

Letter to the Editor

Dear Sir

In a recently published paper¹ Colachis and Clinchot suggest an association between deep vein thrombosis (DVT) and heterotopic ossification. They hypothesise that the veins are compressed by the heterotopic bone resulting in the deep vein thrombosis. Venous compression results from expansion of the heterotopic bone, ie the heterotopic bone comes first.

In a series of cases they state that all the patients were admitted to a spinal centre and received deep vein thrombosis prophylaxis, but they do not give the dates of admission to the centre when the patients began this treatment. The only information that we have concerning the timing of the admission is for one patient who was admitted 166 days after injury. This information is important since there is another possible explanation for this phenomenon that they have observed which would fit the facts, ie that the two factors are associated but not causally so.

It has been shown repeatedly that deep vein thrombosis inevitably occurs following a spinal injury, due to immobilisation. In normal subjects the deep veins of the legs become thrombosed within a week in 15% of patients confined to bed² and in 50% of patients following major trauma.^{3,4} In paraplegic patients a 100% incidence was found in 14 patients by 60 days after injury.⁵ Oral anticoagulants are not immediately effective. They act by reducing the synthesis of various clotting factors, factor 7 initially, and eventually factors 5, 9 and 10. The reduction of factor 7 at an early stage does not ensure effective anticoagulation and it may take a week for factors 5, 9 and 10 to be sufficiently reduced. Prophylactic anticoagulation therapy must therefore be commenced at the earliest possible moment after injury. The rapidity of commencement of anticoagulation is critical. The patient must be admitted before deep vein thrombosis has occurred. It has been shown that in 100 consecutive cases⁶ (42 patients admitted within 2 days of injury) there were only seven episodes of pulmonary emboli.⁷ When the patients were admitted later, in 102 consecutive cases (only 18 patients being admitted within 2 days of injury) there were 21 episodes of emboli.

Similarly, there is a vital role for the early admission of patients to spinal units in the prevention of heterotopic ossification around the hip.⁸ This was substantiated by a study⁹ which reviewed 124 admissions between 1963 and 1968 and showed a relationship between the occurrence of heterotopic ossification and delayed admissions. This was followed up and further research was carried out where 91 consecutive patients admitted to the National Spinal Injuries Centre with paraplegia were studied to ascertain the incidence of heterotopic bone, and the only factor which could be related to the development of PAO was the delay in admission to the spinal centre.¹⁰ There was a much higher incidence of heterotopic ossification in those patients admitted late to a spinal centre.

I would suggest that the reason that DVT and heterotopic ossification occur together is that both conditions are caused by delayed admission to the spinal centre.

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Reply from Drs Colachis and Clinchot

Dr Silver suggests that following acute traumatic spinal cord injury (SCI), delayed admission to a spinal center results in an increased incidence of both deep venous thrombosis (DVT) and heterotopic ossification (HO), and that the delay in admission may account for the association observed in our study. We noted a statistically significant association between DVT and HO in patients with acute traumatic SCI admitted to our rehabilitation unit. Although we do believe that extrinsic compression may have accounted for cases of coexistent DVT and HO in our study, we do not contend that extrinsic compression alone can explain the findings. Certainly other factors could influence the association between DVT and HO. Any delay in admission resulting in increased incidence of both complications could play an important role, as Dr Silver suggested.

Excluding four patients admitted to our unit over 120 days following injury, the average time from injury to admission was approximately 30 days for patients who developed DVT and/or HO during their acute or rehabilitation hospitalization. Sixteen of our 30 patients with DVT had this condition diagnosed either before or at the time of admission to our unit. Although the vast majority of patients admitted received some form of prophylaxis (either heparin or compression pumps) prior to the development of DVT during their acute hospitalization, earlier admission to our unit might have altered our DVT incidence. It is of interest to note that the Model Systems data¹ from over 2000 acute SCI admissions, revealed a DVT incidence of 16.3% and 13.3% for early (day 1) and delayed (day 2–60) admissions, respectively. Patients treated at our institution do not routinely receive prophylaxis against or screening for the presence of HO.

The highly variable incidence of both DVT and HO reported in the literature necessitates a large, well designed, prospective study to clarify the association between DVT and HO observed in our retrospective study. Factors which alter the incidence of both conditions could undoubtedly influence this association; however, casual effects remain to be determined. We agree with Dr Silver regarding early admission to medical centers specializing in the acute and rehabilitation management of traumatic SCI.

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References

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