Accuracy of CT scanning and adrenal vein sampling in the pre-operative localization of aldosterone-secreting adrenal adenomas

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Summary

In primary hyperaldosteronism, it is important to distinguish between unilateral and bilateral disease, as management strategies differ. In the period 1983–95, we identified 34 patients with primary hyperaldosteronism. Following further investigations, a diagnosis of aldosterone-secreting adenoma was made in 17 patients, and surgery was performed. Computed tomography clearly localized an apparent adenoma (discrete adenoma = 1 cm diameter; normal contralateral gland) in only 10 of these patients (59%); two of these ‘adenomas’ were subsequently shown to be hyperplastic glands without adenomas. Histological examination showed adrenal adenomas in the remaining 15 patients. An ‘adenoma’ also appeared to be clearly localized in 3/17 patients later classified as having bilateral adrenal hyperplasia by adrenal vein sampling. CT scanning, therefore clearly localizes adenomas in only 50% of histologically proven cases, and can also produce misleading results. Adrenal vein sampling results altered our management approach in one third of cases. On the basis of our detailed results we would recommend surgery if there is clear evidence of unilateral aldosterone secretion along with CT findings which may not be strictly localizing but are in keeping with the dominant side on adrenal vein sampling. The decision to refer for surgery in primary hyperaldosteronism can be difficult, and we would caution against too heavy a reliance on CT results when recommending adrenalectomy, and suggest that adrenal vein sampling should remain a routine part of the investigation of patients with primary hyperaldosteronism.

Introduction

Primary hyperaldosteronism can be defined as the inappropriate hypersecretion of aldosterone in the absence of activation of the renin-angiotensin system. In some surveys, primary hyperaldosteronism occurs in up to 2% of the hypertensive population, and is most often due to an aldosterone-secreting adrenal adenoma (55–65% of cases) or less commonly, to bilateral adrenal hyperplasia (35–45%).\(^1\) Glucocorticoid-suppressible hyperaldosteronism, primary adrenal hyperplasia and aldosterone-producing adrenal carcinoma are much rarer causes of primary hyperaldosteronism, each accounting for less than 1% of all such cases.\(^2\) A further subset of primary hyperaldosteronism—aldosterone-producing renin-responsive adenoma—has also recently been described.\(^4\) In primary hyperaldosteronism, it is important to distinguish between unilateral and bilateral adrenal disease, as management strategies differ. An aldosterone-secreting adenoma is usually treated by adrenalectomy, whereas hypertension associated with bilateral adrenal hyperplasia is best managed medically with spironolactone. Several techniques have been used for the pre-operative identification of unilateral disease. These include computed tomography, adrenal venous sampling and adrenal scintigraphy.\(^1,5-7\)

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Since 1983, at the Royal Victoria Hospital, Belfast, we have identified 34 patients with primary hyperaldosteronism. Following further investigations, a diagnosis of aldosterone-secreting adenoma was made in 17 patients and surgery was performed. We have reviewed the investigative and pathological findings in these patients to assess the value of CT scanning and adrenal vein sampling in localizing adenomas. We have also reviewed the investigative findings in 17 patients we have classified as having bilateral adrenal hyperplasia.

**Methods**

During the period 1983–1995, 34 consecutive patients with a diagnosis of primary hyperaldosteronism were investigated further in the Metabolic unit of the Royal Victoria Hospital. All patients underwent adrenal computed tomography (CT) while adrenal vein sampling was performed in 32 of the 34 patients. The initial diagnosis of primary hyperaldosteronism was based on the presence of hypertension, hypokalaemia, elevated plasma aldosterone levels, suppressed plasma renin activity and failure of aldosterone to suppress normally during a saline suppression test (Table 1). Supine serum aldosterone and plasma renin activity were measured at 0800 h following overnight recumbency, and then repeated after 2 h of upright posture prior to performance of a saline suppression test.

Serum cortisol and serum aldosterone were measured by radioimmunoassay using reagents supplied by EuroDPC. The interassay coefficients of variation were <3% and <10%, respectively. Plasma renin activity was measured by indirect assay involving the generation of angiotensin I, using reagents supplied by CIS. The interassay coefficient of variation was <11%.

All patients had CT scanning of adrenals performed prior to adrenal vein sampling. Contiguous 4-mm-thick slices were obtained with magnification. Oral contrast was given, but intravenous enhancement with contrast was not used. Scans were interpreted by an experienced radiologist (EMMcI), who was aware that the patients had primary hyperaldosteronism. An adrenal adenoma was diagnosed when scans showed a discrete unilateral ‘nodule’ of diameter 0.8 cm or greater, with the contralateral gland conforming to all radiological criteria of normality.

