publish details of this case; also Dr O P Dinnick, Dr M Harrison and Professor R W Gilliatt for their advice.

References

Eckhoff N L
(1931) *Lancet* ii, 343–345
Fowler T J, Danta G & Gilliatt R W
(1972) *Journal of Neurology, Neurosurgery and Psychiatry* **35,** 638–647
Gilliatt R W
(1981) *Mayo Clinic Proceedings* **56,** 361–370
Health Equipment Information (1980) No. 83, p 12. DHSS, London
Lundberg G
(1970) *Scandinavian Journal of Plastic and Reconstructive Surgery*, Suppl 6; pp 3–113
Moldaver J
(1954) *Archives of Surgery* **68,** 136–144
Ochoa J, Danta G, Fowler T J & Gilliatt R W
(1971) *Nature (London)* **233,** 265–266
Ochoa J, Fowler R J & Gilliatt R W
(1972) *Journal of Anatomy* **113,** 433–455
Rudge P
(1974) *Journal of Bone and Joint Surgery* **56B,** 716–720
Rydevik B & Lundberg G
(1977) *Scandinavian Journal of Plastic and Reconstructive Surgery* **11,** 179–187
Rydevik B, Lundberg G & Bagge U
(1981) *Journal of Hand Surgery* **6,** 3–12

Familial naevus sebaceus[1]

B E Monk MA MRCP
King's College Hospital, London SE5 9RS
D I Vollum FRCP
Lewisham Hospital, London SE13 6LH

The occurrence of a naevus sebaceus of Jadassohn in a mother and her daughter is described. A familial basis to this disorder has not previously been reported.

Case report

A 33-year-old woman was referred with a bald patch on her scalp present since birth, on which a small nodule had developed in the preceding months. Examination revealed a 3 × 2 cm area in the left parietal region devoid of hair and with a yellow verrucous surface, surmounted by a 1 cm diameter red nodule (Figure 1). Histological examination of the excised lesion confirmed the clinical diagnosis of a naevus sebaceus, the nodule being a syringocystadenoma papilliferum arising from within it. The patient's 12-year-old daughter was also referred with a similar smaller bald patch at the occiput, again present since birth. Examination revealed a typical naevus sebaceus,

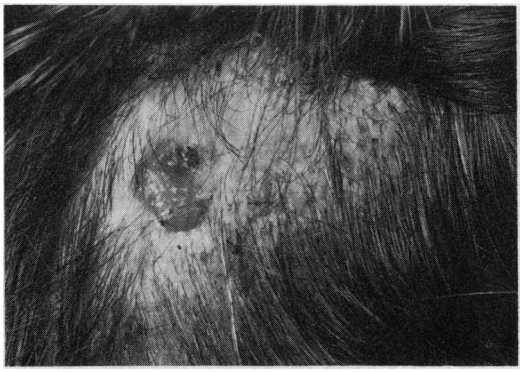

Figure 1. Naevus sebaceus with syringocystadenoma papilliferum arising from within it

approximately 2 × 1 cm (Figure 2), which was confirmed histologically.

Discussion

Naevus sebaceus of Jadassohn is important not only as an unsightly cosmetic blemish but also because of its propensity to the development of a variety of benign and malignant tumours. These are most commonly syringocystadenomas or other benign adnexal tumours, and basal cell carcinomas, but the development of squamous cell carcinoma, keratoacanthoma, and sebaceous carcinoma have all been reported (Rook 1979).

Mehregan & Pinkus (1965), in reviewing 150 cases of naevus sebaceus, found 52 tumours developing within the naevus, of which 21 (14%) were basal cell carcinomas, 27 were benign adnexal tumours, and 4 were keratoacanthomas.

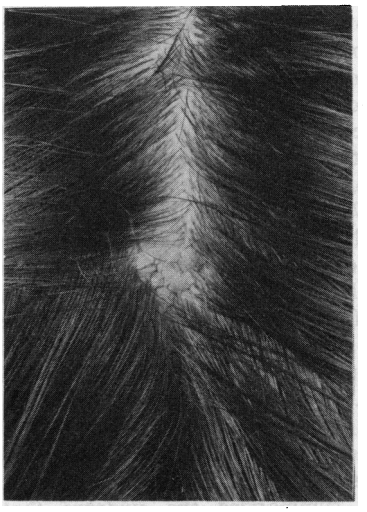

Figure 2. Naevus sebaceus in the occipital region

[1] Case presented to Section of Dermatology, 19 November 1981. Accepted 29 January 1982

0141–0768/82/080660–02/$01.00/0

Wilson Jones & Heyl (1970) found 9 cases of basal cell carcinoma and one squamous cell carcinoma in 140 cases of naevus sebaceus. However, the lower incidence of basal cell carcinoma (6.5%) found in their report may be accounted for by their exclusion of a number of cases showing focal basaloid proliferation which was not considered to have achieved 'biological malignancy'.

