Thomas Addison and his disease

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Abstract

Thomas Addison’s description of the disease named after him first appears in a book entitled ‘On the constitutional and local effects of disease of the supra-renal capsules’ published by Samuel Highley of 32 Fleet Street London in 1855. This is a superb publication graced with excellent figures as can be seen from the accompanying reprint. Case II from the five cases presented in the book has been selected as being possibly the most representative of the disease, which is seldom nowadays seen in such a florid state since modern diagnostic and therapeutic modalities have become widely available.

Keywords

Addison’s disease.

Introduction

It is rare to have a disease named after oneself, and even rarer to have two. Thomas Addison achieved this, the most remembered of ‘his’ diseases being described and integrated into a pathophysiological entity based on his clinical acumen, diagnostic perspicuity and careful autopsy. One feels that not only would few physicians nowadays have the clinical ability to synthesise such varied clinical observations into a single diagnostic entity, but an inability to carry out autopsies (other than at the behest of the coroner) would render the final synthesis well nigh impossible.

Reading the elegant but convoluted prose of Thomas Addison describing his patients does lead one to write in a more florid Victorian style than one had intended. However, beneath the multiply parenthesised syntax is as sharp a description of the clinical manifestations of adrenal failure as you would be likely to find. Such patients presented Thomas Addison with an enigma: they developed a progressive ‘languor’, became eventually torpid and prostrate, and then died, but without any evidence of serious major organ pathology or malignancy. What takes this from a concise clinical description to one of medical genius is the realisation that this was associated with progressive pigmentation, and was pathologically associated with a disordered appearance of the adrenals. It was not for some years that Brown-Sequard was to demonstrate the essential nature of the adrenals to life. Addison referred to the disorder as melasma suprarenale, but (perhaps surprisingly) the great Parisian physician Trousseau acknowledged Addison’s description by first using the descriptor ‘Addison’s disease’. We now know an enormous amount about the biochemistry of adrenocortical steroids, their regulation by ACTH (adrenocorticotropic hormone), and the activation of the melanocortin-1 receptor in the skin by the melanotropic sequence

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contained within excessively elevated levels of ACTH. However, the clinical description, including an increased thirst for salt, remains unimpeachable.

**Thomas Addison**

Thomas Addison was born near Newcastle-upon-Tyne, the son of a grocer, graduated from Edinburgh, and in 1819 was appointed physician to the Carey Street Dispensary (located off Chancery Lane behind the Royal Courts of Justice in London). He eventually became a full physician at Guy’s Hospital in 1837, where he lectured with Richard Bright. In Case II presented here, he clearly acknowledged the clinical insight of his ‘former pupil and continued distinguished colleague’, Dr Gull (the son of a barge-owner who was to become one of the most eminent physicians of his time, and was also one of the first to accurately describe the clinical appearance of hypothyroidism). Nevertheless, in spite of Addison’s numerous insightful descriptions and observations, he was ‘of a melancholic and nervous disposition’, retired early from practice, and fatally threw himself from a window in Brighton at the age of 65.

**Diagnosis**

The modern diagnosis of Addison’s disease, or primary adrenal failure, usually includes assay of low or very low levels of serum cortisol in the presence of elevated ACTH levels, and little or no response of adrenal cortisol secretion to exogenous ACTH (‘Synacthen’). In Addison’s time, most cases were due to tuberculous infiltration of the adrenals, but nowadays most patients have auto-immune adrenal failure (as may one of the original patients of Addison’s who had patches of skin which were ‘morbidly white’: could this be vitiligo?). But would a physician ever again be able to correlate clinical changes with autopsy appearances in today’s environment? Sadly, I think not.

**Further reading**