Bilateral adrenal vein sampling was undertaken by the same experienced radiologist (EMMcI) in 32 patients, irrespective of the CT scan findings. A percutaneous transfemoral approach was used. A 6.5 F preformed adrenal vein catheter (Cook) was used to cannulate the left adrenal vein, and a 5.0 F Sidewinder I catheter used for the right adrenal vein. Catheter placement was checked fluoroscopically prior to sampling, by injecting a small amount of contrast medium. Samples of venous blood were collected from the left adrenal vein (LAV), the right adrenal vein (RAV) and from the lower inferior vena cava (IVC) for aldosterone and cortisol measurement. Accurate cannulation of the adrenal veins was assumed if cortisol levels were elevated (at least doubled) in the adrenal vein samples compared with the lower IVC sample.

**Results**

Table 1 shows clinical and biochemical characteristics of all 34 patients. Mean age was 48 years. All patients presented with hypertension. All but two were hypokalaemic (<3.5 mmol/l) at referral. The diagnosis of primary hyperaldosteronism was confirmed by the demonstration of elevated plasma aldosterone levels, suppressed plasma renin activity and failure of aldosterone to suppress normally after an intravenous saline load (2 l normal saline over 4 h). Although mean supine plasma aldosterone concentration was high, several patients had normal aldosterone levels at some point during their investigative work-up. Plasma renin activity was suppressed below the limit of detection of our assay in many but not all patients. All patients showed a clear failure of aldosterone to suppress during the saline suppression test.

Of the 34 patients, a diagnosis of aldosterone-secreting adenoma was made in 17, and these patients all underwent unilateral adrenalectomy. In 13 of these cases, histology confirmed an adrenocortical adenoma. In two cases histological examination showed cortical hyperplasia only. In the remaining
Investigating primary hyperaldosteronism

17 patients, a decision was made to avoid surgery and these patients were treated medically, with satisfactory improvements in blood pressure control. These cases were classified as having bilateral adrenal hyperplasia.

Accuracy of CT in differential diagnosis

In the 17 patients operated upon, computed tomography clearly localized an ‘adrenaloma’ in only 10 (Table 2, patients 1, 2, 3, 9, 11, 12, 13, 15, 16 and 17). In two of these 10, histological examination showed a hyperplastic adrenal gland without an adenoma (Table 2, patients 15 and 16). This gives CT scanning in our experience a sensitivity of 53% for the detection of adenomas in primary hyperaldosteronism. In the 17 patients not referred for surgery, computed tomography showed unequivocal bilateral adrenal abnormalities in only three (Table 3, patients 24, 28 and 32). In the remaining 14, CT demonstrated a unilateral abnormality. In fact in three patients (Table 3, patients 22, 25 and 30) an ‘adenoma’ was clearly localized using the criteria defined above (Methods). These cases were not referred for surgery because in all three, adrenal vein sampling demonstrated high levels of aldosterone on both sides. In two of the three cases the higher aldosterone level was found on the side of the radiologically ‘normal’ adrenal gland.

Accuracy of adrenal vein sampling in differential diagnosis

Adrenal vein sampling was successfully carried out on 32 patients with no complications such as adrenal infarction, adrenal vein thrombosis or adrenal vein perforation. Adrenal vein cortisol levels were used as an index of successful cannulation. The left adrenal vein was cannulated in 93% of cases and the right adrenal vein in 61%.

Of the 17 patients who underwent unilateral adrenalectomy, venous sampling was carried out in 15 patients including the two who were subsequently shown to have hyperplastic change only (Table 2). The pattern in a Conn’s adenoma is that of unilateral aldosterone hypersecretion, and adrenal vein sampling correctly localized all adenomas in patients in whom the investigation was carried out (13/15, Table 2). Each of these 13 patients showed a high affected to unaffected ratio and a low unaffected to IVC ratio. The mean affected to unaffected ratio was 59 but ranged from 2.2 to 422. The mean unaffected to IVC ratio was 0.9 and ranged from 0.4 to 1.7 (Table 2). A significant number of adenoma cases showed an unaffected to IVC ratio of <1, implying suppression of the unaffected gland. The use of aldosterone to cortisol ratios, or the use of data from left adrenal vein catheterization only, did not improve diagnostic accuracy. Adrenal vein sampling data clarified the need for surgery in six cases where the CT scan findings were equivocal (Table 2, patients 5–8, 10 and 14).

Two patients were misclassified (Table 2). The first (patient 15) had adrenal vein sampling results that were different from the ‘normal’ adenoma pattern. Although CT scanning showed a definite adenoma in the left adrenal with a normal right adrenal, and there was a high level of aldosterone detected in the left adrenal vein, there was also a considerably increased level in the right adrenal vein compared with the IVC. The unaffected to IVC ratio was high (8.7) and in keeping with a diagnosis of bilateral adrenal hyperplasia rather than an adenoma. Histology confirmed hyperplasia. The diagnosis in retrospect was bilateral adrenal hyperplasia, and surgery should have been avoided.

