

## TEMPORAL ARTERITIS FOLLOWING TRIMETHOXY PSORALEN THERAPY

### A case report

A. MAHAKRISHNAN \* AND G. V. SHESAN NARAYANAN †

#### Summary

A 75 years old male who developed temporal arteritis following trimethoxy psoralen with sun exposure therapy for vitiligo is reported. The possibility of temporal arteritis being provoked by trimethoxy psoralen therapy is discussed.

Temporal arteritis was first described by Jonathan Hutchinson in 1980. It is a disorder of the elderly affecting large, medium and small arteries which shows characteristic histology. It affects both sexes. Though the pathology of this condition is well known, the exact etiology is not known. Trauma, sun exposure, shock, anxiety and depression are known to precipitate acute episodes in the disease<sup>1</sup>. Recent review by O'Brien discussed the importance of actinic damage in the pathogenesis of temporal arteritis<sup>2</sup>. We are reporting a case of temporal arteritis which occurred in a 75 year old man following oral trimethoxy psoralen with sun exposure therapy for his vitiligo.

#### Case Report

A 75 year old male patient who complained of vitiligo was put on oral Trimethoxy psoralen (20 mg) followed by sun exposure for 5-10 minutes. 3 weeks after initiation of therapy he

developed intense headache, fever and tenderness over the scalp. There was no history of any other drug ingestion. There was no history of any impairment of vision. Past history revealed no such attacks in the past.

Examination revealed tender, tortuous, dilated temporal arteries (Fig.) with diminished pulsation on both sides. The overlying skin showed erythema and purpuric spots. There was tenderness over the occiput. Fundus was normal. Systemic examination revealed no abnormality.

#### Investigations

Routine urine examination was normal. Total WBC count was 10,600/c.mm. with a differential count of 60 polymorphs, 30 lymphocytes and 6 eosinophil neutrophils, ESR at the end of first hour was 140 m.m. of Hg. Total serum protein was 7 gm percent with albumin globulin ratio of 3:4. Electrophoresis showed raised gammaglobulin: L.E. cell preparation was negative and X-ray of chest normal.

With the above clinical and laboratory findings a diagnosis of temporal arteritis was made. Patient was put

\* Tutor in Dermatology

† Associate Professor of Medicine  
Tirunelveli Medical College  
Tirunelveli

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on 30 mg. of prednisolone. He was asymptomatic in 2 days and the signs of inflammation subsided within one week. The dose of steroid was gradually reduced to 10 mg. of prednisolone which was continued as a maintenance dose. ESR after 6 weeks was 16 mm after 1 hr; total serum proteins 7.5 gms with albumin-globulin ratio of 4.5:3.



**Fig.** Shows dilated tortuous temporal artery (left side). Supraclavicular region shows hyperpigmented patch in response to methoxy psoralen therapy.

### Discussion

Temporal arteritis also known as giant cell arteritis, cranial arteritis and anarthritic heumatism, usually affects the elderly and involves large, medium and small arteries. Though it has a predilection for temporal arteries, involvement of aorta, cervical, coronary occipital, palatine, renal, vertebral and subclavian arteries has been reported. The cutaneous manifestations may vary from erythema to gangrene. Tortuous, tender, dilated arteries with diminished pulsations is

characteristic. The skin over the inflamed artery may show purpuric spots, crusting, bullae, alopecia and pigmentation. Pain over the temporo-mandibular joints, glossodynia, malaise, fever, myalgia and weight loss are other features of the disorder<sup>3</sup>. Prognosis depends upon involvement of cranial and renal vessels.

Pathologically the disease is characterised by degeneration of elastica of internal elastic lamina followed by granulomatous infiltrate with characteristic giant cells<sup>4</sup>. Though shock, depression and sun exposure with sensitivity to sun light are known to be precipitating factors of acute episodes of the disease, the exact cause for the degeneration of elastica is not known. Recent study by O'Brien established the role of actinic damage to elastica in the causation of temporal arteritis. The occurrence of temporal arteritis in our patient 3 weeks after starting trimethoxy psoralen with sun exposure therapy for vitiligo, though may be coincidental, is worth noting. Actinic damage can be enhanced by trimethoxy psoralen and may precipitate temporal arteritis in elderly people prone to develop this. Systemic side effects of PUVA therapy so far reported include nausea, vertigo, pruritus, burning and blistering of the skin, hyperpigmentation, subungual haemorrhage, acneiform eruptions and photo allergy. Hepato toxicity, chromosomal abnormalities and eye damage are theoretical risks. Recently Hollemen and Jansen reported fulminant reactivation of clinically cured hepatitis after oral methoxsalen<sup>5</sup>. To our knowledge temporal arteritis following psoralen therapy has not been reported earlier.

### Conclusion

A 75 years male who developed temporal arteritis while taking oral trimethoxy psoralen with sun exposure for his vitiligo is reported. Such complication of PUVA therapy has not been

reported earlier. Estimation of ESR in a person complaining of headache while on PUVA therapy may be helpful in the early detection of temporal arteritis.

### References

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## Announcement

### International Dermatopathology Symposium

The 2nd International Dermatopathology Symposium, entitled "Dilemmas and directions in differential diagnosis" will be held at Grosvenor House Hotel, Park Lane, London 12-15 July 1981. The symposium is co-organized by Prof. E. Wilson Jones and Dr. Martin Black of the Institute of Dermatology, University of London, and Dr. A. Bernard Ackerman of the Section of Dermatopathology, New York University School of Medicine. The emphasis will be on histologic clues to diagnosis by conventional microscopy, clinical, electron microscopic and immunopathological aspects of inflammations, neoplasms, malformations and deposits of abnormal materials in the skin.

All enquiries should be addressed to: Marcus Summersfield, London Symposium 1981, Conference Co-ordinates, Regent House, 60 King Street, Twickenham, Middlesex TW1 3SH, with the exception of specific enquiries regarding the medical aspects of the programme which should be addressed to: Prof. E. Wilson Jones, Institute of Dermatology, St. John's Hospital for Diseases of the Skin, Lisle Street, Leicester Square, London WC2H 7BJ.