A genetic basis has not hitherto been ascribed to naevus sebaceus, and no familial cases are reported in the literature. It is, of course, impossible to exclude the possibility that the appearance of the condition in a mother and her daughter is purely a chance association. However, if naevus sebaceus does indeed have a genetic basis, it should be recognized as an inherited precursor of malignant disease, albeit of a generally innocuous kind.

References

Mehregan A H & Pinkus H
(1965) *Archives of Dermatology* **91**, 574–588
Rook A J
(1979) In: Textbook of Dermatology. 3rd edn. Ed. A J Rook *et al.* Blackwell, Oxford: pp 168–169
Wilson Jones E & Heyl T
(1970) *British Journal of Dermatology* **82**, 99–117

Myocardial infarction during asthmatic attack induced by ingestion of propranolol[1]

Ann B Millar MB ChB[2]
Royal Liverpool Hospital, Liverpool L7 8XP

There have recently been several reports in the medical literature of patients developing severe bronchospasm following ingestion of beta-blockers, including a patient who had a cardiorespiratory arrest (Harries 1981, Raine *et al.* 1981). A patient is now described with asthma and ischaemic heart disease who developed bronchospasm following ingestion of propranolol and subsequently suffered a myocardial infarction. This emphasizes the danger of beta-blockers in asthmatics and also the problems of patients with asthma and coronary artery disease.

Case report

A 58-year-old married Caucasian clerical officer had suffered from late-onset asthma for six years, usually controlled by regular steroid inhalations for prophylaxis, salbutamol inhalation for relief of acute symptoms and short courses of oral steroids during acute episodes; hospital admission had never been required. She also described a six months' history of substernal aching chest pain, radiating to the left arm, brought on by exercise and relieved within 30 seconds by rest. On the morning of admission to hospital, feeling perfectly well, she went to see her works welfare officer about retirement for non-health reasons. On examination, her blood pressure was found to be elevated at 160/110 mmHg and she was advised to see her general practitioner. At the general practitioner's surgery she was normotensive (blood pressure 120/80mmHg) but in view of the history of angina, propranolol 40 mg twice daily was prescribed. After obtaining the prescription the patient took 40 mg propranolol and within one hour she began to wheeze; within two hours she was unable to walk and dialled for an emergency ambulance.

In the Accident and Emergency Department she had a pulse of 120 beats per minute; her blood pressure was 120/80 mmHg and she could speak and drink only with difficulty. Treatment was instituted with intravenous aminophylline 500 mg, hydrocortisone 200 mg and nebulized salbutamol. Soon after admission the patient developed retrosternal chest pain radiating to the left arm. An ECG showed ST elevation in leads 1, AVL and V1–V3. She was admitted to the Coronary Care Unit where treatment with nebulized salbutamol and intravenous hydrocortisone was continued and 48 hours later she was transferred to the general ward. Her peak flow which was initially 200 l/min had improved to 350 l/min by discharge. Cardiac enzymes peaked at 800 U/l creatinine kinase. The initial ST elevation on the ECG subsided and the T waves became inverted in the anterior standard leads.

Discussion

This patient is unusual in that although asthma and ischaemic heart disease are both common conditions, they are not found coincidentally as often as might be expected. This is perhaps because smoking is uncommon amongst asthmatics (Higenbottam *et al.* 1980).

This patient had an asthmatic attack produced by inappropriately prescribed propranolol which, with other beta-blocking agents, is known to be contraindicated in asthmatics (Williams & Millard 1980, Bernecker & Roetscher 1970).

There are several possible factors contributing to the occurrence of myocardial infarction in this patient, including the drugs used in treatment and the physiological changes which occur during severe bronchospasm. During an acute attack of

[1] Accepted 1 February 1982
[2] Present address: Broadgreen Hospital, Liverpool L14 3LB

0141–0768/82/080661–02/$01.00/0