The second ‘misclassified’ patient (patient 16) showed adrenal vein sampling results that were very typical of unilateral aldosterone hypersecretion. CT scanning suggested an adenoma in the left adrenal. Venous sampling demonstrated a high aldosterone level in the left adrenal vein with a low level on the right side. The affected to unaffected ratio was high, and the unaffected to IVC ratio was 0.5, implying suppression of right adrenal aldosterone secretion—a pattern typical of a left adrenal adenoma. Histology showed hyperplastic change with no discrete adenoma. This case may be an example of the rather rare situation of unilateral or primary adrenal hyperplasia.

Table 3 shows adrenal vein sampling data in patients not referred for surgery. These individuals had high levels of aldosterone in both adrenal veins with high adrenal vein to IVC ratios on both sides. This pattern is in keeping with bilateral aldosterone hypersecretion, and these patients were not therefore considered for surgery despite the presence of lateralizing CT findings in many cases. Adrenal venous sampling data clarified the need to avoid surgery in six individuals (Table 3, patients 18, 22, 23, 25, 30 and 34), in each of whom CT scanning suggested the presence of unilateral adenomas of various sizes.

Accuracy of postural changes in serum aldosterone in differential diagnosis

Serum aldosterone increased following 2 h of upright posture in 83% of all patients classified as having bilateral adrenal hyperplasia. This increase was >30% of the supine aldosterone level in 61% of patients. In patients with histologically-proven adrenal adenoma, aldosterone levels also increased in a significant proportion of patients (69%) in the erect position, but to a lesser degree. In three patients...
Table 2  Adrenal vein sampling and CT scan data in patients operated on

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with adenoma, the increase was >30% of the basal supine aldosterone level.

**Discussion**

Although the initial biochemical diagnosis of primary hyperaldosteronism is usually straightforward, the differential diagnosis can be difficult. It is important to reach an accurate final diagnosis, as management strategies differ. An aldosterone-secreting adrenal adenoma and unilateral primary adrenal hyperplasia should be treated surgically, whereas bilateral adrenal hyperplasia will not respond to surgical treatment and is best treated medically. In practice, the crucial decision in the management of a patient with primary hyperaldosteronism is whether or not to refer for adrenalectomy. This decision is based on whether the adrenal disease is unilateral (aldosterone-secreting adenoma) or bilateral (bilateral adrenal hyperplasia). We have found the decision to refer patients for adrenalectomy is often difficult to make, as in our experience many cases are not straightforward or clear-cut. We have in the past based our decision to refer for surgery on the results of two localizing investigations—CT scanning and adrenal vein sampling.

Imaging plays an important role in the differential diagnosis of primary hyperaldosteronism. Adrenal computed tomography is a fast, easily accessible, non-invasive investigation. It has been associated with a sensitivity of between 70–90% in the detection of Conn’s adenomas with a high positive predictive value and few false positives. This has not been our experience. We have found that CT scan findings can be misleading, and it is for this reason that we always carry out both CT scanning and adrenal vein sampling in all patients with primary hyperaldosteronism.

In our series, adrenal CT scanning, even when interpreted by an experienced radiologist, showed poor diagnostic accuracy. CT clearly localized an adenoma in only 50% of histologically-proven adenoma cases, and suggested the presence of an ‘adenoma’ in two patients who were subsequently shown to have cortical hyperplasia only. In our series, CT scanning also localized ‘adenomas’ in a number of patients not referred for surgery because of unequivocal evidence of bilateral aldosterone hypersecretion on adrenal vein sampling, suggesting that the false-positive rate for CT scan identification of adrenal adenomas in unselected cases of primary hyperaldosteronism may be even higher. Histopathologists have defined some cases of hyperplasia as either diffuse micronodular, macronodular or combinations of both. Confusion remains over what constitutes an adenoma as opposed to a ‘prominent nodule’. Misinterpretation of a prominent macronodule in nodular hyperplasia as a solitary adenoma on CT has been previously reported. The increasing frequency with which incidental non-functioning adrenal adenomas are being recognized further complicates CT scan interpretation.

We used a strict definition for clear localization of an adrenal adenoma, i.e. an adenoma of at least 0.8 cm in diameter with a completely normal contralateral adrenal gland. Most Conn’s adenomas, although small, have an average size of 1.6–1.8 cm diameter. However, about 15–20% of aldosterone-secreting adenomas are <1 cm in diameter, and may be missed on CT scanning. However, with good-quality CT scans, nodules of <1.0 cm can be clearly defined. We could with certainty identify nodules as small as 0.4–0.6 cm in diameter, but accepted 0.8 cm as being a diagnostically safe lower limit. Even when we changed our definition to include adenomas <0.8 cm in diameter, our overall CT accuracy did not improve to the high levels quoted by others. Because of a reported high sensitivity of CT scanning in detecting adenomas, some have suggested that adrenal vein catheterization studies should not be carried out routinely, but only in selected patients when CT scanning does not show a clear lesion. Our results indicate that in a tertiary referral centre a high proportion of patients with primary hyperaldosteronism will require adrenal vein sampling for accurate diagnosis, and that reliance on CT scanning as the sole localizing investigation will result in misclassification of a significant minority of these patients. We would stress the importance of correlating CT findings with endocrinological studies.

