in this case, there is still a lack of evidence for a stronger link between the PF and any substance present in the M. androsaceus extract than the contemporary intake of the extract. The fact that the patient had RA may increase the likelihood that the PF was drug-induced, since patients with auto-immune diseases may more often have adverse drug reactions due to aberrant management of immune complexes.

In summary, we report here a case of PF in association with RA; this case further extends previous observations that drug-induced PF manifests mainly in skin without DIC.

REFERENCES