Bilateral adrenal vein sampling, first described in 1969, is extremely accurate in the pre-operative evaluation of patients with primary hyperaldosteronism, with a sensitivity approaching 100% and a positive predictive value of 90%. Complications such as intra-adrenal haematomas, adrenal infarction, adrenal vein thrombosis and adrenal vein perforation are uncommon in experienced hands. The principal difficulty with the technique is failure to catheterize the right adrenal vein, which occurs in 10–30% of cases.

We have found adrenal vein sampling to be a very important investigation. In our series, sampling results altered the management of a significant proportion of patients. In over a third of cases adrenal venous sampling data was crucial in deciding whether or not surgery was necessary. Catheterization of the adrenal veins (especially the right) can be difficult even in experienced hands. The problem of satisfactory right adrenal vein catheterization is only partly due to problems finding the orifice. As the right adrenal vein is both short and
narrow, catheter ‘wedging’ can make it difficult to obtain an undiluted sample. There have been attempts to compensate for catheterization difficulties and improve the accuracy of adrenal venous sampling by using aldosterone/cortisol ratios or by using data from the left adrenal vein only. Use of these measures did not improve the diagnostic accuracy of our sampling data.

We refer patients with primary hyperaldosteronism for surgery if there is clear evidence of unilateral aldosterone secretion and concordant CT scan findings. CT findings do not need to be strictly lateralizing, but do need to be in keeping with the dominant side on venous sampling. From our series, we can define unilateral aldosterone secretion by the presence of both a raised affected to unaffected aldosterone ratio (>2.2 in all adenomas) and a diminished unaffected to IVC ratio (<1.7 in all adenomas). But we concede that the decision to refer for surgery in primary hyperaldosteronism can still remain a difficult one.

There are a number of other tests that may be useful in the differentiation of aldosterone-secreting adenoma from bilateral adrenal hyperplasia. One such test is the response of serum aldosterone to changes in posture. A rise in serum aldosterone in the upright posture implies that adrenal tissue remains responsive to angiotensin, and this is the case in health and the observed response in bilateral adrenal hyperplasia. In contrast, aldosterone-secreting adenomas are unresponsive to angiotensin, but remain responsive to corticotrophin and aldosterone levels therefore tend to fall during the day while upright, mirroring the fall in corticotrophin and cortisol. In patients with primary hyperaldosteronism, an increase in aldosterone of >30% with adoption of the erect posture is reported to be indicative of bilateral adrenal hyperplasia. Although serum aldosterone did increase following 2 h of upright posture in 83% of our patients with bilateral adrenal hyperplasia, in only 61% was the increase >30%. Furthermore, aldosterone levels increased with the upright posture in a significant number of patients with adenomas (69%), with 23% of patients showing an increase of >30%. Our data suggest that measuring postural changes in aldosterone cannot be relied upon as a differential diagnostic aid. However, our data are limited in that we did not measure postural effects throughout the day, and this might explain the reduced accuracy compared with other studies. We have now modified our investigative work-up to include careful measurement throughout a full morning (4 h). The pattern of secretion of aldosterone and related steroids is also quite different between aldosterone-secreting adenomas and bilateral adrenal hyperplasia. Recent reports that measurement of plasma or urinary levels of 18-hydroxycortisone, 18-oxocortisol and 18-hydroxy cortisol can help differentiate between the main types of primary hyperaldosteronism are of interest, but require confirmation. Levels appear to be higher in patients with adenomas than in those with hyperplasia. We have no experience of their use. Adrenal scintigraphy has also been used to differentiate between unilateral and bilateral causes of primary hyperaldosteronism, but appears to have much lower sensitivity and specificity than the more commonly used techniques of CT scanning and adrenal vein sampling.

In conclusion, while CT scanning has a role in the investigation of the patient with primary hyperaldosteronism, it has in our series a relatively poor diagnostic accuracy. For this reason, we would caution against too heavy a reliance being placed on CT results when recommending adrenalectomy in primary hyperaldosteronism. Adrenal vein sampling is a safe and reliable technique for localizing aldosterone-secreting adenomas and altered our management strategy in one third of our reported patients. Our experience of CT scanning, even allowing for technical advances, has not persuaded us to limit our use of adrenal vein sampling. We suggest that adrenal venous sampling should remain a routine part of the investigation of patients with primary hyperaldosteronism.

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